A Study on Comparison of different Phenotypic methods for detection of Extended Spectrum Beta Lactamase Production among Enterobacteriaceae in Urinary Tract Infection in a Tertiary Care Centre

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY ON COMPARISON OF DIFFERENT PNENOTYPIC METHODS FOR DETECTION OF EXTENDED SPECTRUM BETA LACTAMASE AMONG ENTEROBACTERIACEAE IN URINARY TRACT INFECTION IN A TERTIARY CARE CENTRE"** submitted by **Dr.R.SASIREHA** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D degree Branch– IV (Microbiology) is a bonafide research work carried out by her under direct supervision & guidance.

DR.M.R.VAIRAMUTHU RAJU,M.D.,
DEAN
Madurai Medical College &
Govt.Rajaji Hospital,
Madurai.

DR.P.A.T.JAGATHEESWARY,M.D.,
Director i/c,
Institute of Microbiology,
Madurai Medical College,
Madurai.

CERTIFICATE FROM THE GUIDE

This is to certify that the dissertation **"A STUDY ON COMPARISON OF DIFFERENT PNENOTYPIC METHODS FOR DETECTION OF EXTENDED SPECTRUM BETA LACTAMASE AMONG ENTEROBACTERIACEAE IN URINARY TRACT INFECTION IN A TERTIARY CARE CENTRE"** is a bonafide record of work done by **DR.R.SASIREHA,** under my guidance and supervision in the Institute of Microbiology, Madurai Medical College, Madurai during the period of her Post graduate study of M.D. MICROBIOLOGY from 2014 – 2017.

Prof.Dr.S.RADHAKUMARI
Institute of Microbiology,
Madurai Medical College,
Madurai

DECLARATION

I, **DR.R.SASIREHA** declare that, I carried out this work on, **"A STUDY ON COMPARISON OF DIFFERENT PNENOTYPIC METHODS FOR DETECTION OF EXTENDED SPECTRUM BETA LACTAMASE AMONG ENTEROBACTERIACEAE IN URINARY TRACT INFECTION IN A TERTIARY CARE CENTRE"** at the Institute of Microbiology, Madurai Medical College. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or 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 fulfillment of the rules and regulations for the M.D. Degree examination in Microbiology.

Place : MADURAI **DR.R.SASIREHA**

Date :

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CONTENTS

S.No	Title	Page. No
1	INTRODUCTION	1
2	AIMS &OBJECTIVES	11
3	REVIEW OF LITERATURE	12
4	MATERIALS AND METHODS	39
5	RESULTS	53
6	DISCUSSION	73
7	SUMMARY	85
8	CONCLUSION	87
9	BIBILIOGRAPHY	88
10	ANNEXURE i)Preparation of Gram stain ii)Preparation of Media iii)Data Collection proforma iv)Master Chart v)Ethical Committee Approval Form vi)Anti Plagiarism Certificate	102

INTRODUCTION

INTRODUCTION

Infectious diseases are the major cause of morbidity and mortality and also responsible for worsening the living conditions of many millions people around the World.[41] Molecular studies of pathogenesis of microorganisms revealed an explosion of information about the various microbial and host molecules that lead on to infections and diseases[41].Urinary tract infection (UTI) is one of the most common infection prevalent in humans after respiratory and gastro-intestinal infections. It leads to both community as well as hospital acquired infections (HAI) in developing world and seeks medical attention. About 150 million people are being affected due to UTI across the world[95].In 2010, 3.1% of the people who had been visited emergency department were due to UTI[19]and the incidence rate was about 50,000/million of people in India[95].UTI leads to a number of deaths either due to acute infection or chronic renal failure.

Urinary tract infection is defined as a condition in which the presence and multiplication of bacteria anywhere in the Urinary tract[32].Severity of Urinary Tract Infections mainly depends on factors such as age, time, geographical distribution and immune status. The presence of bacteria in the urine is termed as **Bacteriuria**. The Suprapubic aspiration is most reliable specimen as it is sterile, followed by catheterized urine. There is always a higher risk of contamination of urine samples collected by the patients. Hence Kass introduced the term significant bacteriuria(Kass1956) and it is defined

that the presence of 10^5 or more of the same organism per ml of urine[78] to exclude the bacterial contamination in urine.

Classification of Urinary Tract Infection[25]

The classification of UTI is based on many factors-Anatomically UTI is classified into, Upper urinary tract infection (involves kidney and ureter) and lower urinary tract infection (involves urethra and bladder), with symptoms as Symptomatic bacteriuria and Asymptomatic bacteriuria (ABU) and clinically it is classified into Uncomplicated and Complicated .Uncomplicated urinary tract infection means infection occurring in normal genitourinary tract without prior instrumentation. Complicated urinary tract infection means infection occurring in individual having either structural or functional abnormalities in genitourinary tract or having indwelling catheters.

Epidemiology and Etiology

UTI is one of the commonest infections which needs medical attention. During their life time about 10% of people experience UTI in some form[14].It is one of the important cause for HAI and it accounts for 35% of all HAI. Neonates, young women, prepubertal girls, elderly men, and individual with any structural abnormality or on immune suppression have higher risk for Urinary Tract Infections.

UTI occurs commonly in women than men except in infants and elderly people[41] .In neonatal period UTI incidence is higher in male child due to the congenital anomalies of urinary tract and prostatic hypertrophy in elderly. The

incidence of UTI is higher in female which is about 50-80 % in whom 20-30% of them have recurrent episodes usually within 2 weeks. ABU was found to be more common among 20-40 years of age i.e 5% and it increases to 40-50% in elderly men and women[41]. Most of the UTI are monobacterial (95%)[67] and Escherichia coli is the frequent cause of both community and hospital acquired UTI which accounts for 75%[114] In contrast, recurrence is common in structural abnormalities and associated with polymicrobial infections[67]. Proteus, Pseudomonas, Enterococcus faecalis, Klebsiella, and Enterobacter are common in complicated UTI.

Risk factors[25, 67] - All ages; In both female and male any Urological surgery, Catheterization, Stents, any obstruction in the urinary tract, neurogenic bladder, renal transplantation are the common risk factors. In female with previous UTI, and in males (children and young adults) who have not undergone circumcision are more prone for infection.

Adult female-.Sexual intercourse, use of diaphragm, and pregnancy are the risk factors. Hormonal changes common during pregnancy make urethra and ureter more susceptible to bacterial adhesion and infection. A 70% of pregnant women develop glycosuria due to increased plasma volume and decreased urine concentration resulting in an increase of bacterial growth[28]. UTI is more common in female because Urethra of female is short, so that bacteria have less distance to travel to reach the bladder. In addition urethra is in close proximity to moist, warm vulvar and perianal areas, which are less effective in preventing

bacterial entry. Similarly during sexualinter course bacteria can enter in to the urethra and incomplete emptying of bladder in diaphragm users, as it pushes against the urethra and infection occurs followed by stasis of urine.

Elderly people -In female due to the estrogen deficiency there will be loss of vaginal lacto bacilli which leads them more prone for infection. In post menopausal women- Cystocele is common and affects complete bladder emptying and leads to residual urine followed by recurrent UTI. In elderly male decrease of prostatic secretion which has bactericidal effect also leads to urinary infection.

Pathogenesis Clinical manifestation and Complications[14,67]

Three major routes by which bacteria invade are[8]ascending route, haematogenous and lymphatic spread.

Ascending route - Microorganisms (mainly gram negative bacteria) from gastro intestinal tract able to colonize periurethral region and also in vagina. Adhesion in the uroepithelium is the important step in pathogenesis. Following colonization these organisms gain entry into the bladder through instrumentation or any other manipulation, multiplication happens in the bladder resulting in cystitis. From bladder enter into ureter, and then to the kidney. **Haematogenous route-** Seeding of the kidney occurs due to the systemic infection. **Lymphatic spread-** Whenever there is increase of bladder pressure chance of increase in UTI due to the lymphatic flow to the kidney.

Host defenses in urinary tract depends mainly on - factors like P^H, osmolality, organic acids of urine , presence of bactericidal activity, cytokines and peptides of mucosa of urinary tract, inhibitors of bacterial adherence like Tammhorsfall proteins, lactoferrin, SIgA, low molecular weight oligosaccharides and mucopolysaccharide of bladder are responsible for host defense mechanisms. Humoral and cell mediated immunity, Prostatic secretions are also taking part in this action.

In neonates and children less than 2 years the symptoms are nonspecific. Major manifestations are fever, failure to thrive, and vomiting. In children greater than 2 years localizing symptoms such as dysuria, frequency, and abdominal or flank pain are also observed[12]. In adults, frequent painful micturation is seen due to irritation of vesicle as well as urethral mucosa due to bacteria. Patient may sometimes experience heaviness or pain in suprapubic region and urine may be associated with a tinge of blood or frank blood.
Upper UTI usually manifest with fever with or without chills, frequency, dysuria urgency along with flank tenderness. UTI is asymptomatic in elderly individual and if symptomatic it is not diagnostic as they has been experiencing hesitancy, dysuria, frequency and incontinence very often. Patient with indwelling catheter usually presented with fever and flank pain but without lower urinary tract symptoms.

In Pediatric age group, infection may sometime spread outside the urinary tract resulting in orchitis in boys and sepsis in both sex. The Most

serious complication is Pyelonephritis. In adults recurrent urethritis resulting in urethral narrowing, prostatitis and permanent kidney damage are other important complications. Life threatening complication is sepsis and renal failure. In order to reduce the complication in UTI early intervention with appropriate and adequate dose of antimicrobials. Antimicrobials should bind the target site effectively in order to disrupt the cellular processes for cessation of bacterial growth.

Beta-Lactam antibiotics are used to treat UTI due to their high efficacy, less toxic and well tolerated by the people at any age group. Beta lactam antibiotics act on both gram positive and gram negative bacteria. Antimicrobial resistance is mainly due to any interruption in the essential steps for antimicrobial action it will results in bacterial resistance to antimicrobial action[14].Different aspects of resistances are, Biologic resistance, environmentally mediated resistance and microorganism mediated resistance which is further classified into intrinsic resistance and acquired resistance[8].

Resistance to Beta lactams[14,67]

1. Enzymatic destruction of β-lactam ring by Beta lactamase, produced by the organism
2. Altered target due to the mutation in PBP (Penicillin Binding Protein) resulting in reduced affinity for antibiotic or not able to bind Beta lactams.

3. Decreased uptake or its efflux of the drug due to change either in number or character of porin channels of outer membrane so that the drug does not reach the target site[8].

Worldwide resistance to Beta lactam antibiotics among gram negative uropathogens are increasing because of inappropriate and extensive use of antimicrobial agents. Antibiotic resistances are mainly due to the production of Beta lactamases by uropathogens.

Beta-lactamases are family of enzymes produced by the bacteria which inactivate [59] the Beta - lactam antibiotics by splitting the amide bonds in the Beta lactam ring. Even prior to the use of penicillin in medical practice beta lactamase production was observed in Escherichia coli. Penicillinase was the beta lactamase produced by the Staphylococcus aureus which was plasmid encoded. Due to that there was quick spread of resistance to the other clinical isolates. Naturally occurring chromosomally mediated beta lactamases are usually found in most of the gram negative bacteria. The development of this type of beta lactamases are mainly due to the antibiotic pressure by the organism that found in the environment which are able to produce beta lactam. TEM 1 was the first beta lactamase which was isolated from the Escherichia coli strain from the patient named **Temoniera** of Greece and designated as **TEM.**

Beta lactamase classification - Early classification scheme was by **Richmond and sykes**[59]. **Ambler** proposed more modern scheme based on functional and

molecular characteristics. **Bush-Jacoby Medeiros** proposed another classification which is mainly based on both functional and molecular characterstics. Beta- lactamases are easily transfer from one bacteria to another by their presence in chromosomes or in plasmid. These enzymes located on transposons [59,67] which also contain resistance genes for other classes of antibiotics resulting in multiple drug resistance bacterial strains.

A series of enzymatic variants having broadened spectrum of activity against for newly developed antibiotics appeared in early 1980.These Beta Lactamases are called as **Extended Spectrum Beta Lactamase** which was first reported in the year **1983**. Extended Spectrum Beta Lactamases are the enzymes which confer resistance to penicillins, First, second and third generation Cephalosporins and Monobactams by hydrolyzing the antibiotic and are inhibited by Beta lactamase inhibitors such as clavulinic acid. ESBL belong to the class A of Ambler classification 2be of Bush Jacoby Medeiros classification[45].

Gene responsible for ESBL is located normally in plasmid of 80kb in size or large[10]. This plasmid also carries the resistance determinants for fluroquinolones, aminoglycosides, Tetracyclines Chloramphenicol resulting in multidrug resistant. Multidrug resistance is increasing in Enterobacteriaceae and it is becoming an emerging health problem worldwide as well as in Indian hospital scenario [28,98].ESBL are most troublesome Beta lactamase because most of them encoded in plasmid which facilitate spreading of ESBL from one

organism to another very easily. This plasmid mediated ESBL derived from mutated parent TEM and SHV enzymes [98]. Commonest ESBL types are **TEM, SHV and CTX-M types.**

Depending on different geographical area prevalence of ESBL producers vary and prevalence of ESBL is 28% to 84 % [4] Incidence of community acquired UTI is high in Asia, Denmark, Pacific, Japan, India, Russia and USA ESBL producing E coli in UTI was highest in India (60%), Hong Kong (48%) and Singapore (33%)[106]. Many studies shows there is an increasing emergence of resistance worldwide and also in India for commonly used antibiotics among the uropathogens for the past three decades [90,106]. The reason for the resistance are inappropriate use of antibiotics and lack of knowledge regarding resistance pattern to the corresponding areas lead to the wrong choice of antibiotics[19,90] Among Enterobacteriaceae, Escherichiacoli, Klebsiella, Proteus, Enterobacter are the commonest uropathogens associated with UTI. These are common organism producing Extended Spectrum Beta Lactamase[114]. Incidence of ESBL producing strains are steadily increasing nowadays[106] because they are plasmid mediated. Important reason for therapy failure is the production of ESBL producing strains. Widely used antibiotic for the treatment of Enterobacteriaceae are Beta lactams[10]. Also emergence of Beta lactamase production has become a major problem. The ESBL positive strains show increased mortality and resistance pattern when compared to the non ESBL strain. Multidrug resistance is a major problem in the management of UTI. Many new Beta Lactams were developed over the years. New Beta

lactamase emerged for each new Beta Lactam antibiotics[5]. Due to the overuse of new antibiotics there is an emergence of new variant of beta lactamase.

Antimicrobial resistance surveillance is important for the empirical selection of the antibiotic in order to treat the UTI. This study focuses in detection and incidence of ESBL producing organism in Enterobacteriaceae group of bacteria from the urine sample by different phenotypic methods, (DDST, PCT, CHROM agar and E-test), to compare the sensitivity of different phenotypic methods in detection of ESBL production and also to find out the suitable antibiotic for treating infection caused by ESBL producing bacteria in a tertiary care hospital.

AIMS & OBJECTIVES

Aims and objectives

1. To see the Prevalence of Enterobacteriaceae from urine samples of suspected cases of Urinary Tract Infection.
2. To detect the incidence of ESBL production among the isolated Enterobacteriaceae by Phenotypic methods.
3. To compare the four phenotypic methods in detecting ESBL producing strains among Enterobacteriaceae.
4. To ascertain correlation between Phenotypic and genotypic methods of ESBL detection.
5. To find out the suitable antibiotics for treating the infection caused by Non ESBL and ESBL producing bacteria of Enterobacteriaceae in this setting.

REVIEW OF LITERATURE

Review of Literature

UTI incidence

LatikaJ Shah[62] et al 2015 India defined UTI is the condition in which pathogenic microorganism are detected in the urine with or without presence of specific symptoms. Women are more prone for infection and nearly 20% of women suffer from UTI but this infection is uncommon in men upto fifth decade of life

Besty Foxman[35] et al 2003 Michigan - according to this study 1 in 3 women by the age of 24 years had UTI and need of antimicrobial therapy. UTI was the second most common infection in elderly people and it accounts for nearly 25%.

Chaudhary Navin Kumar[19] et al 2013 India that nearly 40-50% of women experience UTI in their life time. Each year nearly 150 million people are diagnosed as UTI.

Devanand Prakash[28] et al 2013 India -described the UTI as the presence of bacteriuria with urinary symptoms. According to this study the prevalence of infection was 53.82%. The prevalence in women is higher (73.57%) when compared to male (35.14%). Also this study shows that incidence is higher in elderly (63.51%) followed by the age group 26-37 years (58.11%). Incidence of UTI varies with age female to male ratio of age 15-25years is 17:1 and for 26-36years 9.75:1 and for greater than 48years is 0.27:1.

Nader Shaikh[97] **et al 2008** – According to his analysis prevalence of UTI in symptomatic Paediatric population was 7.8%

A Sharma[98]**, et al at Nepal 2011-**During first decade of life nearly 3% of girls and 1% of boys develop UTI. Diagnosis of UTI is one of the markers for urinary tract abnormalities in children. According to this study male to female ratio was 1:1.8.

Ashish Jitendranath[12] **et al 2015 India-**During first 3 months UTI is common in boys. Maximum number of infection is seen in the 0-6 years of age group. Gram positive cocci are seen predominantly in this age group when compared to the other age group.

V.Vijaya Swetha[117] **et al 2014 at India-** Among hospital visits UTI is the second most common cause. In outpatient department nearly 7 million people visit due to UTI and for emergency department 1 million people visits and 1 lakh people are hospitalized annually.

Najar MS[72] **2009 et al** Uropathogens after colonization in to the periurethral region slowly ascent in to the bladder through urethra, to kidney through ureter and to the prostate through ejaculatory ducts. Mechanical barriers that prevent ascension are urethra and uretero vesicle junction. In the bladder after multiplication the organisms colonize the mucosa of the bladder and slowly invade the mucosal surface. Flow of urine and contraction of bladder prevent the stasis of urine and colonization.

Chein-Wei Lin[20] et al 1999 Taiwan said that one of the important cause for fever in neonate is UTI. Diagnosis is difficult because the symptoms are non specific and difficulty in getting sterile samples. Recurrent UTI leads to renal damage. If left untreated, lead to end stage renal disease. In order to prevent these complications early detection of UTI correction of congenital abnormalities of genito urinary tract is important. Incidence of neonate with genitourinary abnormalities is nearly 20-60%. Most common genito urinary tract abnormality is VUR (Vesico Ureteric Reflux) In this study common is UPJ(Uretero Pelvic Junction) stenosis. Low birth weight babies are more prone for UTI. Urine culture is said to be positive if $\geq 10^5$ bacterial colonies in clean catch mid stream urine sample $\geq 10^4$ in intermittent catheterization and any number of colonies in supra pubic aspiration. Main symptoms in neonates are fever, GI problems like Vomiting, Hyperbilurubinemia and poor appetite. In urinary tract obstruction abdominal distension, Oliguria , Urosepsis are common signs and symptoms. Male to female infant ratio of UTI is 1.3:1.

Palak Gupta1[80] 2015 Puducherry UTI manifests in children as fever of unknown origin. Incidence varies with age and sex. In first 3 months of life UTI incidence of Boy to Girl is 3.7:2%. After 3 months ratio is about 1.1:3%. Anatomic and physiological factors play major role in UTI particularly VUR. One of the important reason for recurrence in children is VUR, which leads to dreadful complication like pyelonephritis. Diagnosis of this at appropriate time is important to prevent renal damage.

M Eshwarappa[32] et al 2011 Bangalore India- Here Study group was community-acquired urinary tract infection (CA-UTI). The main aim is to determine the clinical presentation and risk factors associated with UTI. If UTI is associated with risk factors such as higher age, pregnancy, immune suppression and co morbidity the treatment becomes more challenging. According to this study, elderly age group particularly males (50-79) are commonly affected (57.4%).In this age group complicated UTI is common. Uncomplicated UTI is common in female age group of 29 to 44years.Incidence in Pediatric age group is 9.8%. The male: female ratio was 1.63:1in Complicated UTI. In general both in complicated and uncomplicated the most common clinical presentation were fever and dysuria (11.4%). But in acute uncomplicated the common symptom was increased frequency. Children with urolithiasis manifest as dysuria, pain, irritability, and hematuria. In this study diabetes mellitus is the commonest factor (42.6%) responsible for Complicated UTI. Any urogenital instrumentation like stent, TURP, cystoscopy and catheterization increase the incidence of UTI. Chances of development of bacteriuria are greatly increased in patients with catheterization more than two weeks. UTI is not definitely diagnosed only with clinical presentation. In order to diagnose UTI definitely urine culture is very important. Even though UTI is common in developing countries only 9.17% are definitely diagnosed by urine culture.

Taiwo SS [108]et al 2006 Nigeria Any urinary tract instrumentation particularly catheterization contribute to 66-86% of UTI. Patient acquiring infection

through catheterization depends on factors such as host susceptibility, method by which catheter was introduced and duration and quality of catheter. According to previous study 100% of chance of infection is possible if indwelling urethral catheter of more than 4 days draining into an open system and infection rate decreased to 20% if it is maintained in closed drainage. In this study if catheter was in situ for a week, infection rate is about 13.3% and if more than one week rate of infection increases to 98.9%.

Interpretation of urine culture

Oxford text book[78] of 2nd edition - For the diagnosis of UTI demonstration of bacteria in urine is important. But there are certain conditions in which urine is sterile are perinephric tissues, obstucted pyonephrosis and pyogenic abscess of kidney. Just presence of bacteria in the urine does not indicate infection because the urine can be contaminated by the bacteria which are normally present in the anterior urethra and periurethral area. In order to solve this problem Kass introduced one criteria according to which bacterial count $\geq 10^5$ /ml of same bacterial species indicate true bacteriuria which distinguishes from contamination. Accuracy of true bacteriuria is enhanced by the demonstration of pyuria that is more than 10WBC/mm3 but some time in symptomatic women on one occasion they had 10^5 off the same organism /ml of urine and on another occasion count is low. From this observation concept of low count bacteriuria was established. In symptomatic women diagnosis of infection mainly based on the bacterial count 10^2 or more per ml accompanied with pyuria. This low count bacteriuria is very common in UTI associated with

Staphylococcus saprophyticus because it is having longer generation time than other enteral bacteria. In men diagnosis of bacteriuria 10^3 or more of the same organism is sufficient for the diagnosis of true bacteriuria as there is less contamination. Recurrent infection is of two types re-infection and relapse. Relapse means after completion of treatment recurrence of infection with same organism. Reinfection means after eradication of infection with treatment, once again patient is infected with different organism after 7-10days and it is more common than relapse. Treatment failure is defined as the condition in which bacteria are not eliminated from the urinary tract with appropriate antibacterial agent. Main factors which differentiate the true bacteriuria from the contamination are number and nature of the organism. Small number of bacteria or mixed growth is due to contamination.

Kass criteria has been questioned in **CL Saldhana[72] et al 2009**whenthe bacterial counts are 10^2 or more organism per ml when it accompanied by pyuria (>10 wbc/mm^3) in symptomatic young women. The Infectious Disease Society of America (IDSA) slightly modified this Kass criteria. According to IDSA for the diagnosis of cystitis 10^3 CFU/ml and for pyelonephritis it is 10^4 per ml. Epidemiology of urinary tract infections analysis is very helpful for early diagnosis and prevention. In young women annual incidence of uncomplicated UTI is about 0.5-0.7 episodes per patient. In men symptomatic infection is uncommon. Any risk factors which interfere with the normal urinary flow increases the chances of development of infection in both sex of any age group.

Normal flora and pathogens of urinary tract

Conie mahon[25]- New born urine is sterile but in prepubertal age group, the commonest organisms are Micrococci, alpha and non haemolytic Streptococci, and Coliforms. In adults Lactobacillus acidophilus, Staphylococcus epidermidis are predominant.Lactobacillus acidophilus, Yeast, and Staphylococcus epidermidis are the predominant normal flora in pregnancy**.** Common pathogens associated with UTI are Enterobacteriaceae, Pseudomonas, Enterococci , Staphylococcus aureus , Streptococcus agalactiae, less common are Gardnerella and Ureaplasma. In acute pyelonephritis cystitis, CAUTI(Catheter Associated Urinary Tract Infection) Enterobacteriaceae is the commonest. In recurrent and chronic UTI adherent Escherichia coli is common.

Classification of Enterobacteriaceae [25,59]- In humans and animals organisms belonging to the Enterobacteriaceae are normally found in the intestinal tracts. Also they are commonly found in the environment such as soil, water and plants. These types of organisms are frequently recovered from the clinical specimens. Immunocompromised patients are more prone for HAI, either after colonization or by invasive procedures in which mucous membrane are transected or traumatized. Genera and important species of this family discussed in this text book table 6-5.according to Bergey's Manual of Systematic Bacteriology there are 44 genera and 176 named species in Enterobacteriaceae family.Among this Enterobacteriaceae family Escherichia coli, Klebsiella, and Proteus species are having more uropathogenic features and also commonly recovered from the clinical specimens**.**

Virulence factors

Escherichia coli[46,67] is highly uropathogenic due to the presence of virulence factors such as fimbriae, Siderophores, Haemolysin and relative resistance to vaginal fluids. Due to the presence of fimbria it binds firmly to the urothelium. Three types of fimbriae'S' fimbriae (S FA-1), Type 'P' fimbriae and Type 'Dr' fimbriae.

Rozalski [8] **A et al 1997 Poland** according to this uropathogenic feature of Proteus is due to the presence of virulence factors such as fimbriae or afimbrial adhesions, swarming phenomenon, invasiveness, proteolysis, and hemolytic activity .

Archana gupta[9] **et al – New York 2003** presence of extracellular capsule in the **Klebsiella** protect the bacteria from phagocytosis. Fimbrial , non fimbrial adhesions and somatic O antigens serve as virulence factors in addition to the capsule.

Prevalence of Enterobacteriaceae in UTI[7,14]

Yee-Hsuan Chiou[14]**,** Escherichia coli(66.6%) was the commonest organism in neonates followed by Klebsiella(10%) and Enterobacter(7%).In recurrent UTI Escherichia coli, Enterobacter cloacae and Proteus are the commonest organism.

Taiwo[108] **SS et al-**Pathogens like Escherichia coli, Proteus, Klebsiella Pseudomonas, Enterococci, Serratia, Enterobacter, and Candida are associated

with Catheter Associated Urinary Tract Infection. In this study commonest organism is Klebsiella(36.6%),followed by Pseudomonas(27%) and Escherichia coli (20.6%).

Sharma[7] et al-In this study the most frequently isolated pathogens were Escherichia coli 33.3% followed by Klebsiella pneumoniae 11.1% Proteus species 7.4% Edwardsiella tarda 3.7%,Citrobacter fruendii 3.7%.Morganella morganii 3.7%.

Treatment of UTI and role of Beta-Lactams in UTI

Thana Khawcharoenporn[111] et al 2013 Chicago USA-According to IDSA (Infectious Disease Society of America) for uncomplicated cystitis, routinely prescribed drugs are Nitrofurantoin and Sulphamethoxazole - Trimethoprim. But for complicated UTI and for pyelonephritis ceftriaxone, fluroquinolones, carbapenems and aminoglycoside are preferred.

John L Brusch[48] et al- Usually UTI in males are considered as complicated UTI .If the patient is having any obstructive conditions or associated with any comorbid conditions they have to be admitted and these patients should be treatedwith ceftrioxone ceftazidime (third generation cephalosporins), fluroquinolones,or an aminoglycoside.

Richard Colgan[92] et al 2011 Mary land university- For uncomplicated UTI oral antibiotics like Sulphamethoxazole, Trimethoprim Nitrofurantoin, and fluroquinolones are sufficient. If pathogens are resistant to the above antibiotics

then beta lactams have to be given. In pregnant women commonly used antibiotics are Ampicillin, Amoxicillin,and Cephalosporins. In children with UTI, Sulphamethoxazole-Trimethoprim Cephalosporins, and Amoxicillin with Clavulinic acid can be given. Children with acute kidney infections are treated with Cefixime and Gentamicin.

Mechanism of action of Beta lactam antibiotics

Goodman and Gillman[38]- Worldwide the most common group of antibiotic used for infection control purpose are Beta-lactam antibiotics. Among the antibiotic group, Betalactam is the largest group. All the members of this group contains four membered Beta lactam ring. Based on the chemical nature of the ring structure fused to beta lactam, which is divided into groups- Penicillins, Cephalosporins, Carbacefs Monobactams,and Carbapenems. Main action of beta lactam antibiotics is inhibition of the bacterial cell wall synthesis by acting on the peptidoglycan layer. Peptidoglycan is composed of glycan chains cross linked with peptide chain. Repeating units of N acetyl muramic acid and N acetyl glucosamine constitutes the glycan chain and strength and stability to the bacterial cell wall is provided by the cross linkage. Main role of trans peptidase is to cleave the terminal D alanine, in order to release the energy and this energy is used for the cross linking of peptide chain. The process of cross linking is called as transpeptidation which is catalysed by PBP and are made up of transpeptidase and its related proteins. Spectrum of antimicrobial activity that is from narrow to broad spectrum and its efficacy and safety can be

enhanced by modification of the moieties attached to Penicillins and Cephalosporins

Resistant mechanisms of Beta lactams[67]- Mechanism of occurrence of the drug resistance to the Beta lactam antibiotics are1) alteration in the target site 2) affinity of PBP which is decreased for beta lactam antibiotic by modification of exsisting PBP and import of new PBP 3)destruction of Beta lactam antibiotic by Beta lactamase enzyme and decrease of Beta lactam antibiotic concentration inside the cell by restriction of the entry of antibiotic due to the a) loss of porins and b) pumping it out by efflux mechanism. Among these resistance mechanism the production of beta lactamase enzymes by the organisms is the commonest, and antibiotic inactivation by beta lactamase depends on, hydrolysis rate, over production of beta lactamase ,structure modification of resident beta lactamase, import of new beta lactamase, and target protein susceptibility. The reasons for the resistance to beta lactam antibiotics is in Gram positive cocci like MRSA changes in PBPs which are normally present in cellwall or acquiring insensitive beta lactam PBP. But in Gram negative bacteria it may be due to combination acquired beta lactamase endogenously with impermeability and efflux of the drug.

The beta- lactamases- **Murray - Manual of Clinical Microbiology[84] -** according to this text lactamase is a heterogeneous group of Penicillin recognizing proteins. They belong to the super family of active site serine proteases. The mechanism by which it act by cleaving an amide bond of beta-

lactam ring and form an acyl-enzyme complex. These enzymes can inactivate any beta lactam antibiotics. There are about nearly 170 enzymes of this kind.

Classification of Beta-lactamases[67] AmblerClassification

Class	Active site	Enzyme Type	Substrates	Examples
A	Serine	pencillinases	Benzyl, arboxy amino and ureido penicillins,narrow spectrum cephalosporins.	In staphylococcus aureus PC1
		Extended Spectrum **(ESBL)**	Broad spectrum substrates, oxymino beta lactams(Ceftazidime,Cefotaxime ,Ceftrioxone)and Monobactams.	In Enterobacteriaceae TEM,SHV derived,CTX-M derived,VEB-1,VEB-2,PER-1,GES-1,GES-2, IBC-2 in pseudomonas aeruginosa
		Carbapenamses	Extended spectrum with cephamycins and carbapenems.	KPC-1,KPC-2,KPC-3 in Klebsiella pneumonia
C		**Cephalosporinases**	Cephamycins with Extended spectrum substrates	AmpC type enzymes
D		Oxacillinases Broad spectrum	Amino and uriedopenicillins, cloxacillin, methicillin,oxacillin and some narrow spectrum cephamycins	OXA in pseudomonas aeruginosa
		Extended – spectrum	Broad spectrum substrates with oxymino beta lactams and monobactams	OXA-derived in P.aeruginosa
		carbapenamases	Extended-spectrum substrates with cephamycins and carbapenems.	OXA-derived in Acinetobacter
B	Metallo beta lactamases(zn^{2+})	carbapenemases	Extended-spectrum substrates with cephamycins and carbapenems.	IMP,VIM,

Functional Classification of beta lactamases by Bush-Jacoby-Medeiros

Group	Enzyme Type	Inhibition by Clavulinate	Molecular Class	Examples
1	Cephalosporinase	No	C	Enterobacter cloacaeP99(c)
2a	Penicillinase	yes	A	Bacillus cereus,Staphylococcus aureus (B)
2b	Broad -spectrum	yes	A	SHV-1(B),TEM-1(P)
2be	Extended - Spectrum	yes	A	Klebsiella oxytoca K1(C),TEM-3(P)
2br	Inhibitor resistant	Diminished	A	TEM-30(IRT-2)(P)
2c	Carbenicillinase	yes	A	AER-1(C)PSE-1(P)
2d	Cloxacillinase	yes	DorA	Streptomyces cacaoi(C) OXA-1(P)
2e	Cephalosporinase	yes	A	Proteus vulgaris (C) FEC-1(P)
2f	Carbapenamase	yes	A	IMI-1(C)NMC-A(C)
3	Carbapenamase	No	B	Stenotrophomonas maltophilia L1(C),IMP-1(P)
4	penicillinase	No		Burkholderia cepacia(C),SAR-2(P)

Types of beta lactamases[15,67,89] There are more number of beta lactamases and most of them are the derivatives of TEM or SHV enzymes.

TEM derived - Beta lactamase which is commonly encountered in gram negative bacteria is TEM -1 especially in Escherichia coli and Klebsiella pneumonia. The TEM derived beta lactamase was first reported in 1965 from Escherichia coli. In Escherichi coli Ampicillin resistance is commonly (90%) due to TEM-1. TEM 3 has increased activity against Extended Spectrum Cephalosporins and reported in 1988. TEM derived ESBL are susceptible to B lactamase inhibitors. Nowadays nearly 140 TEM type enzymes are available. In United States TEM-10, TEM-12, TEM-26 are common.

SHV (**S**ulp**h**ydryl**v**ariable)**derived**- an another important beta lactamase are primarily derived from Klebsiella species. SHV1 is resistant to broad spectrum penicillins not to oxyiminocephalosporinswhich is chromosomally encoded in most of the isolates of Klebsiella pneumoniae but in Escherichia coli it is generally plasmid mediated. Ampicillin resistance is due to plasmid mediated which accounts for 20% in this species. Nearly 60 SHV types have been described so far. This type is predominant in US and Europe. The most common types are SHV-5 and SHV-12.

CTX-M (Cefotaximase) derived – they are not related to TEM and SHV which acquire from chromosomal ESBL gene found in Kluyvera species a Gram negative rod found in the environment. These member hydrolyze 3rd generation Cefotaxime so that they were designated as CTX-M and it is better inhibited by Tazobactam rather than clavulinic acid. Nowadays CTX-M enzymes are most prevalent ESBL and CTX-M 15 is common in Escherichia coli.

OXA1 type is common in Pseudomonas aeruginosa. But this type is also seen in 1-10% of Escherichia coli. According to Ambler classification OXA belong to molecular classification class D and functional group 2d. They are commonly resistant to Ampicillin and Cephalothin and poorly inhibited by Clavulinic acid. ESBL phenotype is also expressed by this type, by amino acid subsititutions in OXA. **PER** type hydrolyze penicillins and cephalosporins and are inactivated by clavulinic acid. It was detected in Pseudomonas aeruginosa,

Salmonella enterica, E.coli and Proteus mirabilis. **GES type** resembles class A ESBL. GES-1, GES-2 normally found in South Africa whereas **BES-1,IBC-1,SFO-1,**and**TLA-1** are uncommon ESBL found only in Enterobacteriaceae.

Detection of beta – lactamases[76]- By various biochemical tests beta-lactamase enzymes can be detected. This test was mainly based on measuring Penicilloic acids which was produced when Beta-lactamases hydrolyse benzyl Penicillins. There are three methods by which the acid production was determined. **Acidometric method-** by measuring the change in pH of an indicator dye the acid production was detected. **Iodometric method**- based on the ability of Penicilloic acid to reduce iodine and reverse the formation of the blue colour when iodine complexes with starch. **Chromogenic Cephalosporin method-** Here Nitrocephin was used. Generally Nitrocephin was yellow in colour but when the beta-lactam ring was hydrolysed it turns in to red.

β -lactamase inhibitors[59]-These compounds structurally resemble Beta-lactam antibiotics. Reversibly or irreversibly they can bind to beta-lactam antibiotics by that they protect the antibiotics from destruction. They act as **suicide bombers** utilizing all available enzymes. These compounds also have weak antibacterial activity but they are potent inhibitors of most of the plasmid-encoded and some of the chromosome encoded beta-lactamases. There are three important beta-lactamase inhibitors. They are Clavulanic acid, Sulbactam and Tazobactam. Only low level of antibacterial action was present in Clavulanic acid but when combined with beta lactam antibiotics, bacterial

inhibition is enhanced which are otherwise resistant to beta-lactam antibiotics. Sulbactam has broader spectrum of inhibition but they are less potent. Tazobactam is as potent as Clavulanic acid.

Extended spectrum of β-lactamase- Enzymes which are capable of hydrolyzing major beta-lactam antibiotics including third generation Cephalosporins are called as Extended Spectrum Beta- Lactamases.

ESBL Definition: Jung Hun Lee[51] et al 2010 Korea-IDSA declared ESBL producing Enterobacteriaceae, Multidrug resistant Pseudomonas aeruginosa, Acinetobacter baumanii, MRSA, Vancomycin resistant Enterococcus faecium and among the fungus Aspergillus species are dangerous pathogens.In1987 the term Extended broad spectrum beta lactamases was introduced and they are the counterpart of broad spectrum. Beta lactamases which are plasmid mediated mediate resistance to Extended spectrum Cephalosporins and it was proposed in the year 1987. The word broad has been removed and ESBL was used from the year 1989. As per the **functional or the classic definition** suggested by Giske ESBLs are the enzymes which are able to hydrolyze Penicillins, Extended spectrum Cephalosporins, Monobactams and not able to hydrolyze Cephamycins or Carbapenams and inhibited by beta lactamase inhibitors hydrolyzed by ESBL. Up to this date there are three kinds of definitions for ESBL. According to that 1.classic **definition** beta-lactamases belong to Ambler class A and 2be of functional group,2 in **broadened definition of ESBL,** classical ESBLs, with non TEM and non SHV ESBLs, OXA type

ESBLs and AmpC type ESBLs are included but not carbapenamases 3. **in all inclusive definition** along with broadened definition of ESBL, Carbapenamases are included. According to all inclusion definition there are three classes ESBL 1) $ESBL_A$ named for class A ESBL which is further divided in to high and low prevalent ESBL. Different guidelines for detection of functional ESBL applicable to only $ESBL_A$ class.2)$ESBL_M$ (miscellaneous ESBL) further divided in to $ESBL_{M-C}$(class c plasmid mediated AmpC relavant to AmpC ESBL) and $ESBL_{M-D}$ (class D relavant to OXA –ESBL. Detection of pathogens that produce both $ESBL_A$ and ESBL $_{M-C}$. are difficult. Latter is common in Enterobacter, Serratia and Citrobacter because clavulinic acid inhibition on ESBL $_A$ is hidden by AmpC beta lactamase.3) ESBL $_{CARBA}$. – along with 1 and 2 it includes Carbapenamases. Livermore explained the limitation of all inclusive definition that is in general carbapenamase activity is not one of the feature of ESBL. He agreed $ESBL_A$ and $ESBL_M$. According to Bush also ESBLs are successfully treated by carbapenems. So that $ESBL_{CARBA}$ designation is not necessary. ESBL $_{M-C}$ $ESBL_{M-D}$ ESBL $_{CARBA}$ are not inhibited by betalactamase inhibitor. Finally Bush states that $ESBL_A$ is only included in the ESBL category.

Risk factors for ESBL-According to **Michael Osthoff[73] 2015 Australia** if ESBL producing organisms are resistant to three classes of antibiotics, Aminoglycoside, Trimethoprim-sulfamethoxazole and fluro quinolones, then they are considered as multiresistant. The important risk factors for ESBL-GNB UTI are recent overseas travel, repeated exposure of antibiotics

particularly in the previous 6wks, duration of stay in the hospital as inpatient, colonization of rectal and urinary tract, diabetes mellitus, immune suppression, cancer.

Mahesh[65] et al 2010 Bangalore India-Important risk factors such as past history of any genitourinary surgery and catheterization play a major role in acquisition of infection by the organism of particularly ESBL positive strains . Local immunity status of the urinary tract is disturbed by recent urological procedures. In diabetes mellitus secretion of local cytokine is decreased which lead to decrease in number of leukocyte by which natural host defence mechanism was lowered.

Beta Lactamases in Enterobacteriaceae Thenmozhi[112] et al 2013 India- Recently new antibiotic resistance are acquiring in the bacteria and we have been forced to fight against the new type of resistance. Different types of Beta Lactamases, particularly ESBLs are produced by the Enterobacteriaceae. Nowadays Proteus mirabilis produce ESBL commonly next to that of Escherichia coli and Klebsiella. Most of the ESBLs were derivatives of TEM-1 and 2 types, SHV -1 and CTX-M types. Usually multidrug resistance type of phenotypes is exhibited by the ESBL producing organism. **Antibiotic resistance** may be intrinsic or acquired. Mutations happening in the existing genetic material or acquiring new genetic element from other bacteria are the two important mechanism by which bacteria prevent the antibiotic effect. Naturally Escherichia coli are susceptible to Ciprofloxacin and Ampicillin but

nowadays they are resistant to the above drugs. Ciprofloxacin resistance is due to the mutation of existing genes and Ampicillin resistance due to acquisition of beta lactamase coded gene. Enterobacteriaceae group of organism are able to produce AmpC and ESBL. In Enterobacter, Providentia, Citrobacter,and Serratia AmpC production is common. But ESBL are commonly produced by Escherichia coli, Klebsiella and Proteus. On exposure to antibiotics AmpC production are induced. The strongest inducers are Penicillins, first generation Cephalosporins, Cefoxitin and Carbapenems. In Enterobacter hyperproduction of AmpC type 1 beta lactamases are seen, but in Klebsiella spp, Escherichia coli,and Proteus there is no hyper production but acquire beta lactamases AmpC and ESBLs through plasmid mediated of which ESBL is more common. There are certain basic differences between Ampc and ESBLs such as AmpCs are not derivatives of TEM and SHV(parent beta lactamases) , inactivate cephamycins, not inhibited by beta lactamase inhibitors such as Clavulinic acid.

Dissemination of ESBL- Alma Brolund[3] et al 2013 Sweden- Global epidemiological survey through different surveillance regarding resistance is important to detect bacterial strains with new type of resistance and also very helpful in gaining knowledge about emerging clones. Two important mechanisms by which ESBL dissemination happening and they are **Reservoirs of resistance gene** and **Clonal expansion.**

Prevalence of ESBL Yong Chong[119] et al 2013 Japan -During the early 1980, ESBLs were detected in Europe and it slowly disseminated throughout the world. Klebsiella pneumoiae was a frequent ESBL producer till 1990 and it was the most important organism responsible for nosocomial outbreaks. During 21st century only ESBL producing Escherichia coli increased its number. Compared to other regions, in Asia ESBL producing isolates are greater in number. According to 2007 studies the prevalence of ESBL exceeds 30%. One study of Japan showed that prevalence of ESBL steadily increasing. In 2003 data it was 5.41% in Escherichia coli and 0.87% in Klebsiella but in 2009 in Escherichia coli it was 17.12% and for Klebsiella it was about 10.47%

METHODS OF ESBL DETECTION [24,34,42,]

Several phenotypic methods are available to detect the ESBL production. Among the various phenotypic methods some of them are discussed below.

a. Double-disk approximation test [34,42]

n Muller – Hinton agar plate Organism is swabbed. An antibiotic disk containing one of the Oxyimino beta-lactam antibiotics is placed 20mm (centre to centre) from the Amoxicillin –Clavulanic acid disk. If there is an any enhancement of zone of inhibition of the Oxyimino beta-lactam towards the Clavulanate present in Amoxy-clav disk indicates the ESBL positive[43, 45].

b.Three Dimensional test[24]

The main advantage of this test is simultaneous determination of antibiotic susceptibility and beta- lactamase substrate profile. Two types of inoculums are prepared.

Inoculum-1: contains $10^9 – 10^{10}$ CFU/ml of active ESBL producers.

Inoculum-2: Contains 0.5 Mc Farland Std. (150 million organisms/ml)

Plate is inoculated as for disc diffusion procedure with inoculum - 2. In the inoculated plate a circular slit was cut on the agar 4mm inside the position at which the antibiotic discs were placed and inoculum1(10^9-10^{10}CFU/ml) was poured into it. Any distortion or discontinuity in the circular zone of inhibition is interpreted as positive for ESBL production.

E test: Prabha[93] et al 2016 Pondicherry

Bacterial susceptibility to the antibacterial agents can be quantitatively determined by this E- test. Determination of MIC in microgram per ml for various antibacterial agents against bacteria is possible by this method.

Features and advantages of E-test[93] (Ezy MIC™ strip HIMEDIA**)** Ezy MIC™strip is made up of porous material**.** MIC values and antibacterial agents are distributed on both sides of the strip so that it can be placed on the agar surface by any side. Within 60 seconds strip was absorbed due to its porous nature. Proper method of reading of MIC values by without opening the lid of MH plate. Here for the detection CTX/CTX+ and CAZ/CAZ+ are used. CTX codes for Cefotaxime 0.25-16µg/ml and CTX+ codes for Cefotaxime0.016-

1μg/ml plus 4 μg/ml of Clavulinic acid. CAZ codes for Ceftazidime 0.5-32 μg/ml and CAZ+ 0.064-4 μg/ml plus Clavulinic acid. E test ESBL strips have 2 gradients i.e on one end CTX or CAZ and on the opposite end CTX+ or CAZ+. MIC is the point of intersection of the inhibition ellipse with the E-test strip edge. Ratio of CT MIC and CTL MIC $\geq$ 8 indicates presence of ESBLs.

Phenotypic Confirmation Test [23,59]- First Lawn culture was made on MHA plate with test organism of 0.5 Mac Farland's standard and 3rd generation cephalosporin, Ceftazidime (30μg) disc was tested alone and along with their combination for 10mg of Clavulanic acid. If there is 5mm increase in zone of inhibition for Ceftazidime / Clavulanic acid (30μg/10μg) are confirmed as ESBLs. (CLSI recommends MIC $\geq$ 2μg/ml for Cefotaxime, Ceftazidime, Aztreonam, Ceftriaxone (or) Cefpodoxime as potential ESBL producers).

Two indicators of ESBLs are

1. 4 fold reduction in MIC when 3 Generation Cephalosporins are used with Clavulanic acid.
2. 5mm increase in diameter of Zone of inhibition when using disc diffusion method with 3rd generation Cephalosporin alone and combination with Clavulanic acid.

Koneman's[59] Text Book of Diagnostic Microbiology Sixth edition according to this Chromogenic agar is one type of media in which artificial substance like chromogens are incorporated in the media. Chromogens are hydrolysed by specific microbial enzymes and produce specific coloured compounds. It was

first designed by H.Killian and Bulow in order to identify the Escherichia coli in urine. Nowadays chromogenic media are used for the presumptive identification of bacteria and enzyme producing strains. By using this media there was reduction in inoculation time >50% and reduction in work up time >20%.

He´ le`ne Re´ glier-Poupet[43] et al 2008- Chrom ID medium contains antibiotics for inhibition of Gram positive bacteria and also contain Cefpodoxime which is a marker for ESBL resistance mechanism. CHROM agar also inhibits yeast. Urine sample is directly inoculated and incubated for 24hrs to 48 hrs. A colour chart, provided by the manufacturer is used for identification of ESBL strain. According to that chart ESBL producing Escherichia coli pink or burgundy Proteae tribe light to dark brown Klebsiella,Citrobacter ,Serratia and Enterobacter groups blue or green in colour.

Kjersti Sturd[58] et al 2013 Norway-Generally Chrom agar contains different chromogenic substances targeting different enzymes generally beta-galactosidase or beta glucuronidase and deaminase.

Detection of ESBL among AmpC producers Deepika Handa[27] et al 2013 Meerut India -In this study cefoxitin disk was used as a screening agent for AmpC production. Isolates which showed resistance to cefoxitin (zone of inhibition is less than 18mm) were considered as screen positive for AmpC production.In this study they used two methods IBM and M3D for the

detection of AmpC(Manchanda and Singh).The isolates were considered as AmpC producers if there is any distortion in zone of inhibition for cefoxitin and non producers when there is no distortion in zone of inhibition.

Paul R. Ingram[86] et al 2011 Australia-Tris-EDTA test otherwise called as AmpC disc test was used for the detection of AmpC production. Antibiotic discs supplemented with boronic acid and cloxacillin were used for inhibitor based test because both the compounds inhibit AmpC activity.

Jaspal kaur[45] et al 2016 - Jalandhar, India - Compared to clavulinic acid tazobactam and sulbactam are less likely to induce AmpC beta lactamases. In the presence of AmpC beta lactamases, Cefepime is used for the detection of ESBL because it is minimally affected by AmpC betalactamase. In Modified double disk synergy test Cefepime and Piperacillin-Tazobactam are used. In this test PTZ disc was placed at a distance of 22-25mm from cefepime disc and also disc of AMC (augmentin) was placed in MHA with cefotaxime, cefpodoxime, ceftazidime,and cefepime at a distance of 16-20mm from it .If the isolate shows synergism for only cefepime and PTZ then it was considered as ESBL positive.

Sasirekha Bakthavatchaluet[96] al 2013 Bangalore India -Multiple beta lactamases are produced due to the inappropriate use of beta lactam antibiotics particularly cephalosporin leading to therapeutic failure for beta lactam or beta lactam with beta lactamase inhibitors. For the detection of ESBL CLSI established confirmation methods. But for AmpC production there are several

methods for confirmation but there is no standard guide lines by CLSI for the confirmation of AmpC production. In the presence of AmpC, detection of ESBL is not possible by routine CLSI PCT (phenotypic confirmation test)) method. This is because Clavulinc acid induces the chromosomal AmpC expression in high level and this masks the synergy arising from inhibition of an ESBL. So if the strain containg both ESBL and AmpC it results in false negative test for ESBL detection.In this study important substance used for the detection of AmpC is boronic acid. For the detection of AmpC, cefoxitin and cefoxitin with boronic acid was used and with three dimensional disk method it was confirmed.

Molecular detection methods: Tests previously described only presumptively identify the presence of ESBL. For studying ESBL earlier determination of iso-electric point was sufficient. But nowadays s there are more than 90 TEM type and 25 SHV type of beta lactamase and many of them have same iso-electric point, so it has become impossible to detect the individual ESBLs. PCR is the easiest and most reliable molecular method used to detect ESBLs with oligonucleotide primers which are specific for a beta-lactamase gene. These primers can be chosen from sequence available in Gene Bank.

Medical significance of detection of ESBL[59]- Increased risk of treatment failure is common with expanded spectrum beta-lactam antibiotics in patients with infection caused by ESBL producing organism. If the organism is confirmed as ESBL producer then it is considered as resistant to all 3rd

Generation Cephalosporins. Many ESBL isolates will not be phenotypically resistant; even through their MIC is so high. Epidemic diseases are produced by the ESBL producing strains especially in Intensive Care Units and failure to control the outbreaks has resulted in new mutant types in some institution.

Treatment for ESBL- Eshwar singh[33] et al, Kelley E[56] Martinet al 2015 and Dominick[30] J 2015 **C**arbapenems are most effective and reliable treatment for the infection caused ESBL strains. Due to the presence of Trans 6 – hydroxy ethyl group they are highly resistant to the hydrolytic activity of all ESBLs. Amino glycosides and fluoroquinolones may be used alternatively if they show in vitro activity. A Beta- lactam and Beta-lactamase inhibitor combination such as Cefeperazone-sulbactum and Piperacillin Tazobactam may also be a further option to consider[48] even though clinical data for their use are absent. For these agents susceptibility pattern varies among ESBL producers so it should be used with caution. Cephamycins, such as Cefotetan and Cefoxitin although active in vitro they are not recommended for treating such infections, because of the relative ease with which these strains decrease the expression of outer membrane proteins, rendering them resistant.In urinary tract infection combination with Beta lactamase inhibitor such as Clavulanic acid can be used[41].

Prevention and control measures Jaumana[49]N et al 2003,-In order to prevent spreading and outbreaks of ESBL producing bacteria Proper infection control practices and barrier methods are essential. Other practices that reduce the occurrence of ESBL's are, controlling the rational use of antimicrobial drugs in the community, hospital and veterinary settings and also to Support the antimicrobial surveillance programmes both at local and national levels.

MATERIALS AND METHODS

Materials and Methods

The present study was conducted in Government Rajaji Hospital, Madurai Medical College, Madurai. Ethical committee clearance from the Institution was obtained and before collecting the specimens, informed written consent was obtained from the patients.

Study Period: September 2015 to August-2016

Study Population: Patients attending as op and in wards of various departments like Medicine, Surgery, Nephrology, Paediatrics, Urology, STD, Obstetrics and gynaecology at Government Rajaji Hospital, with fever, dysuria, frequency, urgency, lower abdominal pain / flank pain and supra pubic tenderness that are suggestive of upper and lower Urinary tract infections were considered and included in the study.

Sample Size : 400 urine samples

Study Centre: Government Rajaji Hospital and Institute of Microbiology, Madurai Medical College, Madurai.

Inclusion criteria :

1. The Patients with symptoms of UTI of all age groups
2. Patients with symptoms of UTI attending op and in wards of various departments.
3. Catheterized patients with symptoms of UTI like flank pain, fever.

Exclusion criteria

1. Patients with UTI but without any symptoms
2. Patient with prior antibiotics
3. Catheterized patients without symptoms of UTI
4. Severly ill Patients
5. Pregnant women.

Specimen Collection[14,59,75]

Patients from various departments with symptoms of urinary infections were instructed to collect clean catch midstream urine (CCMSU) in a sterile, dry, and wide mouthed leak proof screw capped container. Before the collection of urine sample the following instruction was given to male and female patients.

For females; Patients were advised to wash their hands and cleanse the genital area with soap and water and dry the area with sterile gauze pad. Patients were asked to hold the labia apart and asked to collect 10-20ml of Clean Catch Midstream Urine (CCMSU) in a sterile container.

For males – Patients were advised to clean the glans penis with soap and water then completely rinse with clean water. They were advised to retract the fore skin and asked to collect 10-20 ml of Clean Catch Midstream Urine in a sterile container.

For catheterized patients – clamping to be done above the catheter port and the collecting port was disinfected with 70% ethanol. By using sterile syringe and needle 5 to10 ml of urine was aspirated.

Supra pubic aspirate [59]– for this procedure bladder must be full and skin over the bladder site was disinfected before the procedure. Urine was collected directly by a sterile syringe with needle inserted percutaneously just above the pubis. This procedure is mainly for the infants.

Specimen transport - collected urine specimen was transported to the laboratory within one hour. If there is any delay in transport specimen was refrigerated at 4-6ºC.

Processing of sample[14,75]

Macroscopy- Initially macroscopic examination was done for the collected Urine specimes for the presence of colour, turbidity and deposits. All samples were subjected for initial screening methods like wet mount preparation and Gram Staining.

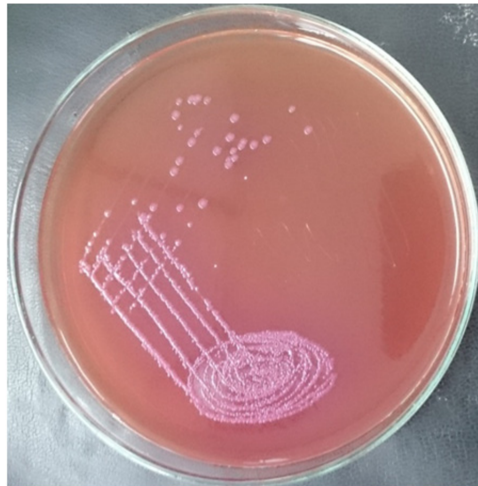
Microscopy

Direct Gram Staining - A smear was made from a drop of well mixed un centrifuged urine sample in a clean glass slide which was air dried heat fixed , stained and examined under oil immersion objective lens. Presence of 1to 5 bacteria per oil immersion field generally correlated with significant bacteriuria ($\geq 10^5$CFU/ml). Presence of Pus cells were examined and its presence taken as definite indication of UTI[34].

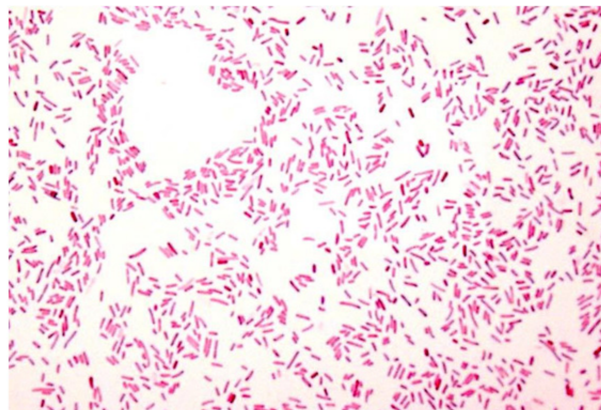
Wet mount preparation - One drop of well mixed uncentrifuged urine sample was placed at the centre of the cleaned glass slide and cover slip was placed

Semi Quantitative Culture in Blood Agar Plate

Semi Quantitative Culture in Mac Conkey Agar Plate



Gram Negative Bacilli



over the drop. It was examined for the presence of pus cells under 10X , and pus cell count more than 8/mm^3 was correlated with pyuria.

Culture - Before inoculation, the urine sample was thoroughly mixed. The calibrated loop which delivers 0.001ml of urine volume was flamed and cooled. It was vertically inserted in to the sample container. The centre of the culture plate (Nutrient agar, Bloodagar and MacConkey agar and CLED) was touched with the loop containing fixed volume of sample. From the point of inoculation it was spreaded initially by drawing vertical line across the diameter of the plate without any intermittent heating. In order to produce isolated colonies the loop was drawn across the entire surface of the culture plate by crossing the primary streaking several times and inoculated plates were incubated for 24hrs at 35ºc.

Interpretation of culture[14]-With the help of hand lens, the inoculated culture plates were examined after 24hours for the growth of organisms, colonies were counted on each plate. To determine the number of microorganisms per ml in the original specimen the number of colonies in the culture plate was multiplied by 1000. Interpretative criteria may vary according to the type of urine i.e clean catch mid stream, Catheterized, or Suprapubic specimen. The interpretation of the culture was done according to the following table given in ref[14.]

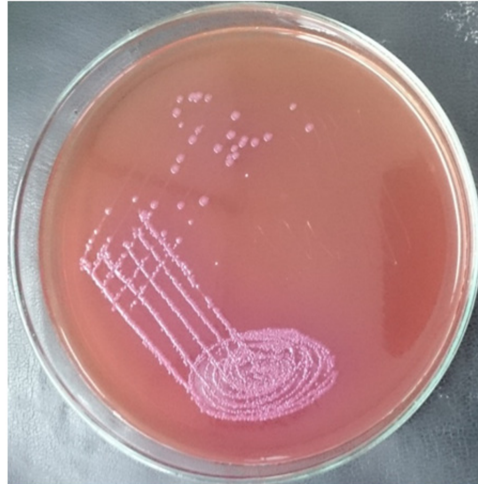
Result	Specimen type and clinical condition	Processing of sample
If CFU/ml is ≥ $10^4$of a single potential pathogen or two potential pathogens	CCMS/acutecystitis, pyelonephritis,or catheterized urines.	Complete processing of the sample to be done.
If CFU/ml is ≥ $10^3$of single potential pathogen.	CCMSurine/symptomatic male, acute urethral syndrome, or catheterized urines.	Complete processing of the sample to be done.
If ≥ three type of organisms without predominating type of organism.	CCMS urine or catheterized urine.	Possibility of contamination so no need for processing.
If there is two or three types of organism with one predominant type and≤ 10^4CFU/ml of other types of organisms.	CCMS	Complete processing of the sample to be done only for the predominant type of organism.
If there is ≥ $10^2$of any number of organism types	Suprapubic aspirates or surgically obtained during (ileal conduits, cystoscopy)	Complete processing of the sample to be done.

Identification of Bacteria

For the identification of Enterobacteriaceae the isolated bacteria from the culture media was subjected to the following tests.

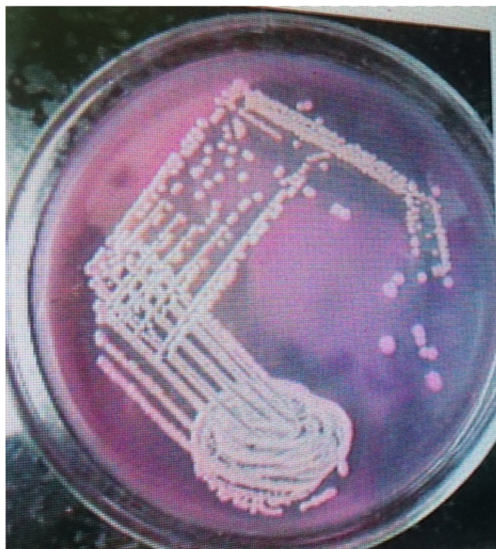
- Gram staining
- Demonstration of motility by Hanging drop method
- Standard biochemical reactions(Standard biochemical reactions. (Catalase test (Tube method), Oxidase test, Nitrate reduction test, Indole test Methyl Red test (MR Test) ,Voges Proskauer test (VP TEST),Citrate utilization test Triple Sugar Iron agar, Urease test, Oxidative – Fermentative test (OF TEST) Decarboxylase test (LAO TEST),Phenylalanine Deaminase Test, Sugar Fermentation test).

Escherichia coli in Mac Conkey Agar Showing Lactose Fermenting Colonies



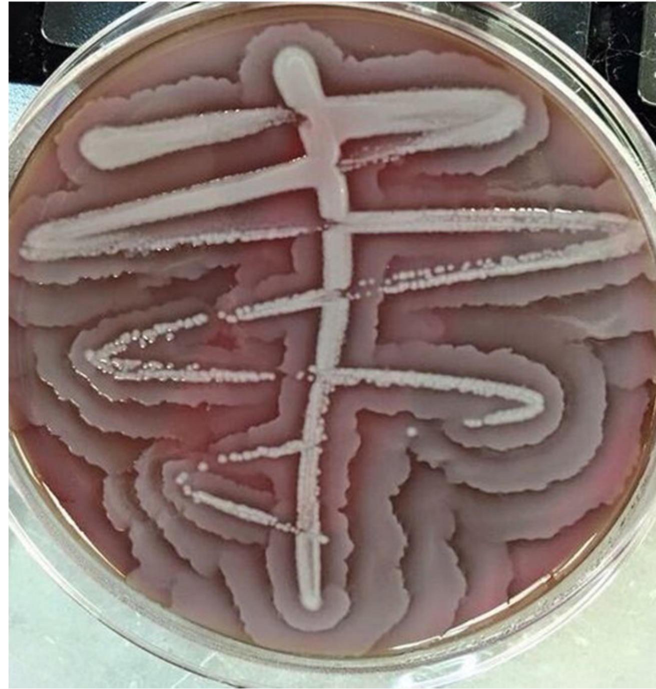
Escherichia coli-Biochemical Reactions

Klebsiella Pneumoniae in Mac Conkey Agar Showing Lactose Fermenting Colonies

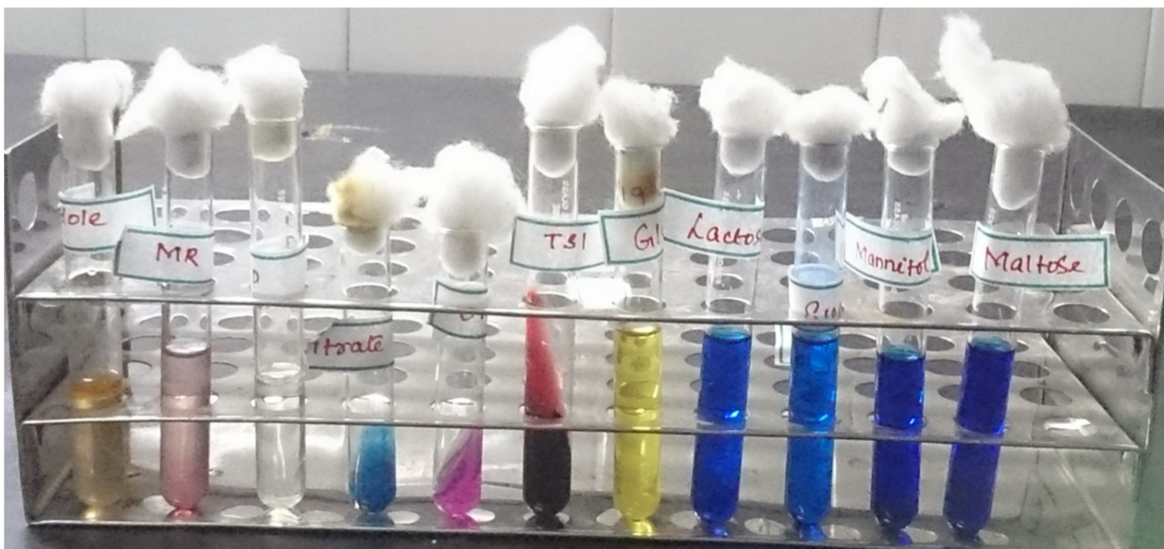


Klebsiella Pneumoniae - Biochemical Reactions

Proteus mirabilis in Blood Agar Showing Swarming



Proteus mirabilis – Biochemical Reaction



Antimicrobial sensitivity testing[53]

As per CLSI 2016 guidelines using antibiotic discs (Hi-media, Mumbai),the antimicrobial sensitivity pattern for all the isolates isolated from significant bacteriuria were done in Mueller Hinton Agar (MHA) by modified Kirby – Bauer disc diffusion method.

Preparation of inoculum :

4 to 5 well isolated representative colonies were taken from the 24 hrs culture plate with the help of a sterile loop and transferred to a test tube containing 4-5ml of sterile peptone water and incubated for 2-6 hrs at 35◦C. Then the turbidity was adjusted to 0.5 McFarland standards. This is done by holding both the standard and inoculum tube side by side and no more than one inch from the face of the Wickerham card (with adequate light present). This inoculum was used for sensitivity testing.

Inoculation of MHA plates

A sterile cotton swab was dipped in to the inoculum and with firm pressure the swab was rotated several times inside the wall of the tube to remove the excess broth from the swab. Then the entire dried surface of Mueller Hinton agar plate was inoculated by streaking with the swab. This procedure was repeated by rotating the plate two more times by rotating the plates at 60 degree to ensure an even distribution of inoculums. Finally, the rim of the agar was swabbed. The lid was replaced and left for 3-5 minutes to allow any excess moisture to be absorbed. Within 15 minutes the antibiotic discs were applied.

Control strains used with each batch

i. Escherichia coli ATCC 25922

ii. Staphylococcus aureus ATCC 25923

Antibiotic sensitivity test

According to CLSI guidelines all the isolates were tested with predetermined battery of antibiotic discs (HIMEDIA, Mumbai)of Ampicillin, Gentamicin Amikacin, Cotrimoxazole Nitrofurantoin, Norfloxacin, Levofloxacin, Cephalexin, Cefuroxime, Ceftazidime, Cefotaxime, Cefoxitin, Cefepime, Amoxyclav and Imipenam. Along with the above drugs Erythromycin and Vancomycin were tested for Gram positive cocci. Piperacillin-Tazobactum and Cefeperazone-Sulbactum were used only for Enterobacteriaceae.

Application of discs to inoculated Muller Hinton agar plates [59]

With the help of forceps, the antibiotic disks were placed on agar plates. In order to ensure the complete contact of the disk with the agar surface disks were pressed down. Discs were distributed evenly so that they were not closer than 24 mm from centre to centre of the disc and incubated at 37° C for 16 – 18 hrs.

Reading of AST and interpretation of results

After overnight incubation, each plate was examined. With the help of antibiogram scale around each disks the zones of complete growth inhibition including the diameter of the disk was measured. The zones were measured to the nearest millimeter. For measuring the size of the zone, ruler was held on the

back of the Petri dish. The Petri dish was viewed with reflected light against a black non reflecting background. With unaided eyes if the zone margin shows no obvious visible growth it was considered as a zone of inhibition. According to CLSI standard the sizes of the zones of inhibition were interpreted and reported as 'susceptible', 'intermediate' or 'resistant' to the drug.

Screening for ESBL production[22]

For ESBL detection Quality control Klebsiella pneumoniae ATCC 700603(ESBL positive) Escherichia coli ATCC 25922 (ESBL negative).

1. Modified Kirby Bauer disc diffusion method

According to CLSI isolates showing zone of inhibition ≤ 22mm with Ceftazidime(30 μg) and ≤ 27mm with Cefotaxime(30 μg)were interpreted as probable ESBL producers. For the confirmation of ESBL production different phenotypic methods were used. Here the methods used were

1. Double Disk Synergy Test (DDST)
2. ESBL CHROM Agar
3. Phenotypic Confirmation Test (PCT)
4. E –Test

1. Double disc synergy test[42,93]

This test mainly used to demonstrate a synergistic action of 3rd generation Cephalosporin or Monobactam with Clavulanic acid. Inoculum was prepared as said above and with the help of sterile swab, lawn culture was made on MHA plate. Two different third generation cephalosporins Cefotaxime CTX(30μg) and Ceftazidime CAZ(30μg) were placed at a distance of 20mm centre to centre

from the Amoxicillin Clavulanate (AMC20µg/10µg) and incubated at 37°C for 16 – 18 hrs. Enhancement of zone of inhibition to any one of the third generation antibiotic disk on the side of the disk containing clavulanate was interpreted as ESBL producer.

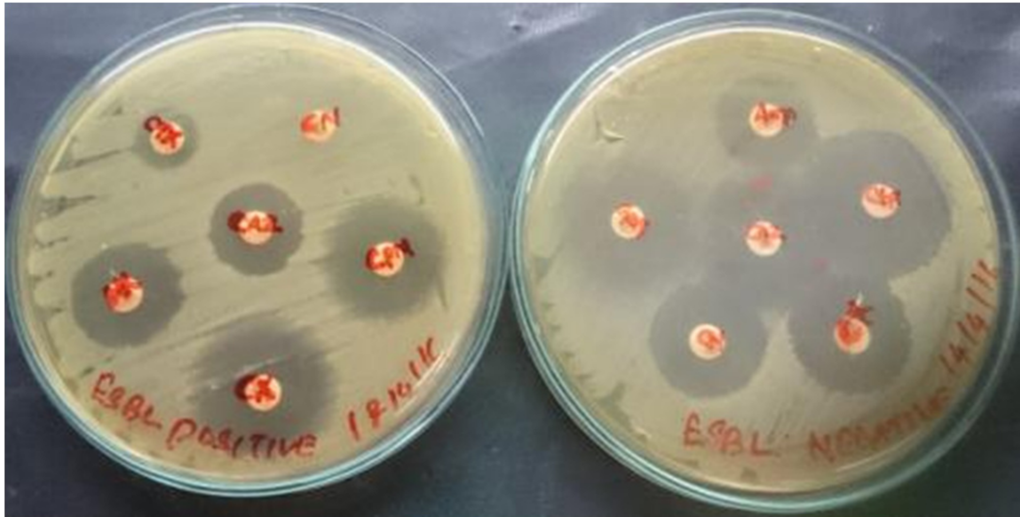
2. ESBL CHROM Agar[43,58,82]

Chrom agar consists of nutritive base, Chromogenic substrates with mixture of antibiotics including Cefpodoxime enable the growth of ESBL producing Enterobacteria. Ready prepared ChromID™ ESBL Agar plate from Biomerieux was inoculated. It was incubated with the cover bottom side at 37ºc for 18-24hrs.

Principle of this CHROM agar medium is the use of chromogenic substrates revealing metabolic enzymes specific for certain species of bacteria

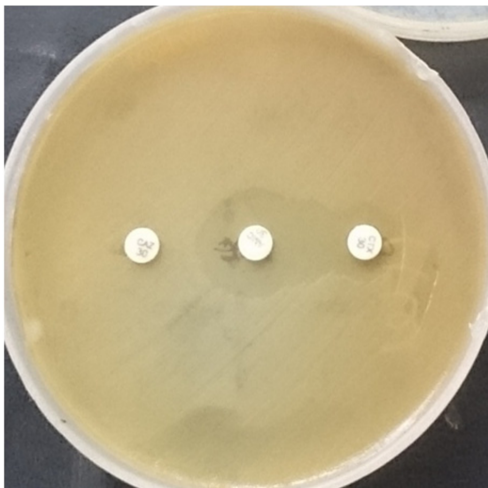
Escherichia coli – Spontaneous pink to burgundy coloration of strains expressing beta glucuronidase. Klesiella ,Enterobacter, Serratia, Citrobacter- spontaneous green, brownish green, or blue colouration of the strains expressing a beta-glucosidase. Proteeae (Proteus, Providencia, Morganella)- spontaneous dark brown to light brown coloration of strains expressing deaminase.

ESBL SCREENING

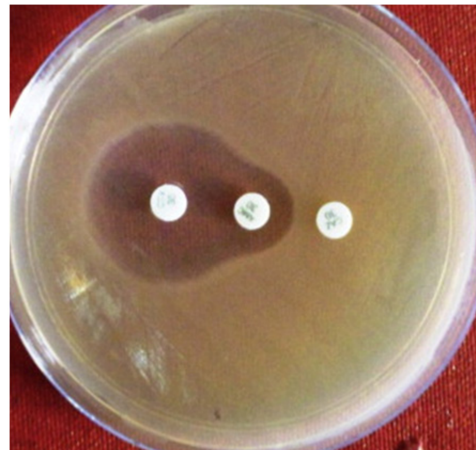


DOUBLE DISC SYNERGY TEST

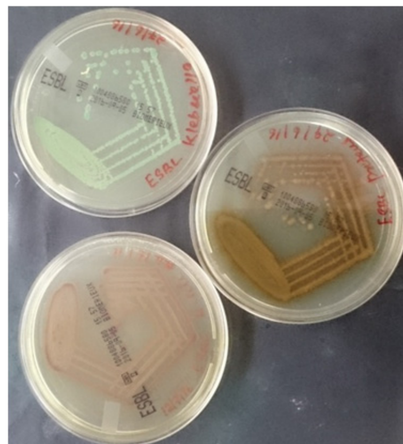
E.coli



Proteus mirabilis



ESBL CHROM agar (showing E.coli, (Pink) Proteus mirabilis(brown) Klebsiella spp(green))



Interpretation done according to the instruction given by manufacturer.

MICROORGANISMS	TYPE OF COLONY
ESBL Escherichia coli	Pink to burgundy colour
ESBL Klebsiella, Enterobacter, Citrobacter, Serratia	Green or Blue colour
ESBL Proteus species	Dark brown to light brown
Non ESBL strains	Inhibited

3. Phenotypic confirmation Test (PCT)[23]

Using a sterile cotton swab that was soaked with broth, a lawn culture was made onto the dried surface of Mueller –Hinton agar (MHA). The plates were allowed to dry for 15min. Cefotaxime(30µg), Cefotaxime-clavulanate(30µg/10µg) ceftazidime(30µg) and ceftazidime-clavulanate (30µg/10µg) were placed on to the inoculated MHA plate at a distance of 20mm. Then incubation was done at 35◦C for 16-18 hrs. The zone diameter was recorded and interpretation was done as per CLSI guideline.

Interpretation

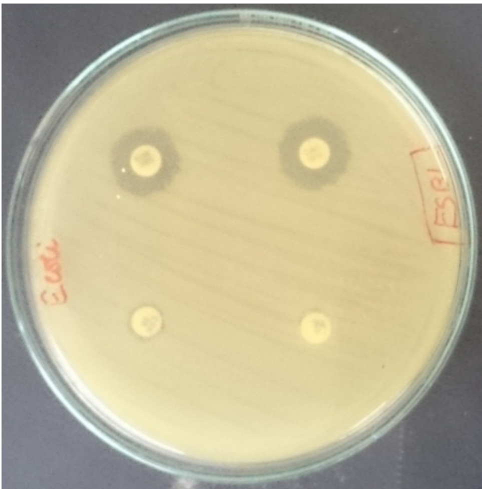
A ≥ 5mm increase in the zone diameter for either antimicrobial agent tested in combination with clavulanate vs its zone diameter of the agent when tested alone that isolates are regarded as ESBL producing bacteria.

4 E-test ESBL[93](Ezy MIC™ strip HIMEDIA)

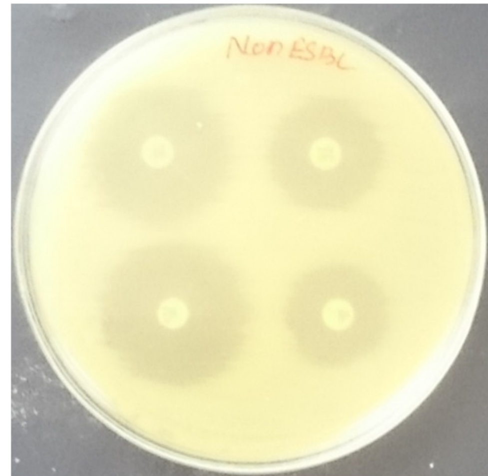
By using E-test strips the both disc diffusion and Minimum Inhibitory Concentration (MIC) were studied. All test isolates were tested with the E-test strip containing Ceftazidime gradient at one end and Ceftazidime plus Clavulanate gradient on the opposite end, also Cefotaxime at one end and Cefotaxime plus clavulanate at another end. Before the procedure the E-test

PHENOTYPIC CONFIRMATORY TEST (PCT)

ESBL

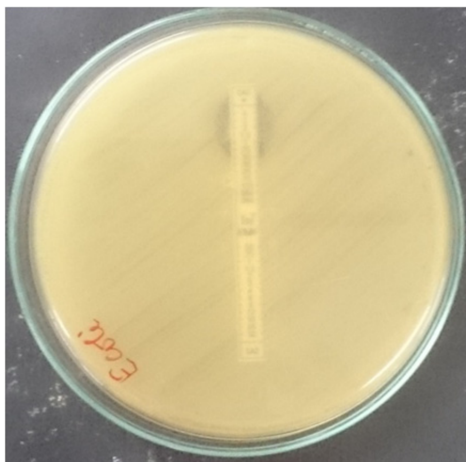


NON ESBL

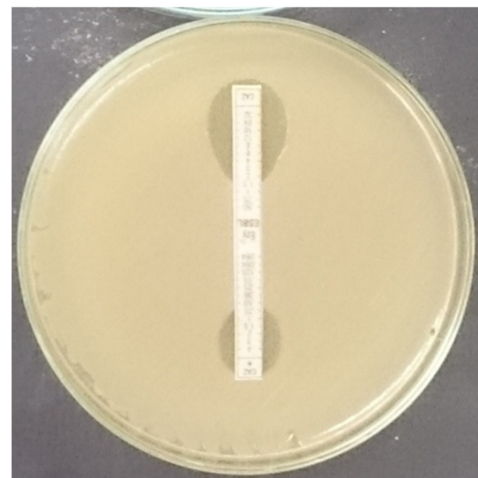


E-test

ESBL



NON ESBL



strips were brought to room temperature. MHA were inoculated as for disc diffusion and with the help of forceps the E-test strip was placed over the agar surface and incubated for 35º-37ºc MIC was the point of intersection of the inhibition ellipse with the E-test strip edge. When the ratio of the value obtained for Ceftazidime(CAZ): the value of Ceftazidime in combination with Clavulanic acid is more than 8 interpreted as ESBL positive strain . Also when the ratio of the value obtained for Cefotaxime(CTX): the value of Cefotaxime in combination with clavulinic acid is more than 8 interpreted as ESBL producer. For both if the value is less than 8 it is interpreted as ESBL negative strain.

Finally for the ESBL producing Enterobacteriaceae the sensitivity pattern is noted.

Molecular characterization of ESBL producing Enterobacteriaceae

Phenotypically confirmed ESBL positive isolates were further processed in HELINI Biomolecules, Chennai to detect the presence of beta lactamase encoding genes of family TEM, SHV and CTX-M (Cefotaxime). DNA was extracted by using pure fast® bacterial DNA purification kit. 2X PCR Master mix contained 2U of Taq polymerase,10X Taq reaction buffer, 2mM $Mgcl_2$, 1µl of 10mM dNTPs mix and Red dye PCR additives. Agarose gel electrophoresis was performed with agarose, 50X TAE buffer, 6X gel loading buffer and Ethidium bromide.

Bacterial DNA purification procedure

1ml of overnight culture was centrifuged at 6000 rpm for 5minutes and supernatant was discarded. Pellet was suspended in 0.2ml PBS. 180µl of lysozyme digestion buffer and 20µl of Lysozyme(10mg/ml) was added and

incubated at 37ºC for 15min. 400µl of binding buffer, 5µl of internal control template and20µl of Proteinase K were added and it was mixed well by inverting several times. This mixture was incubated at 56ºC for 15min and 300µl of ethanol was added and mixed well By using pipette, the entire sample volume was transferred to pure fast spin column and centrifuged for 1 minute. Flow through was discarded and 500µl of wash buffer-1 was added. It was centrifuged for 1minute. Flow through was discarded and 500µl of wash buffer-2 was added. Then it was centrifuged at for additional 1 minute.

The flow through was discarded and the column was centrifuged for additional 1 minute to remove any residual ethanol. The Pure fast® spin column was transferred into a fresh 1.5ml micro centrifuge tube. 100µl of elution buffer was added to the centre of Pure fast® spin column membrane and again incubated for 1 minute at room temperature and centrifuged for 2 minutes.

PCR Primer

TEMF-GATAACACTGCGGCCAACTT

TEMR-CTGCAACTTTATCCGCCTCC

SHVF-CGCCGCCATTACCCATGACGCGAT

SHVR-ACCCGATCGTCCACCATGCCACT

CTXF-ACGTGGCGATGAATAAGCTG

CTXR-AACCCAGGAAGCAGGCAGTC

PCR amplification :

The PCR reactant mixture for each sample is prepared by adding 10µl of PCR master mix, 5µl of primer mix and 5µl of purified DNA of each sample to a total final volume of 20µl.

PCR Procedure:

20µl of the PCR reactant mixture was mixed gently, spin down briefly and placed into PCR machine. It was programmed as follows:

Initial Denaturation : 95ºCfor5min

Denaturation : 94ºCfor30sec

Annealing : 58ºCfor30sec

Extension : 72ºCfor30sec

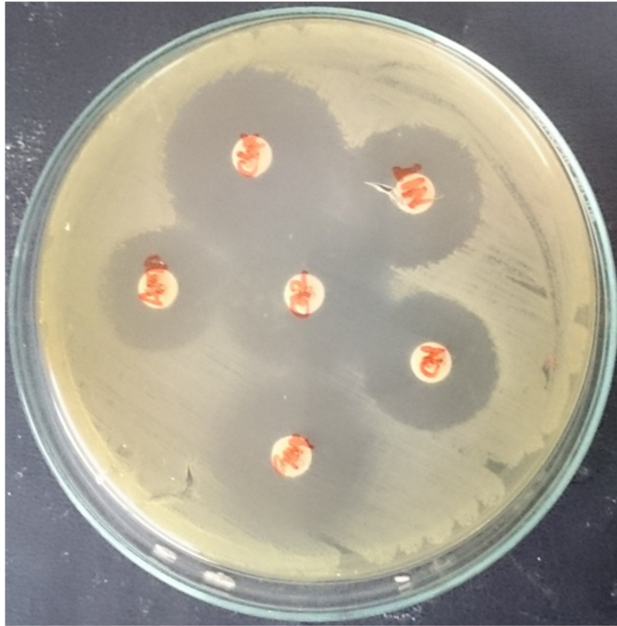
} 35cycles

Final extension : 72ºCfor5min

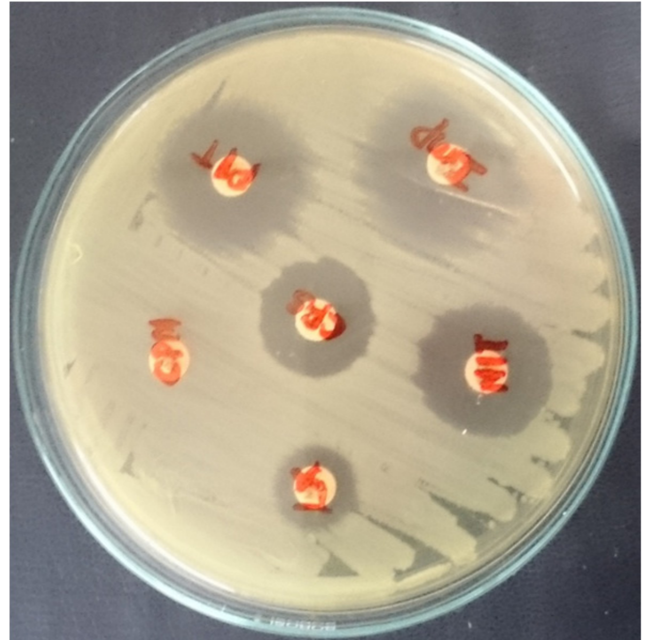
Loading: 2% agarose gel was prepared by mixing 2gm of agarose in 100ml of 1XTAE buffer. 8µl 6X Gel loading dye was added to each PCR vial and 5µl of PCR sample was loaded. After that run electrophoresis at 50V till the dye reaches three fourth distances. The bands were observed in UV transilluminator.

Agarosegel electrophoresis: 2% agarose was prepared by the addition of 2gm agarose in 100ml of 1X TAE buffer (melted using microoven).When the agarose gel temperature was around 60°C,5µl of Ethidium bromide was added. Warm agarose solution was poured slowly into the gel platform. The gel set was kept undisturbed till the agarose solidifies. 1X TAE buffer was poured into submarine gel tank. The gel platform was placed carefully into tank. The tank buffer level was maintained 0.5cm above than the gel.PCR Samples were loaded after mixed with gel loading dye along with 10µl of 100bp DNA Ladder. (100bp,200bp, 300bp,400bp, 500bp, 600bp, 700bp, 800bp, 900bp,

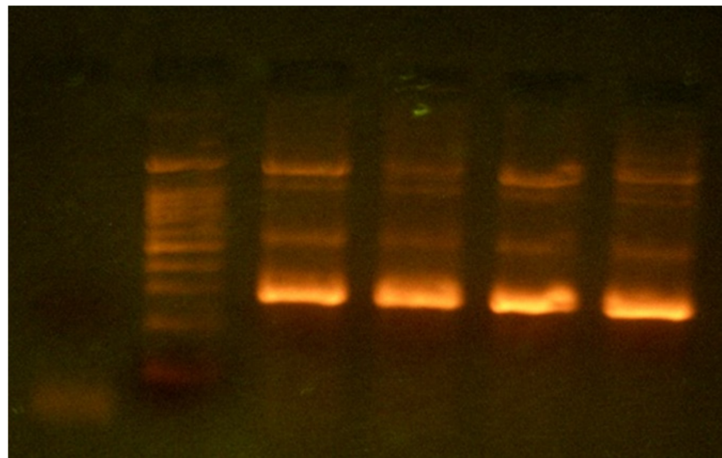
Antibiotic of Choice for NON ESBL Producers



Antibiotic Of Choice For ESBL Producers



ESBL Strain Showing TEM, SHV, CTX-M Genes



1000 and 1500bp). Then electrophoresis was run at 50 V till the dye reaches three fourth distance of the gel. Gel was viewed in UV trans illuminator and observed the bands pattern.

INTERPRETATION:

The presence of TEM, SHV and CTX-M genes were indicated by the amplification of 250bp 276 bp and 296bp PCR product from the clinical isolates respectively.

STATISTICS: For statistics soft ware SPS 16 was used. UTI Prevalence age and sex distribution, ESBL prevalence were expressed in percentage. Chi Square method was used for comparison of the four Phenotypic methods for detection of ESBL among Enterobacteriaceae.

RESULTS

RESULTS

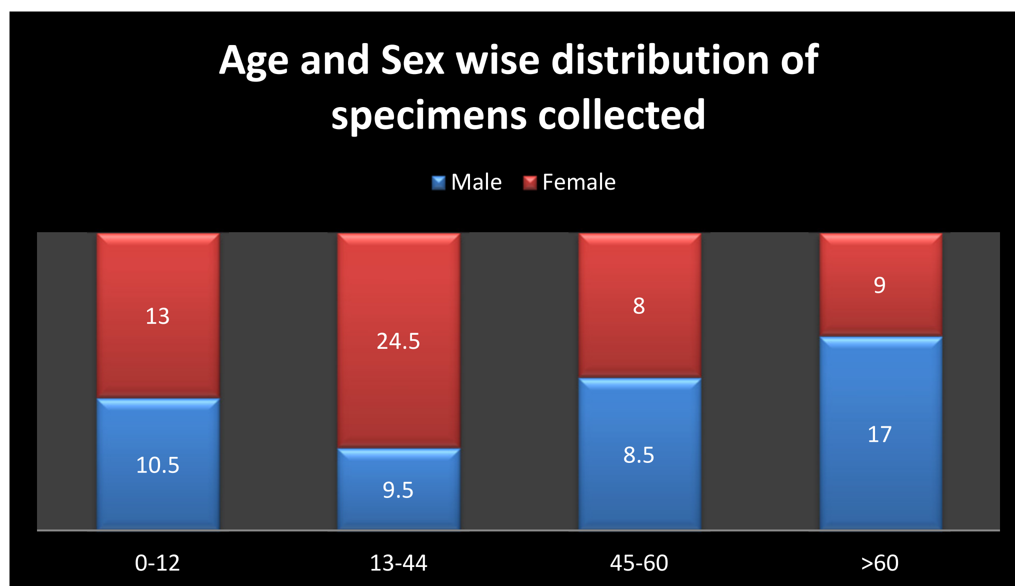
A total of 400 samples were collected from both inpatients and out patients with age group ranging from 0-80 years with symptoms suggestive of UTI. Out of 400 patients 23.50% were less than 12years 34 % were in the 13-44 age group, 16.5 % were in middle age group and 26% belong to older age group >60years. This is shown in Table 1 and Figure 1

Age and Sex wise distribution of specimens collected (n= 400)

Table 1

Age group	Male	%	Female	%	Total	%
0-12	42	10.50	52	13.00	094	23.50
13-44	38	09.50	98	24.50	136	**34.00**
45-60	34	08.50	32	08.00	066	16.50
>60	68	17.00	36	09.00	104	26.00
	182	45.50	218	54.50	400	100

Figure1



400 specimens were collected from the patients with symptoms suggestive of UTI from the various departments by aseptic methods (clean catch midstream urine, catheterized and Suprapubic).Out of 400 specimens collected 154 samples showed significant growth by that prevalence of UTI was 38.50%. This is shown in Table2.

Prevalence of UTI among specimens collected (n= 400)

Table 2

Total specimen collected from patients with symptoms	**Patients with UTI** (significant growth)	**% of UTI**
400	154	38.50%

Out of 154 UTI patients 45.46 % patients were from male and 54.54 % patients were from female. Male to female ratio was 1: 1.2. The Prevalence of UTI was greater in age group of 13-44years (36.38%) followed by older age group 27.92%. prevalence rate in paediatric age group was 23.37 and in middle age group it was12.33%. In < 12 yrs group 2 specimens were collected from the male new born baby by supra pubic aspiration method. In older age group among 27.92%, 23 patients (14.94%) were catheterized and 20 patients (12.99%) were non catheterized. This is shown in Table 3and Figure 2

Distribution of UTI according to age and sex (n=154)

Table 3

Age group	**Male**	**%**	**Female**	**%**	**Total**	**%**
0-12	12	07.79	24	15.58	36	23.37
13-44	18	11.70	38	24.68	56	**36.38**
45-60	10	06.49	09	05.84	19	12.33
>60	30	19.48	13	08.44	43	**27.92**
Total	70	45.46	84	54.54	154	100

Figure2

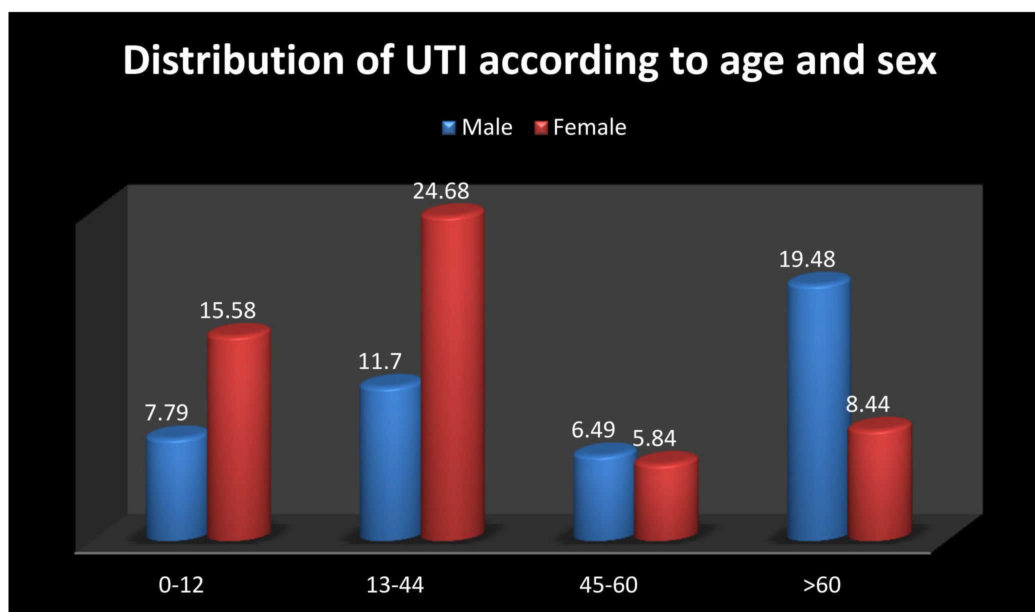


Table No 4 shows out of 154 patients with UTI, 115(74.68%) patients were inpatients and 39(25.32%) were out patients. According to Department wise, prevalence of UTI was highest in Medicine department i.e 29.87% particularly in patients admitted in Medicine Ward followed by Paediatrics 23.37% ,

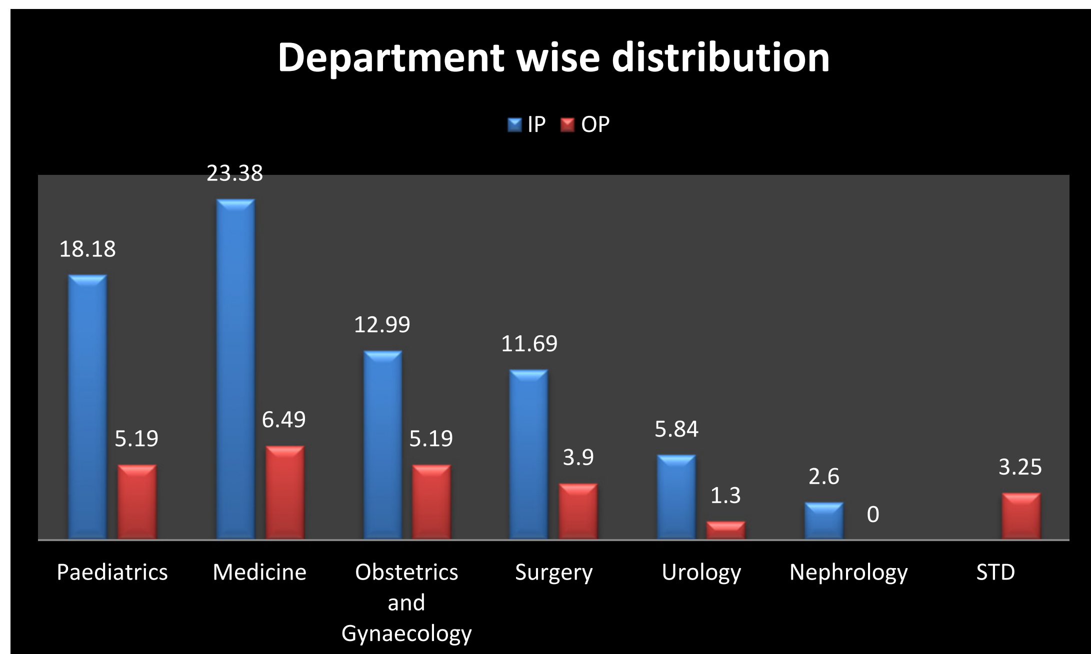
Obstetrics and gynaecology 18.18%,Surgery15.59%, and Urology 7.14%. Least distribution was seen in department of STD 3.25% and Nephrology 2.6% it was observed that prevalence of UTI was higher in Inpatients. This is shown in Table 4 and Figure3.

Department wise distribution of UTI (n=154)

Table 4

Department	IP		OP		Total	
	Number	%	Number	%	Number	%
Paediatrics	28	18.18	08	5.19	36	23.37
Medicine	36	23.38	10	6.49	46	**29.87**
Obstetrics and Gynaecology	20	12.99	08	5.19	28	18.18
Surgery	18	11.69	06	3.90	24	15.59
Urology	09	05.84	02	1.30	11	07.14
Nephrology	04	02.60	-	-	04	02.60
STD	-		05	3.25	05	03.25
	115	74.68	39	25.32	154	100

Figure3



Pattern of isolates in specimen with significant growth

Out of the 400 specimens collected 154 specimens were with significant bacteriuria in which 148 specimens were with single isolate and 6 specimens with 2 isolates each. This is shown in Table5

Table 5(n=154)

Total Samples	One Isolate	Two Isolates
154	148	06
%	96.10	3.90

Distribution of isolates among specimens with UTI

Distribution of isolates among the specimens with single isolate (n=148)

Table 6 showed out of 154 specimens 148 specimens with single isolates. Among the 148 isolates the predominant organism was Enterobacteriaceae 119(80.41%) 80.41% followed by Coagulase negative Staphylococcus 9(6.08%) NFGNB 9(6.08%) and Staphylococcus aureus 6(4.05%) and Enterococcus spp 5(3.37%). This is shown in Table6

Table6 (n=148)

Name of the organism	Number	%
Enterobacteriaceae	119	80.41
NFGNB	09	06.08
Staphylococcus aureus	06	04.05
Enterococci	05	03.38
CONS	09	06.08
Total	148	100

Figure 4

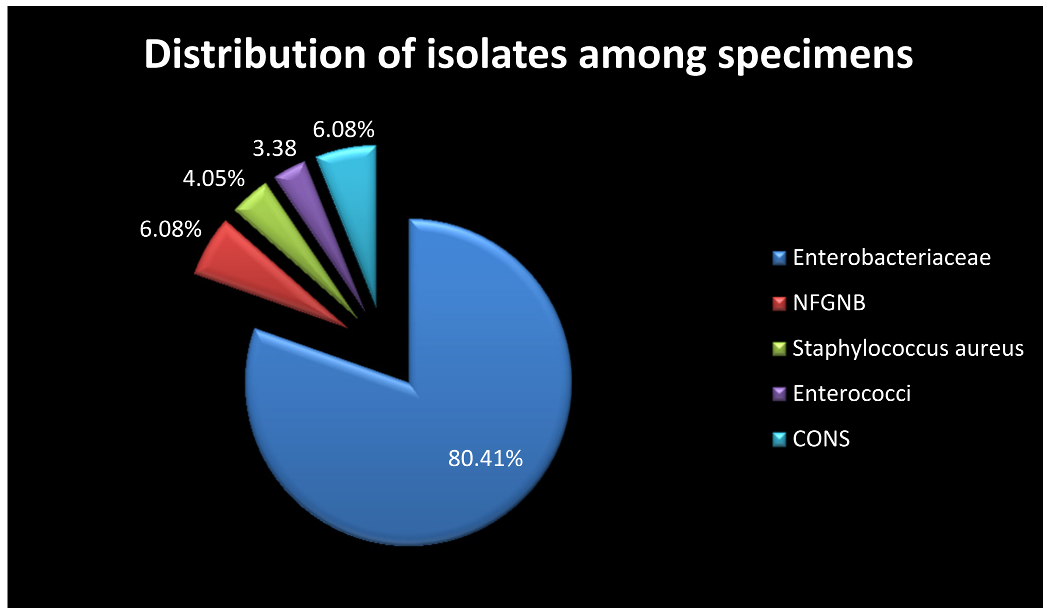


Table 7 shows that among 154 specimens, 6specimens showed two isolates. Out of this 6 specimen totally 12 pathogens were isolated. Among the 12 pathogens 5 were CoNS followed by 3 Enterobacteriaceae, 2 NFGNB and Enterococci each.

Distribution of isolates among the specimens with two isolates (n=6)

Table 7

Organisms	Number
Escherichia coli with CoNS	1
Escherichia coli with NFGNB	1
Klebsiella pneumonia with CoNS	1
Enterococci with CoNS	2
NFGNB with CoNS	1

Prevalence of urinary pathogens

Among 154 specimen processed the major cause for UTI was GNB 133 (83.13%) particularly Enterobacteriaceae (76.25%) followed by NFGNB 6.88% and by Gram positive cocci was 27(16.87%). Out of 27 GPC, 14 were CoNS of which 9 were isolated alone and 5 were associated with other pathogen. Among CoNS 5 isolates were Staphylococcus saprophyticus.

Distribution of organism among total isolates (n=160)

Table 8

Organism	Among specimen with one isolate n=148	Among specimen with two isolates n=6 (N=6×2=12)	Total n=160	%
Enterobacteriaceae	119	3	122	**76.25**
NFGNB	09	2	11	06.88
Staphylococcus aureus	06	0	06	3.75
Enterococci	05	2	07	4.37
CoNS	09	5	14	8.75
Total	148	12	160	100

Figure5

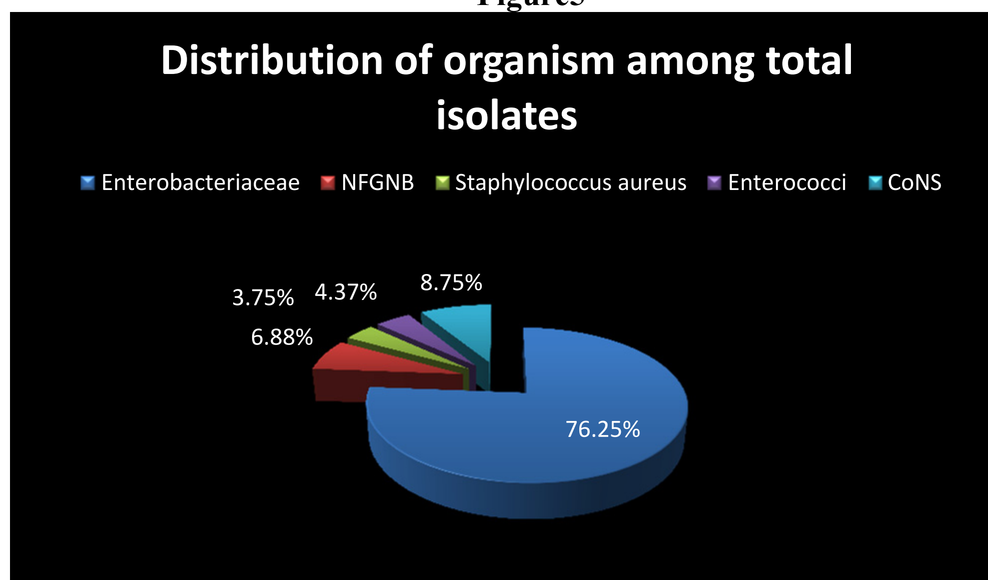


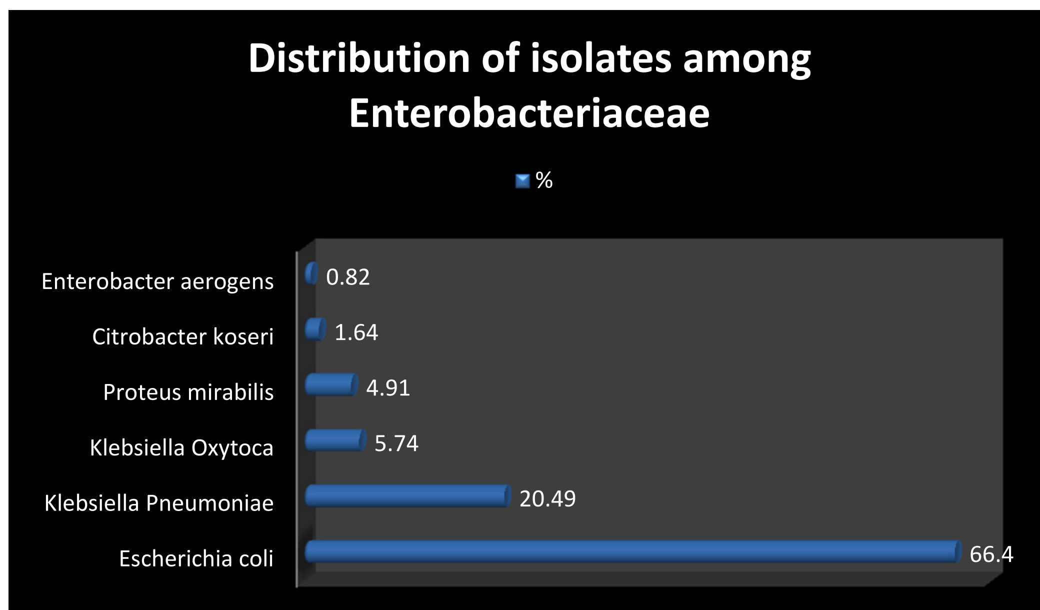
Table No 9 shows that among the 122 Enterobacteriaceae, Escherichia coli and Klebsiella spp alone constitutes 92.64% and remaining 7.37% was constituted by Proteus mirabilis, Citrobacter koseri and Enterobacter aerogens. This is shown in Table9, and Figure 6

Distribution of isolates among Enterobacteriaceae in UTI (n= 122)

Table 9

Enterobacteriaceae	Number	%
Escherichia coli	81	**66.40**
Klebsiella Pneumoniae	25	20.49
Klebsiella Oxytoca	07	05.74
Proteus mirabilis	06	04.91
Citrobacter koseri	02	01.64
Enterobacter aerogens	01	00.82
Total	122	100

Figure6



Antimicrobial susceptibility pattern

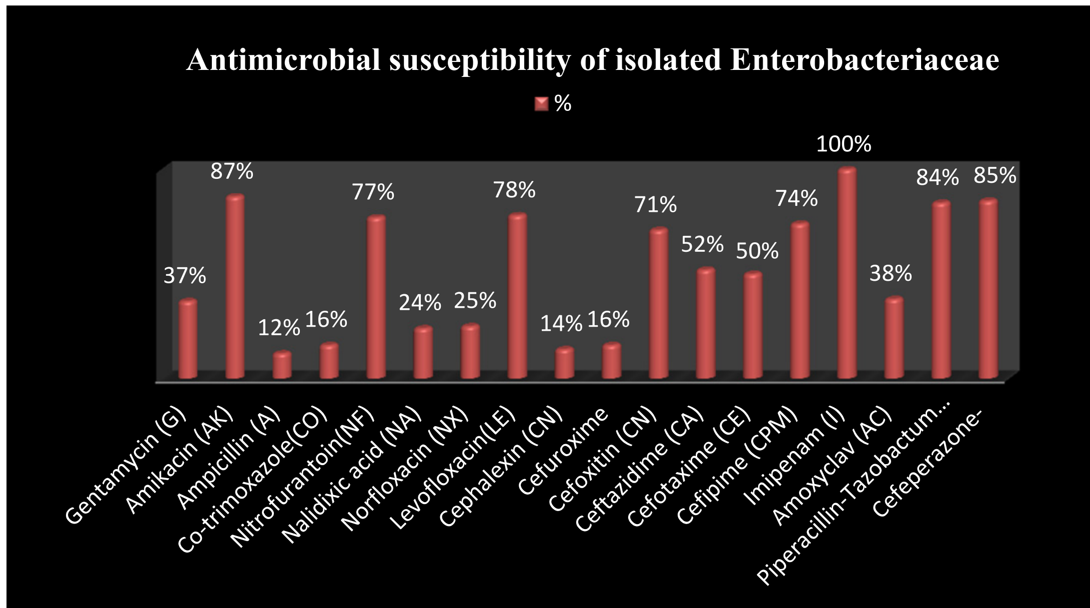
All the isolates isolated from154 urine samples with UTI were processed for Antibiotic Sensitivity Test. AST of Enterobacteriaceae was represented in the following table.

Antimicrobial susceptibility of isolated Enterobacteriaceae(n=122)

Table 10

Sl. No		Escherichia coli (n=81)	Klebsiella spp (n= 32)	Proteus spp(n=6)	Citrobacter spp(n=2)	Enterobacter aerogens(n=1)	Total n=122
1.	Gentamycin (GEN)	30(37%)	11(34%)	3(50%)	1(50%)	1(100%)	46(37%)
2.	Amikacin (AK)	72(89%)	26(81%)	5(83%)	2(100%)	1(100%)	106(87%)
3.	Ampicillin (AMP)	12(15%)	-	2(33%)	-	-	14(12%)
4.	Co-trimoxazole(COT)	14(17%)	04(13%)	0	1(50%)	1(100%)	20(16%)
5.	Nitrofurantoin(NIT)	69(85%)	24(75%)	-	1(50%)	0	94(77%)
6	Norfloxacin (NX)	20(25%)	09(28%)	1(17%)	1(50%)	0	31(25%)
7	Levofloxacin(LE)	62(77%)	26(81%)	4(67%)	2(100%)	1(100%)	95(78%)
8	Cephalexin (CN)	12(15%)	04(13%)	1(17%)	0	-	17(14%)
9	Cefuroxime(CXM)	14(17%)	05(16%)	1(17%)	0	-	20(16%)
10.	Cefoxitin (CX)	60(74%)	21(66%)	3(50%)	2(100%)	-	86(71%)
11.	Ceftazidime (CAZ)	41(51%)	15(47%)	4(67%)	2(100%)	1(100%)	63(52%)
12.	Cefotaxime (CTX)	40(49%)	14(44%)	4(67%)	2(100%)	1(100%)	61(50%)
13.	Cefipime (CPM)	58(72%)	24(75%)	5(83%)	2(100%)	1(100%)	90(74%)
14.	Imipenam (IPM)	81(100%)	32(100%)	6(100%)	2(100%)	1(100%)	122(100%)
15.	Amoxyclav (AMC)	30(37%)	12(38%)	2(50%)	0	-	46(38%)
16.	Piperacillin-Tazobactum (PIT)	70(86%)	28(87.5%)	5(83%)	0	0	103(84%)
17.	Cefeperazone-Sulbactum (CFS)	70(86%)	25(78%)	6(100%)	2(100%)	1(100%)	104(85%)

Figure 7



Enterobacteriaceae showed highest sensitivity to Imipenam (100%) followed by Amikacin(87%), Levofloxacin(78%), Nitrofurantoin(77%) Cefepime(74%), Cefoxitin(71%). Lower sensitivity pattern observed to Ampicillin(12%), Cephelexin(14%) Co-trimoxazole(16%), Cefuroxime(16%), Norfloxacin(25%) and Gentamicin(37%). In Citrobacter koseri and Enterobacter aerogens highest sensitivity was observed in third generation Cephalosporins.

Phenotypic detection of ESBL Distribution of ESBL detected among Enterobacteriaceae by phenotypic screening methods.

Table No11 shows that out of 81 isolates of Esherichia coli 41 (50.62%) were resistant to any one of the third generation cephalosporins. Among 32 isolates of klebsiella spp 17(53.13%) showed resistance among 6 isolates of Proteus mirabilis 2(33.33%) resistant to any one of the third generation

cephalosporins. In Citrobacter and Enterobacter no resistance noted for third generation cephalosporins. Among 122 Enterobacteriaceae 63(51.64%) isolates of Enterobacteriaceae showed resistance to any one of the third generation Cephalosporins. This is shown in Table 11 and Figure 8. All the 122 isolates were subjected to the phenotypic methods DDST, CHROM agar, PCT and E-test for detection and confirmation of ESBL.

Table 11(n=122)

Organism	Number of isolates	Organism sensitivity to third generation Cephalosporins	%	organism with resistance to any one of third generation Cephalosporins (Propable ESBL Producer)	%
Escherichia coli	81	40	49.38	41	50.62
Klebsiella spp	32	15	46.87	17	53.13
Proteus mirabilis	06	04	66.67	02	33.33
Citrobacter koseri	02	02	100.00	00	00
Enterobacter aerogens	01	01	100.00	00	00

Figure8

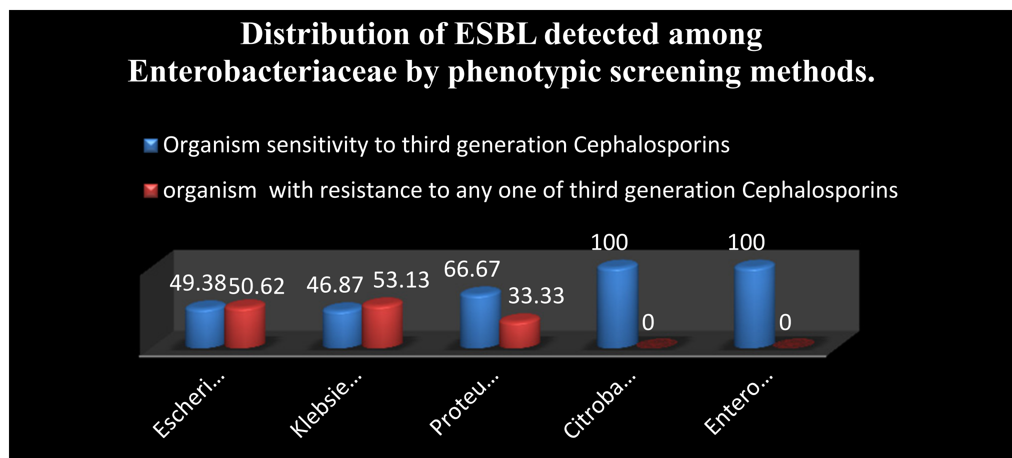


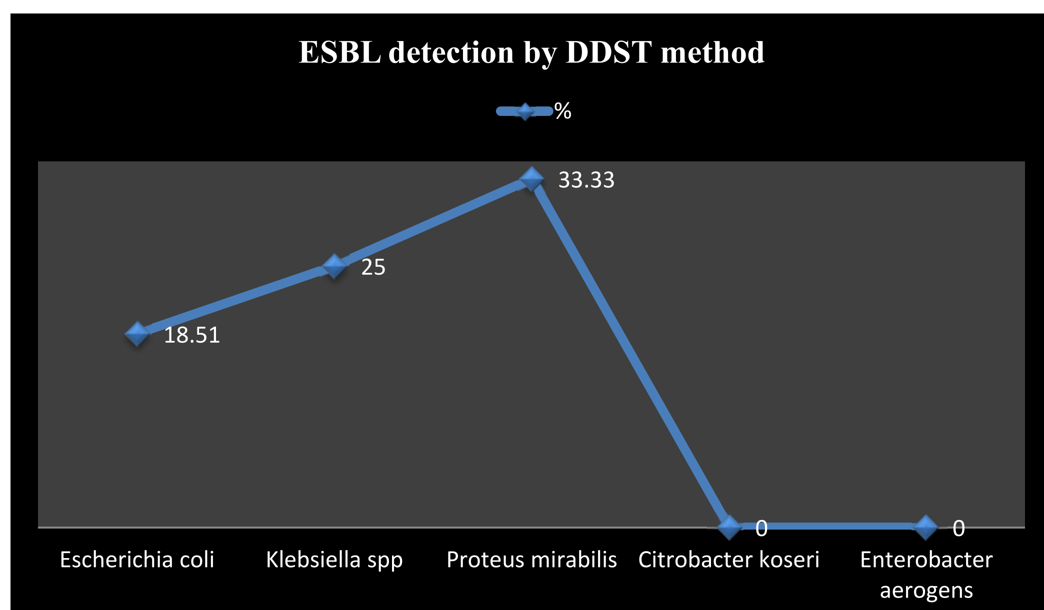
Table No 12 shows that by DDST method of 81isolates of Escherichia coli 15 isolates (18.51%), 8 (25%) of 32 isolates of Klebsiella species and 2 (33.33%) of 6 isolates of Proteus mirabilis were detected as ESBL. This is shown in Table 12 and Figure 9

ESBL detection by Double Disk Synergy Test (DDST method) n=122

Table 12

Organism	Number of isolates	Detection of ESBL by DDST method	%
Escherichia coli	81	15	18.51
Klebsiella spp	32	08	25.00
Proteus mirabilis	06	02	33.33
Citrobacter koseri	02	00	-
Enterobacter aerogens	01	00	-

Figure 9



ESBL detection by CHROM agar (n= 122)

Table 13

Organism	Number of isolates	Detection of ESBL by Chrom agar method	%
Escherichia coli	81	34	41.98
Klebsiella spp	32	14	43.75
Proteus mirabilis	06	02	33.33
Citrobacter koseri	02	00	00
Enterobacter aerogens	01	00	00

ESBL detection by PCT method (n=122)

Table 14

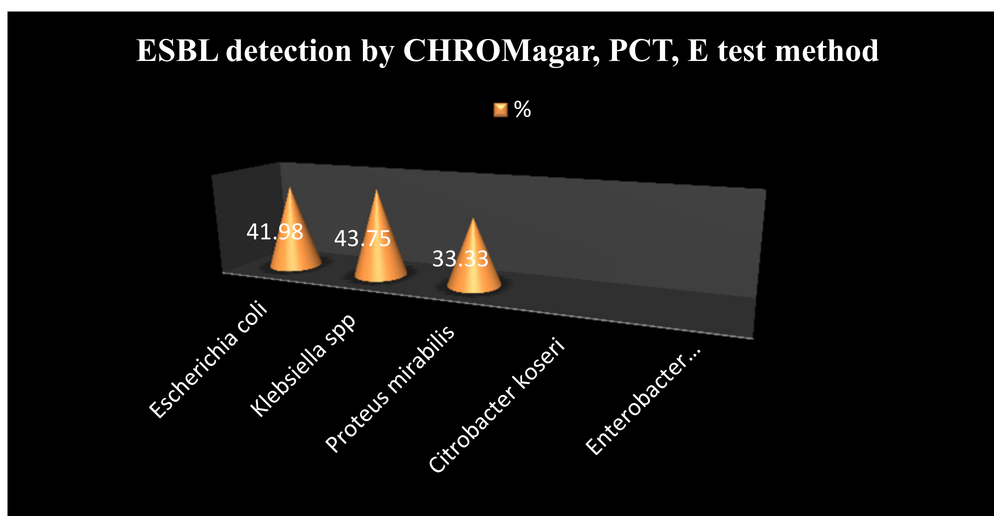
Organism	Number of isolates	Detection of ESBL by PCT method	%
Escherichia coli	81	34	41.98
Klebsiella spp	32	14	43.75
Proteus mirabilis	06	02	33.33
Citrobacter koseri	02	00	
Enterobacter aerogens	01	00	

.ESBL detection by E test method(n=122)

Table 15

Organism	Number of isolates	Detection of ESBL by E test method	%
Escherichia coli	81	34	41.98
Klebsiella spp	32	14	43.75
Proteus mirabilis	06	02	33.33
Citrobacter koseri	02	00	
Enterobacter aerogens	01	00	

Figure10



From the above tables 13, 14, 15 and fig 10 it was observed that by CHROM agar, PCT, and E test method among 81 isolates of Escherichia coli 34 isolates (41.98%) among 32 isolates of Klebsiella species 14 isolates(43.75%) and among 6 isolates of Proteus mirabilis 2 isolates(33.33%) were detected as ESBL producer.

From the above tables 12,13,14,15 it was observed that out of 122 only 25 isolates were confirmed with DDST method. By Chrom agar, PCT, and E test method 50 isolates were confirmed as ESBL positive. All the isolates which were positive for ESBL by the above methods subjected to genotypic method for the study of prevalence of TEM, SHV and CTX-M among the ESBL producer.

Comparison of different methods for ESBL detection in Enterobacteriaceae

Detection of ESBL among Escherichia coli by CHROM agar, PCT, and E-test was higher than DDST method and this was statistically significant (P=0.026). Also detection of ESBL among Klebsiella spp by the other methods was higher than DDST method however this was not statistically significant (P=0.432). This is shown in Table 16.

Table16

Organism	DDST (%)	Chrom agar (%)	PCT (%)	E-Test (%)	P value	Interpretation
Escherichia coli	**15**(18.51%)	**34**(41.98%)	**34** (41.98%)	**34** (41.98%)	0.026	Significant
Klebsiella spp	**08**(25%)	**14**(43.75%)	**14** (43.75%)	**14** (43.75%)	0.432	Not Significant
Proteus mirabilis	02(33.33%)	02(33.33%)	02 (33.33%)	02 (33.33%)	-	-

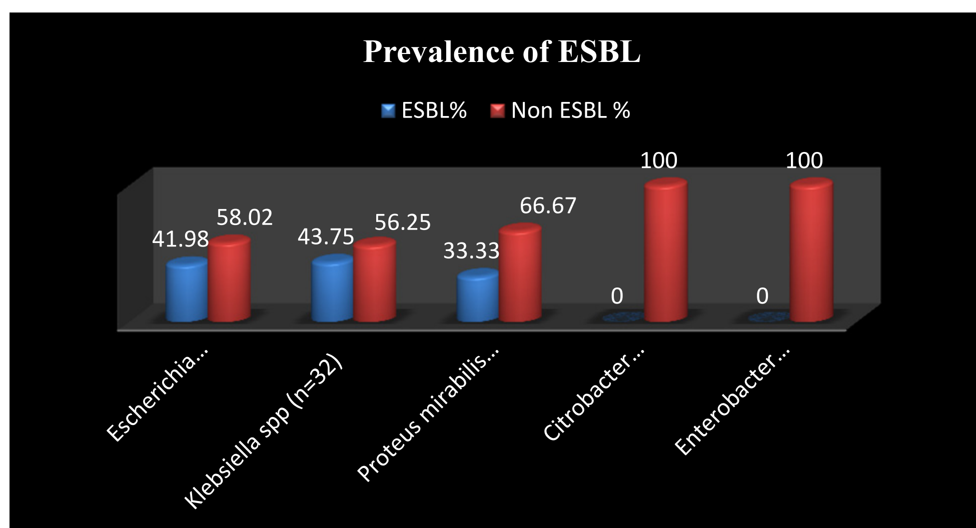
Table No 17 shows that Prevalence of ESBL among Enterobacteriaceae was 40.98% and Non ESBL 59.02%.

Prevalence of ESBL (n=122)

Table 17

Organism	ESBL	%	Non ESBL	%
Escherichia coli(n=81)	34	41.98	47	58.02
Klebsiella spp (n=32)	14	43.75	18	56.25
Proteus mirabilis (n=6)	02	33.33	04	66.67
Citrobacter koseri(n=2)	00	-	02	100
Enterobacter aerogens(n=1)	00	-	01	100
Total(n=122)	50	**40.98**	72	**59.02**

Figure 11



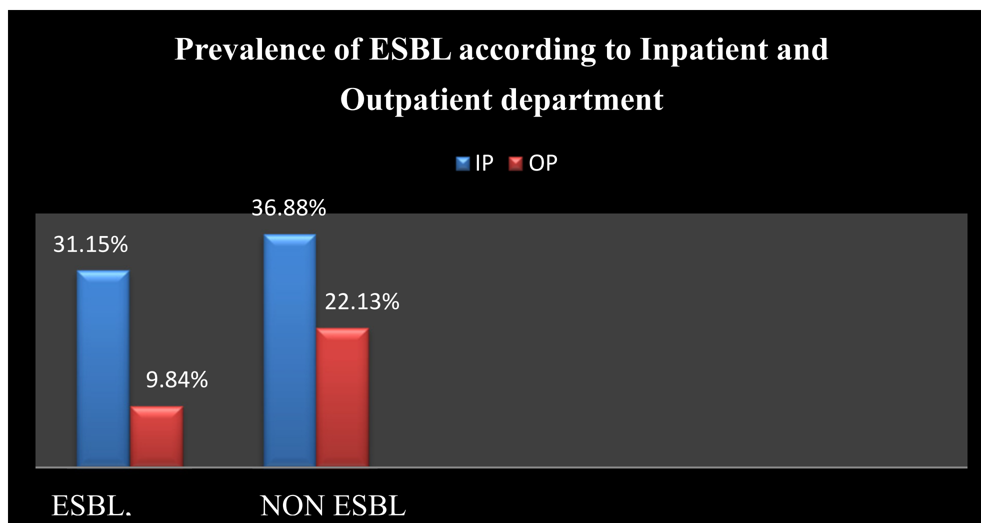
Prevalence of ESBL was higher in inpatients (31.15%) compared to the out patients (9.84%). In Non ESBL prevalence of in patients was (36.88%) and out patients was (22.13%). Generally both ESBL and Non ESBL prevalence was greater in inpatients. But regarding the out patients prevalence of Non ESBL was higher (22.13%) than ESBL (9.84%). This is shown in Table 18

Prevalence of ESBL according to Inpatient and Outpatient Department

Table18 (n=122).

organism	ESBL		Non ESBL	
	IP	**Op**	**IP**	**OP**
Escherichia coli(n= 81)	30 (37.04%)	04 (4.94%)	33 (40.74%)	14 (17.28%)
Klebsiella spp(n=32)	06 (18.75%)	08 (25%)	6 (18.75%)	12 (37.50%)
Proteus mirabilis (n=6)	02 (33.33%)	00	04 (66.67%)	00
Citrobacter koseri (n=2)	00	00	01 (50%)	01 (50%)
Enterobacter aerogens (n=1)	00	00	01 (100%)	00
Total n=122	**38** (31.15%)	12 (9.84)	**45** (36.88%)	27 (22.13%)

Figure 12



Comparison of the antimicrobial sensitivity pattern of ESBL producer and non ESBL producers number & Percentage

Organism		AMP	GEN	AK	COT	CN	CXM	CX	CAZ	CTX	CPM	NIT	NX	LE	IPM	CFS	PIT	AMC
E coli	ESBL (n=34)	0	8 (24)	26 (76)	2 (6)	0	0	28 (82)	0	0	17 (50)	29 (85)	5 (15)	24 (76)	34 (100)	29 (85)	28 (82)	6 (18)
	NonESBL (n=47)	12 (25)	22 (47)	46 (97)	12 (26)	12 (26)	14 (30)	32 (68)	41 (87)	40 (85)	41 (87)	40 (85)	15 (31)	38 (81)	47 (100)	41 (87)	42 (89)	24 (51)
Klebsiella spp	ESBL(n=14)	-	2 (14)	10 (71)	1 (7)	0	0	7 (50)	0	0	8 (57)	10 (71)	3 (21)	10 (71)	14 (100)	10 (71)	12 (85)	02 (14)
	NonESBL (n=18)	-	9 (44)	16 (89)	3 (17)	4 (22)	5 (28)	14 (78)	15 (83)	14 (78)	16 (89)	14 (78)	6 (33)	16 (89)	18 (100)	15 (83)	16 (89)	10 (71)
Proteus mirabilis	ESBL(n=2)	0	0	2 (100)	0	0	0	1 (50)	0	0	1 (50)	-	0	1 (50)	2 (100)	2 (100)	2 (100)	0 0
	NonESBL (n=4)	2 (50)	3 (75)	3 (75)	0	1 (25)	1 (25)	2 (50)	4 (100)	4 (100)	4 (100)	-	1 (25)	3 (75)	4 (100)	4 (100)	4 (100)	2 (50)

In the present study Antimicrobial resistance pattern in Escherichia coli, Klebsiella spp, and Proteus mirabilis of ESBL producer and non- producer, were compared and presented in the above Table.

Multiple drug resistance was more common in ESBL producer when compared to the non- ESBL producers. In case of E. coli, sensitivity of Gentamicin is reduced from (47% to 24%). Amikacin shows (21%) reduction in sensitivity. The co-resistance activity was found in Co- trimoxazole showing decreased sensitivity from 26% to 6%. Fluroquinolones also showed co-resistant pattern, Norfloxacin (31% to 15%), and Levofloxacin (81 % to 76%).

Table 20 shows that distribution of ESBL genes among the Enterobacteriaceae.

Resistance genes in ESBL strains

Table 20 (n=50)

organism	**TEM only**	**SHV only**	**CTX-M only**	**TEM & SHV**	**TEM& CTX-M**	**SHV& CTX-M**	**TEM,SHV &CTX-M**	**Total**
Escherichia Coli(n=34)	3	2	6	4	7	5	7	34
Klebsiella Spp(n=14)	2	1	3	1	2	2	3	14
Proteus mirabilis (n=2)					1		1	02
Total (n=50)	5	3	9	5	10	7	11	50

Percentage of resistance genes

Table 21 (n=50)

Genus	Number of Isolates	%
CTX-M only	9	**18**
TEM only	5	10
SHV only	3	06
CTX-M,TEM	10	20
CTX-M,SHV	7	14
TEM,SHV	5	10
CTX-M,TEM, SHV	11	**22**
Total	50	100

Out of 50 ESBL positive isolates, 9(18%) isolates were positive for CTX-M, 5(10%) isolates were positive for TEM, 3(6%) isolates were positive for SHV only,10(20%) isolates were positive for CTX-M & TEM, 7(14.%) isolates were positive for CTX-M &SHV, 5(10%) isolates were positive for TEM & SHV and 11(22%) isolates were positive for TEM,SHV & CTX-M.

DISCUSSION

Discussion

Urinary Tract Infections(UTI) are the most prevalent bacterial infection among the humans in general clinical practice. Every year nearly 150 million people are affected with UTI and due to this incidence health care expenditure is about 6 million dollars[60]. If the UTI are not properly treated it can lead to complications like stone formation, pyelonephritis and renal failure. One of the most important factor which has got an impact in the management of UTI is the emergence of antimicrobial resistance among the uropathogens over the past decade. The most common mechanism of antimicrobial resistance among the gram negative bacteria is the production of the Extended Spectrum Beta Lactamase enzymes. This prospective study was undertaken to know the prevalence of ESBL by various phenotypic methods and confirmation by genotypic methods. During the study a total of 400 non repetitive (Clean catch midstream, catheterized and suprapubic) urine samples were collected aseptically. According to Chau et al[21], the main aim of clean catch midstream urine collection is to avoid contamination during voiding by urethral and perineal flora.

By **Morton RE[74] 1982** for the diagnosis of UTI in paediatric population adequate result was obtained by MSU if properly collected and SPA is indicated when there is in need of accurate diagnosis or if MSU can't be obtained. Among the total 400 samples collected 154 (38.50%) showed significant growth of bacteria. The similar prevalence has been reported by seen **in Trupti Bajpai et al[114] (38.3%)at Madhya Pradesh.** The Prevalence

rate was higher in the present study when compared to the study done by **Elizabeth[17]et al (32.1%) Bangalore**. From the 154 samples which showed significant growth totally 160 uropathogens isolates were isolated this was because 6 of them grew two isolates each.

In the present study the gender wise prevalence of UTI showed 45.46% were male and 54.54% were females with male to female ratio was 1:1.2. In Tamilnadu the same ratio (1:1.3%) was seen in study of **Baby Padmini[13] et al** 2004 Coimbatore. From several previous studies by **Carolin Elizabeth George[17] et al (60.7%) Bangalore, Astal [11]et al (65%) Palestine and Ahmed[1] et al (84%) Kashmir** it was observed that females are more prone for UTI than male. In present study more number of UTI were found in reproductive age group 36.38% followed by older age group 27.92%.But in **study Sood and Gupta[105] et al 2011 Rajasthan** prevalence was higher in older age group(35%) followed by reproductive age group(23%). Reason for higher incidence in female is because of shorter urethra, and the opening of urethral meatus in to the moist introitus and close approximation with rectum. favours the colonization of bacteria resulting in bacterial cystitis. Other factors which favours the occurrence of UTI in reproductive age group are sexual intercourse and pregnancy. In young men the important factor which increases the risk for UTI is lack of circumcision resulting in symptomatic urethritis. In older age group prevalence was slightly higher in males and in the present study male to female ratio was 2.3:1 and this is in concordance with study of **Andrea Cove[6] et al (2:1) U.K.**

In this study next to the reproductive age group, and older age group, the prevalence of UTI was higher in Paediatric age group 23.37% which was lesser when compared to the study of **Palak Gupta[80] 35.4%.**From the studies of **Palak Gupta[80], Riccabona[91]et al** it was observed that in the first year of life UTI is seen more commonly in male child 3.7% and 2% in female child and there after incidence steadily increasing in female population. If the infection occurs in preschool boys it is usually associated with congenital abnormalities. When bacteriuria was first detected in Paediatric population there was chance of presence of some referral urinary tract abnormality in one third of this population. The presence of bacteriuria in Paediatric group defines a population at higher risk for the development of bacteriuria in adult group.

Worldwide Prevalence of Uropathogens

Organism	Latin America P.H.A Bours et al[88]	Brazil Daynae Moraes[26]	Mathya Pradesh Trupti Bajpai[114]et al	Tamil nadu Ramseh[89] et al	Present study
Enterobacteriaceae	83.6%	82.8%	63.70%	64.90%	76.25%
NFGNB	-	0.8%	13.88%	20.65%	06.88%
Staphylococcus aureus	3.3%	2.5%	01.78%	-	03.75%
Enterococcus	-	3.5%	07.12%	9.5%	04.37%
CoNS	-	9.4%	01.42%	5%	08.75%
Other pathogens	13.25	-	-	-	-

In the present Study organism belonging to the Enterobacteriaceae family were commonly isolated. Study conducted in **India by Trupti Bajpai[114]et al** in **Latin America by P.H.A Bours et al[88]** and in **Brazil**

byDaynae Moraes[26] also showed that Enterobacteriaceae group of organism were commonly isolated from the UTI.

Distribution of organism among Enterobacteriaceae World wide

Enterobacteriaceae	Latin America[88]	Brazil [26]	Madhya pradesh[114]	Tamil nadu[89]	Present study
Escherichia coli	57.89%	73.69%	68.57%	46.15%	66.40%
Klebsiella spp	03.95%	07.58%	28.57%	34.51%	26.23%
Proteus mirabilis	02.63%	11.37%	0.57%	09.60%	04.91%
Citrobacter koseri	-	01.67%	-	03.94%	01.64%
Enterobacter aerogens	07.89%	04.03%	0.57%	04.53%	0.82%

Among Enterobacteriaceae in the present study, Escherichia coli (66.40%) was the commonest organism isolated followed by Klebsiella (26.23%), Proteus mirabilis (4.91%) and the least isolated was Citrobacterkoseri(1.64%), and Enterobacter aerogens (0.82%). This prevalence was almost similar to the study done at Brazil by **Dayane Moraes**[26] **et al**, India at Madhya Pradesh by **Trupti Bajpai** [114]**et al** and in Tamilnadu by **Ramseh**[89] **et al** Coimbatore.

According to this study the most effective antibiotics against organism of Enterobacteriaceae were Imipenam (100%) followed by Amikacin (86%), Levofloxacin (78%) and Nitrofurantoin (77%). Similar findings were reported by **Carolin**[17] **et al**(90% for Amikacin and 70% for Nitrofurantoin) and **Sarasu**[95] **et al**(for Amikacin 100%, Levofloxacin 64% and for Nitrofurantoin 68%) from India. From the present study the alarming finding notified that most of the strains were resistant to Ampicillin (14%) and Cotrimoxazole(14%)

Also in studies of **Curtis Nickel[47]et al, Anbumani [5]et al, and Sarasu [95]et al** sensitivity to Ampicillin was (45%,19%,16%,). In the study of **Sarasu[95] et al(18%) Tamilnadu and study of Mandira Mukerji[68] et al India (13.5%)** less sensitive for Cotrimoxazole was observed. Reason for this resistance may be because these antibiotics have been extensively used in this region for a longer period and also due to the misuse of antibiotics, which has led to the emergence of resistant bacteria today. Hence generally in India Cotrimoxazole and Ampicillin cannot be recommended as an empiric therapy for the treatment of UTI.

Nowadays one of the challenge faced by every Microbiologist is the detection of ESBL production by the Enterobacteriaceae. The main aim for the ESBL detection is to prevent the dissemination by co transmission and also for the epidemiological purpose. By controlling the dissemination therapeutic failure can be prevented.

In the present study four phenotypic methods DDST, CHROM agar,PCT and E-test were compared for the detection of ESBL. Among these two methods (DDST, PCT) widely used in the routine testing and the other two methods(E-test, CHROM agar) are specifically developed to detect ESBL production. The main aim of this study to achieve most sensitive method for ESBL detection in a Enterobacteriaceae family by using the combination of routine method with a specific ESBL test method.

According to the CLSI all the organisms of Enterobacteriaceae were screened for potential ESBL producer by Kirby – Bauer disc diffusion method on MHA. Since some times ESBL isolates show false susceptibility to third generation Cephalosporins **Anbumani[5] et al** in standard disc diffusion method so it is must to do the specific Phenotypic methods along with screening methods.

In the present study PCT, E-test, and CHROM agar detected totally 50(40.98%) isolates as ESBL out of 122 isolates, but DDST detected only 25(20.49%) isolates as ESBL positive. Thus additional 20.49% were detected as ESBL by the other methods. The similar findings i.e lesser detection of ESBL by DDST method was also observed by **Mohammed Hisham[85] et al 2016 at Kerala and by Prabha[93] et al 2016 at Pudhucherry**. According to CLSI guidelines also PCT is more effective in detection of ESBL producer than DDST method.

In this study DDST was less sensitive than the other methods CHROM agar, PCT, and E-test. But study conducted by **Ewelina Kaluzana[34] et al 2014** showed higher sensitivity to DDST method . In the present study ESBL Chrom agar detected 41.98 % of ESBL of Escherichia coli 43.75% of ESBL of Klebsiella spp and 33.33% ESBL of Proteus mirabilis. But according to **Ewelina Kaluzana[34] et al 2014** Chrom ID ESBL method used for the detection of ESBL strains are characteristically showed relatively high sensitivity with low specificity, so it has got chances for false positivity. This is due to the fact

that Chrom ID detected not only ESBL enzymes but also broad spectrum beta lactamases. Both PCT and E test method detected 41.97% in Escherichia coli , 43.75% in Klebsiella spp and 33.33% in Proteus mirabilis.

Prevalence of ESBL at different Geographical areas

Geographical areas	**E.coli**	**Klebsiella spp**	**Proteus mirabilis**	**Citrobacter spp**	**Enterobacter aerogens**	**Authors**
Nepal	13.5%	16.55%	-	-	-	Anil Chander et al 2013[7]
Islamabad	51%	40.90%	-	-	-	Shamin Mumtaz et al 2006[77]
Mumbai (India)	40.62 %	27.58%	19.05%	-	-	K.Aruna et al 2012[10]
Pune (India)	28.72 %	15.90%	-	-	-	ParulAgrawal et al 2008[81]
Rajasthan (India)	56.92 %	67.04%	41.89%	27.59%	-	Meetha Sharma et al 2013[71]
Kerala (India)	62.3%	67.4%				Shashikala et al 2007[89///]
Coimbatore (Tamilnadu)	41%	40%	-	-	-	Baby Padmini et al 2004[13]
Chennai (Tamilnadu)	60%	-	-	-	-	Anbumani Narayana samy et al 2010[5]
Present	**41.98**	**43.75%**	**33.33%**			

This study reported 40.98% ESBL producers among Enterobacteriaceae unlike the studies made **by Tankhiwale [110]et al (48.3%) and Khurana [57]et al. (26.6%).**

Regarding the prevalence of ESBL in the present study 41.98% is in Escherichia coli 43.75% in Klebsiella spp and 33.33% in Proteus mirabilis. Similar findings were seen in the study done by **Baby Padmini[13] et al in Tamil nadu** in Mumbai by **K.Aruna[10] et al.** In this study prevalence of ESBL

was low when compared to the study done by **Meetha Sharma**[71] **et al** at **Rajasthan** and **Anbumani Narayanasamy**[5] **et al at Chennai.** This ESBL prevalence was quite high when compared to the study done by **ParulAgrawa**[81] **et al at Pune and Anil Chander**[7] **et al at Nepal.**

In the present study, among the in-patients, ESBL producing Escherichia coli (37.04%) was found to be most prevalent organism followed by Proteus mirabilis(33.33%) and Klebsiella spp (18.75%).While in outpatients Klebsiella (25%) was the most prevalent ESBL producing organism, followed by Escherichia. coli (4.94%). A similar finding i.e Klebsiella more prevalent in outpatients was observed by **Mumtaz** [77]**et al in Pakistan.**

Nowadays ESBL are the most common problem in hospitalized patients throughout the world. The prevalence of ESBLs among clinical isolates varies greatly worldwide and in geographical areas and are rapidly changing over time[63].

Multiple drug resistance was seen in the ESBL producers than the non-ESBL producers. In present study in Escherichia coli sensitivity of Gentamicin is reduced from 47 % to 24 %. Whereas in Amikacin it was reduced from 97% to 76%. Co-trimoxazole sensitivity reduced nearly 20%. Fluroquinolones also showed co- resistance pattern, Sensitivity reduced in Nalidixic acid (38% to 9%), Norfloxacin (31% to 15%), and Levofloxacin (81 % to 76%). Whereas,in Klebsiella spp Gentamicin, sensitivity decreased from (50% to14 %). Other drugs shown to be resistance are Co-trimoxazole (44% to 14%), Levofloxacin

(89% to 71%). In Proteus mirabilis highest reduction in sensitivity was observed in Gentamicin, Amikacin and Levofloxacin.Similar type of Co-resistance pattern was observed in the study of **Singh S[103] et al at Khanpur**.

Resistance gene coding to Quinolones and beta - Lactam antibiotics are located on the same plasmid and thus passed on together among different species of Enterobacteriaceae, in addition to loss of porins (or) efflux pump and these multiple factors play a major role in co-resistance[50].Further studies showed that CTX-M gene in Escherichia.coli highly associated with MDR phenotype.

Co-resistance pattern seen in Co- trimoxazole and Gentamicin and this is due to that single plasmid which carry resistance gene to these agent along with ESBL gene[87].In this study, the resistance to Fluoroquinolones varied from 11% – 75% for Enterobacteriaceae and this was in concordance with study done by **Mahesh[65] et al Bangalore where** 27.6 to 90% of resistance was observed. Quinolones are the most active agents against UTI pathogens in **North America** as per the study of **Gordon [39]et al.**

In the present study the co-resistance was low for Amikacin (21%). Similar findings were observed by Baby Padmini and Appalaraju [28] and V.P Sarasu et al [95]

According to this study sensitivity for ESBL producing Escherichia coli, Klebsiella spp and Proteus mirabilis for Piperacillin-Tazobactum and Cefeperazone-sulbactum were (82%,85%,100%) and (85%,71%,100%)

Whereas in **Mangalore, Shigu et al**[85] showed highest sensitivity of the ESBL producing Escherichia coli to CFS and PIT(100%/100%) and for ESBL producing Klebsiella spp(98%/88%)The present study sensitivity regarding CFS and PIT was low when compared to **Shigu et al** but sensitivity was high when compared to the study conducted by **Anbumani**[5]**et al (for PIT 49%)**

According to the present study it was observed that the most effective antibiotic against ESBL producing Escherichia coli Klebsiella spp and Proteus mirabilis in UTI are Imipenam (100%,100%,100%), Cefeperazone-sulbactum (85%,71%/100%),Piperacillin-Tazobactum, (82%,85%,100%), Amikacin (76%,71%,100%), Levofloxacin(76%,71%,50%) and Nitrofurantoin (85%, 71%-),Similar findings were observed in the study of Baby Padmini et al 2004. According to **Gaurav Dalela**[37] **et al** 2012 highest sensitivity observed to Cefepime (83.2%) for Piperacillin-Tazobactum,(75.8%) and for Amikacin it was (74.7%). For the above drugs resistance observed in non ESBL producers but this was due to different mechanism other than Extended Spectrum Beta lactamase such as AmpC beta lactamase, metallobetalactamase etc.

Highest sensitivity for beta lactamase and beta lactamase inhibitors was observed in the Study conducted in **Kerala** by **Shasikala et al**[99]. In that study Piperacillin-Tazobactum (96.8%), Cefeperazone-sulbactum (92.2%), were sensitive to ESBL producers.

When compared to the other oral antibiotics Nitrofurantoin has shown least resistance especially for Escherichia coli followed by Klebsiella spp. Most of the Indian studies **Khurana S et al**[57] **Tankhiwale SS et al**[110] have

demonstrated that Nitrofurantoin can be used as the first line drug in the treatment of uncomplicated UTI as it is highly concentrated in the urine and it can be administered orally. The reason for least resistance is limitation of its use because it has no role in the treatment of other infection. Main drawback of Nitrofurantoin is that cannot be used in upper UTI and also in Proteus mirabilis infection because of its intrinsic resistance nature to Proteus mirabilis (CLSI 2016).

From the present study and also from the previous study it was observed that the most reliable treatment for infection caused by ESBL producing Enterobacteriaceae are Carbapenams. Despite their utility chances for the emergence of resistance so carbapenams can be reserved for serious infections. Alternate drugs like Nitrofurantoin, Piperacillin Tozabactam, cefaperazone Sulbactam ,Amikacin, Levofloxacin and cefepime can be given for the ESBL producing UTI.

In the present study CTX-M (64%) is the most common and it is present as either alone or in combination with TEM, SHV,or both. The high prevalence of CTX-M gene in the present study was in concordance with **Mohamad Hisham PP et al [85] (56%) and Meetha Sharma et al[71] (82.5%)** But in study of **Bali et al Turkey[31]** TEM (73%) type was predominant and in study of **Kawthar[55] et al Egypt SHV**(69.2%) was predominant. CTX-M was low when compared to the present study. Also data from the last 10 years and Livermore et al stated that worldwide CTX-M gene was most prevalent which is replacing

SHV and TEM types as predominant ESBL in Asian and in many European countries. According to **Goyal et al**[40] majority of ESBL strains harbored two or more gene and this was in concordance with our present which showed more than one type of beta lactamases in 22 out of 50 isolates.

SUMMARY

Summary

The study was conducted at Government Rajaji Hospital and Institute of Microbiology Madurai over a period of one year from September 2015 to August 2016 with 400 patients suffering from UTI, which included 45.50 % of males and 54.50 % of females. Among 400 patients 154 (38.50%) of them had significant bacteriuria. Among 154 specimens tested more than 1 isolate was obtained in 6 specimens (2 organisms isolated) UTI was higher in the age group 13-44years 36.38% followed by elderly people 27.92%.

- In this study, totally 133 Gram Negative Bacilli and 27 Gram Positive Cocci were isolated among which 122 were to Enterobacteriaceae. Among the Enterobacteriaceae Escherichia coli was the commonest organism isolated (66.40%) followed by Klebsiella spp (26.23%), Proteus mirabilis (4.91%), Citrobacter koseri (1.64%) and Enterobacter aerogens (0.82%).
- ESBL producing organisms accounts for treatment failure leading to high morbidity and even mortality. Hence early detection of the ESBL producing organisms is very important for the treatment aspect. In the present study comparison methods were employed for the detection of ESBL. It was observed that out of 122 only 25 isolates (20.49%) were confirmed as ESBL with DDST method. By Chrom agar, PCT, and E test method 50 isolates (40.98%) were confirmed as ESBL positive. Phenotypic method DDST showed 20.49% false negative result when compared to the other Phenotypic methods PCT, CHROM agar, and E-test.

Prevalence of ESBL production was found in 41.98 % of the E. coli, 43.75% of the Klebsiella spp and 33.33% of the Proteus mirabilis.

- All 50 ESBL strains detected by phenotypic methods were confirmed with genotypic methods for the presence of ESBL gene (TEM, SHV and CTX-M). CTX-M only (18%) TEM only (10%) SHV only (4%) CTX-M,TEM (20%) CTX-M,SHV (16%) TEM,SHV (10%) CTX-M,TEM,SHV(22%). By comparing Phenotypic and genotypic method DDST is less sensitive. But in detecting the ESBL other three methods were equally effective. CHROM agar and E-test being costly, PCT can be performed as routine test for detection of ESBL.
- Multiple drug resistance was seen in ESBL producing strains than the non ESBL production.
- Non ESBL producing Escherichia coli, Klebsiella spp, and Proteus mirabilis UTI are highly sensitive to Ceftazidime Cefotaxime and Amikacin (87% /83%/100% (85%,78%100%) and (97%89%75%)
- The sensitivity pattern ESBL producing Escherichia coli, Klebsiella spp and Proteus mirabilis in UTI are Imipenam (100%,100%,100%), Cefeperazone-sulbactum(85%,71%/100%),Piperacillin-Tazobactum, (82%, 85%, 100%),Amikacin(76%,71%,100%),Levofloxacin (76%,71%,50%) and Nitrofurantoin (85%,/71%,/-).

CONCLUSION

Conclusion

- ESBL producers among uropathogens is increasing in incidence**.**
- Although genotypic methods are more sensitive, Resource constraints prevent these tests from being used in routine diagnostic laboratories.
- PCT method can be performed as routine test for the detection of ESBL as it is more sensitive, simple to perform and cost effective.
- Non ESBL producing strains are sensitive mainly to aminoglycoside and third generation Cephalosporins. As aminoglycosides are injectables and nephrotoxic third generation Cephalosporins can be used for treating UTI as they are less toxic and also orally effective.
- All the ESBL isolates are 100% sensitive to Imepenam. Even though they are highly sensitive to Imepenam, there is chances for the emergence of resistance to Carbapenem, so it should be kept in reserve as the second line of drug. Next higher sensitive drugs like Nitrofurantoin and Levofloxacin which are most economic and orally effective can be given to outpatients. Amikacin, Cefepime, beta-lactamase-Inhibitors Cefeperazone- Sulbatum, and Piperacillin-Tazobactum, can be given to inpatients.
- Based on the prevalence rate of the ESBL production, institutional antibiotic policy can be tailored in a health care facility to achieve superior therapeutic outcome and also to bring about a reduction in healthcare costs.. Drug resistance pattern varied from place to place which is related to the nature of the pathogen and usage of antimicrobial agents.

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ANNEXURES

ANNEXURE-1

PREPARATION OF GRAM STAIN:

GRAM STAIN REAGENTS

1. Methyl violet – Primary stain

   Methyl violet 10g

   95% ethyl alcohol 100ml.

   Distilled water 1L

2. Gram's lodine – Mordant

   lodine 10g

   Potassium lodide 20g

   Distilled water 1 L

3. Acetone – Decolouriser

4. Dilute Carbol Fushsin – Counter stain

   Basic fushsin 0.3 g

   95% Ethyl alcohol 10 ml

   Phenol crystals, melted 5 ml

   Distilled water 95 ml

Basic fuchsin was dissolved in alcohol 5% phenol solution was added and was allowed to stand overnight. Then the solution filtered through coarse filter paper.

ANNEXURE-2

PREPARATION OF MEDIA

PREPARATION OF NUTRIENT AGAR

Contents:

- Peptone – 5 g
- Beef extract –1.5 g
- Yeast extract –1.5 g
- Sodium choloride – 5 g
- Agar – 15g

28 g of the contents were suspended in 1000 ml of distilled water. It was heated to boiling to dispense the medium completely. Medium was sterilized by autoclaving at 121 degree C at 15 lbs pressure for 15 minutes.

PREPARATION OF BLOOD AGAR

Nutrient agar 100 ml

Sheep blood (defibrinated) 10 ml

- The sterile nutrient agar was melted by steaming and cooled to 45 deg C
- 5%-10% sheep blood was added aseptically with constant shaking.
- The blood was mixed with molten nutrient agar thoroughly but gently, to avoid froth formation. To remove the bubbles, media was flamed.
- Immediately poured into petri dishes and allowed to set.

PREPARATION OF MUELLER – HINTON AGAR

Contents:

Beef extract 2.0 gm

Acidicase peptone 17.5 gm

Starch 1.5 gm

Agar 17.0 gm

Distilled water 1000 ml

Final pH 7.4+0.2

Dissolved the ingredients in one liter of distilled water. Mixed thoroughly. Heated with frequent agitation and boiled for one minute. Dispensed and sterilized by autoclaving at 121 deg. C for 15 minutes. Should not be overheated. When remelting the sterile medium, heated as briefly as possible.

நோயாளியின் பெயர்: ________________ வயது: ________ இனம்: ________

விலாசம்: ____

<u>தகவல் அளிக்கப்பட்ட ஒப்புதல் படிவம்</u>

மேற்குறிப்பிட்ட மருத்துவ ஆய்வில் ஓர் பங்கேற்பாளராக சேர்க்கப்பட்ட இதன் மூலம் நான் சுதந்திரமாக என் ஒப்புதலை அளிக்கிறேன்.

இந்த மருத்துவ ஆய்வின் நோக்கம் மற்றும் முக்கியத்துவம் பற்றி மற்றும் அதனால் ஏற்படும் எனது பொறுப்புகள் பற்றி எனக்கு தகவல் தெரிவிக்கின்றார். இதோடு கூடுதலாக ,நான்

தேதியிட்ட எனக்கு அளிக்கப்பட்ட நோயாளிக்கான தகவல் தாள் மற்றும் தகவல் அளிக்கப்பட்ட ஒப்புதல் படிவத்தில் அடங்கிய விபரங்கள் பற்றி படித்து புரிந்து கொண்டுள்ளேன். மருத்துவர் போதிய மற்றும் விரிவான விதத்தில் என் பங்கேற்பு பற்றித் தீர்மானிக்க எனக்குப் போதிய நேரம் இருந்தது.

இந்த மருத்துவ ஆய்வு நடத்தப்பட்ட மிக முக்கிமானதாக என் மருத்துவரின் குறிப்புகளை நான் பின்பற்றுவேன். எந்த காரணமும் அளிக்காமல், எனக்கு எந்த நஷ்டமும் ஏற்படாமல் எந்த நேரத்திலும் ஆய்வை விட்டு விலக எனக்கு உரிமை உண்டு.

இந்த மருத்துவ ஆய்வில் சேகரிக்கப்படும் எனது சொந்த தகவல்,குறிப்பாக எனது மருத்துவ ரெகார்டுகளில் எனது பெயர் மற்றும் பாலினம் மற்றும் இனம் குறிக்கப்படும் என்பதற்கு நான் சம்மதிக்கிறேன் இந்த தகவல் ஆனது

1. எலக்ட்ரானிகல் முறையில் அல்லது ஒரு பகுதி காகித வடிவில் பதிவு செய்யப்படும் பத்திரமாக வைக்கப்படும் மற்றும் மதிப்பீடு செய்யப்படும்.
2. விஞ்ஞான மதிப்பீடு மற்றும் கூடுதல் விஞ்ஞான உபயோகத்திற்காக மற்றும் அளிக்கப்படும்.
3. உகந்த தேசிய மற்றும் சர்வதேச ரெகுலேட்டரி அதாரிட்டிகளுக்கு அனுப்பப்படும்.

இதோடு மட்டுமின்றி அங்கீகரிக்கப்பட்ட பிரதிநிதிகள் எனது சொந்த விபரங்கள் உடனான மருத்துவ ரெகார்டுகளை பரிசோதிக்கலாம். விஞ்ஞான மதிப்பீடு மற்றும் மருத்துவ ஆய்வின் செயல் திறனுக்காக தகவலை முழுமையாக சரியாகப் பரிமாற்றம் செய்ய இது உதவுகிறது.

நான் இந்த ஆய்வில் இதுவரை பங்கேற்று இருக்கவில்லை மற்றும் இந்த ஆய்வு ஆரம்பிக்கும் முன்பு 30 நாட்களில் நான் மற்றொரு ஆய்வில் பங்கேற்றிருக்கவில்லை என்பதை உறுதி செய்கிறேன்.

நோயாளிக்கான தகவல் தாளின் ஒரு அசல் உடன் கையெழுத்ததிட தகவல் அளிக்கப்பட்ட ஒப்புதல் படிவத்தை நான் பெற்றுள்ளேன்.

நோயாளி:

____________________ __________________ ____________________

பெயர் பெரிய எழுத்துகளில் கையெழுத்து தேதி

சாட்சி:

__________________________ __________________ _____________________

பெயர் பெரிய எழுத்துகளில் கையெழுத்து தேதி

நோயாளிக்கு உறவு முறை: __________________________

நான் டாக்டர் ______________________________ மேற்கண்ட பெயருடைய நோயாளிக்கு ஆய்வின் நோக்கம் மற்றும் தன்மை பற்றி விளக்கியுள்ளேன் என்பதை உறுதி செய்கிறேன். மேலும் நான் அனைத்து ஆய்வு சம்பந்தப்பட்ட கேள்விகளுக்கும் பதில்கள் அளித்துள்ளேன். மற்றும் ஆய்வின் நிபந்தனைகளை அவர்களுக்கு விளக்கியுள்ளேன் என்பதை உறுதி செய்கிறேன்.

மருத்துவர்: __________________ ____________________ ____________________

பெயர் பெரிய எழுத்துகளில் கையெழுத்து தேதி

PROFORMA

Name:	Serial No:
Age:	Lab No:
Sex:	OP/IP No:
Education:	D.O.A:
Occupation:	D.O.D:
Income:	Provisional Diagnosis:

Address:

Chief complaints:

Fever

Dysurea

Frequency

Urgency

Lower abdominal/ flank pain

H/O Present illness:

Associated conditions- instrumentation/ surgery in urinary tract

Calculi

Diabetes mellitus

Chronic kidney and liver diseases

Benign Prostatic Hypertrophy

Pregnancy

Immuno compromised state

Treatment History: H/O anti biotic intake, duration

Past History: H/O Similar episode in the past

Instrumentation/ surgery in urinary tract

Family History:

Personal History:

General Examination: Stature, nourishment, anaemia, jaundice, cyanosis, clubbing, lymphadenopathy, pedal edema.

Vital signs: Temperature, pulse rate, respiratory rate, blood pressure.

Systemic examination: Abdomen

Inspection: shape of the abdomen

Position of the umbilicus

Movements of the abdominal wall

Skin and surface of the abdomen

Palpation : Mass

Tenderness (Suprapubic)

Rigidity

Organomegaly

Percussion : Any free fluid

Auscultation : Bowel sounds

Bruit

Examination of groin and genital region

P/V:

P/R:

Examination of other systems

CVS: RS; CNS:

Definitive Diagnosis

WORKSHEET

Specimen: Urine

Method of collection : MSU/Indwelling catheter/Cystoscope/Suprapubic aspiration

I. Macroscopic Examination: Color

Turbidity

II. Microscopic Examination: Wet mount

Gram staining

III. Culture : Nutrient agar

MacConkey agar

Blood agar

CLED agar

IV. Biochemical Reactions:

Gram staining :

Motility :

Catalase :

Oxidase :

Sugar fermentation tests :

IMViC :

Urease :

TSI :

LAO :

Special Tests:

Micro organism isolated :

V. Anti Microbial Susceptibility test:

VI. Screening for ESBL : 1. Antibiogram(resistant to any one of the third generation Cephalosporins

VII. Conformation of ESBL : 1. Double disc synergy test

2. CHROM agar test

3. PCT test

4. E Test

MASTERCHART

S.NO	MICRO NO	NAME	AGE	SEX	and propable	W	I	O	ORG	GEN	AK	AMP	COT	NIT	NX	LE	C N	CXM	CX	CAZ	CTX	CPM	IMP	AMC	PIT	CFS	ERY	VAN	INTERPRET	DDST CHROM PCT ETEST	Result
1	1221	Kala	28	F	burning micturation	M		OP	E.coli	S	S	S	R	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		Non esbl
2	1222	Selvi	27	F	dysuris																								NG		
3	1223	Devi	48	F	abd pain																								NG		
4	1224	Karupu	21	M	abd pain																								NG		
5	1225	Durai	75	M	dysuria	U		OP	E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		non esbl
6	1312	Rekha	32	F	frequency	S		OP	K.p	S	S	-	R	S	S	S	R	R	R	R	R	S	S	S	S	S			SG	S S S	ESBL
7	1313	Mala	33	F	dysuria																										
8	1314	Murugan	48	M	flank pain	S	IP		CONS	-	-	S	S	R	R	-	S	-	S	-	-	-	-		-	-	S		SG		
9	1315	Kanagavel	80	M	catheterized ,fever	U	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S S S**	ESBL
10	1714	Karthi	55	F	dysuria																								NG		
11	1715	Malathi	64	F	burning micturation	M		OP	K.p	R	S	-	R	S	R	S	R	R	R	S	S	S	S	R	S	S			SG		nonesbl
12	1876	Subha	41	F	burning micturation	OG	IP		Enterococci	S(HLG)	-	S	-	S	S	-	-	-	-	-	-	-	-	-	-	-	-	S	SG		
13	1877	Priya	17	F	fever																								NG		
14	1878	Kannan	42	M	dysuria	STD		OP	Klebpneumoniae	S	s	S	R	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		
15	1879	Kavitha	71	F	frequency																										
16	2234	Senthil	73	M	urgency	M	IP		E.coli	R	S	R	S	R	R	R	R	R	R	R	R	S	S	R	S	S			SG	**S S S**	ESBL
17	2235	Raja	1	MCH	burning micturation	P	IP		S.aureus	s	-	S	S	R	R	-	-	-	S	-	-	-	-	-	-	-	R		SG		NON ESBL
18	2236	Ragavan	4	MCH	dysuria																								NG		
19	2343	Raji	44	F	burning micturation	OG	IP		Koxytoca	S	S	-	R	S	R	R	R	R	R	R	R	R	S	R	S	S			SG	S S S S	ESBL
20	2344	Renuka	16	F	lower abdominal pain																								NG		
21	2345	Ram	54	M	fever																								NG		
22	2672	Raj	68	M	burning micturation																								NG		
23	2673	Sarasu	6	FCH	dysuria	P		OP	E.coli	S	S	R	R	S	S	S	R	R	R	S	R	S	S	R	S	S			SG	**S S S**	ESBL
24	2674	Saratha	58	F	dysuria																								NG		
25	2691	Rani	7	FCH	fever																								NG		
26	2692	Sam	2	MCH	fever	P	IP		Enterococci	R(HLG)				S	S													S	SG		
27	2693	Jothi	69	F	lower abdominal pain	OG		OP	Klebоxytoca	R	R	-	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	S S S	ESBL

28	2694	Kaliammal	33	F	dysuria																								NG		
29	2789	Kannan	48	M	dysuria	M	IP		Proteus	S	S	S	R	-	S	S	S	S	S	S	S	S	S	R	S	S			SG		NON ESBL
30	2790	Devan	58	M	lower abdominal pain																								NG		
31	2791	Maruth	57	M	dysuria																								NG		
32	2792	Pandiyammal	16	F	dysuria	M	IP		Koxytoca	R	S	-	R	S	S	S	R	R	R	R	R	R	S	R	S	S			SG	S S S S	ESBL
33	2843	Pandi	41	M	burning micturation	M	IP		NGGNB	R	S	R	R	S	R	S	R	R	R	S	R	S	S	R					SG		
34	2844	Mari	46	F	fever																								NG		
35	2845	Mani	78	M	dysuria	S	IP		NFGNB	S	S	R	R	R	R	R	R	R	R	S	S	S	S	S					SG		
36	2846	Vasantha	62	F	burning micturation																								NG		
37	2847	Kumar	1M	MCH	fever	P	IP		E.coli	S	S	S	S	S	R	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
38	2880	Kumari	18	F	dysuria																								NG		
39	2881	Arasu	66	M	burning micturation																								NG		
40	2882	Rahul	11	FCH	dysuria																								NG		
41	2883	Revathy	38	F	dysuria																								NG		
42	2884	Mani	65	M	catheterized ,fever																								NG		
43	2945	Iswarya	9	FCH	lower abdominal pain																								NG		
44	2946	jey	3	MCH	fever																								NG		
45	2947	ganga	13	F	dysuria																								NG		
46	2948	mari	38	M	fever																								NG		
47	2949	ravi	41	M	lower abdominal pain	U		OP	Kleb pneumoniae	S	S	-	S	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
48	2950	selsi	5	FCH	fever	P		OP	Kleb pneumoniae	R	S	R	S	R	R	S	R	R	S	R	R	R	S	R	S	R			SG	S S S S	ESBL
49	2951	mari	57	F	dysuria																								NG		
50	2952	Kaliammal	48	M	fever																								NG		
51	3002	mani	80	M	catheterized ,fever																								NG		
52	3003	arasu	8	FCH	fever																								NG		
53	3004	mala	14	F	lower abdominal pain																								NG		

54	3005	kala	64	F	lower abdominal pain																								NG		
55	3006	devan	5	MCH	fever																								NG		
56	3007	kanmani	34	F	dysuria																								NG		
57	3008	Senthil	35	M	fever																								NG		
58	3067	Manju	63	F	lower abdominal pain																								Candida		
59	3068	Panchu	25	F	dysuria	OG		OP	Kleb pneum oniae	S	S	-	R	S	S	S	R	R	S	S	S	S	S	R	S	S			SG		
60	3069	Pandi	72	M	fever	S	IP		Proteus	R	S	R	R	-	R	R	R	R	R	R	R	S	S	R	S	S			SG	**S S S S**	ESBL
61	3070	pannai	65	M	flank pain burning micturation																										
62	3071	natchi	60	F	lower abdominal pain	S	IP		E.coli	S	S	S	R	R	R	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
63	3121	Natchim uthu	59	M																									NG		
64	3122	Anu	40	F	lower abdominal pain																								NG		
65	3123	Banu	40	F	fever																								candida		
66	3124	Mathi	47	F	dysuria																								NG		
67	3125	Ram	60	M	fever	U	IP		Klebpn eumoni ae	R	S	-	R	R	R	S	R	R	S	S	S	S	S	R	S	R			SG		NON ESBL
68	3179	Saranya	68	F																									NG		
69	3180	Sarasu	65	M	catheterized ,fever																								NG		
70	3191	Ram	21	M	dysuria																								NG		
71	3182	Radha	42	F	lower abdominal pain																								NG		
72	3183	Kumari	39	F	burning micturation	M		OP	E.coli	R	S	R	R	R	R	S	R	R	R	R	R	R	S	R	S	S			SG	**S S S**	ESBL
73	3184	Pothum	57	F	dysuria	OG		0P	E.coli	S	S	S	S	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
74	3185	Sandi	72	M	flank pain burning micturation																								NG		
75	3186	Pavai	65	F	flank pain burning micturation	S	IP		Kleb pneum oniae	R	R	R	R	R	R	S	R	R	S	R	R	R	S	R	S	S			SG	S S S	ESBL
76	3187	Vasanthi	5	FCH	fever																										

77	3241	Vasan	4	MCH	fever	p		op	K.p	R	S	R	R	S	R	R	R	R	R	R	R	S	S	S	S	S			SG	S S S S	ESBL
78	3242	Kalil	50	M	lower abdominal pain																										
79	3243	Kani	28	F	dysuria	N	IP		S.saprophyticus	-	-	R	R	R	R	-	-	-	S	-	-	-	-	-	-	-	S		SG		
80	3244	Kannan	64	M	flank pain burning micturation	U	IP		E.coli	R	R	R	R	S	R	S	R	R	R	R	R	R	S	R	S	S			SG	**S S S**	ESBL
81	3245	Angel	6	FCH	fever																								NG		
82	3246	Selvan	34	M	dysuria	M		OP	Klebpneumoniae	R	S	-	R	S	R	S	R	R	R	R	R	R	S	R	S	S			SG	S S S	ESBL
83	3247	Selvi	8	FCH	fever	P	IP		NFGNB	S	S	R	R	R	R	S	R	R	R	S	R	S	S	R					SG		
84	3248	Ponni	27	F	fever																								NG		
85	3249	Vijaya	66	F	dysuria																								NG		
86	3250	Vidhya	9	FCH	burning micturation	P	IP		E.coli	S	S	S	S	S	S	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
87	3251	Babu	3	MCH	fever																								NG		
88	3252	Kali	80	M	catheterized ,fever																								NG		
89	3253	Malliga	38	F	burning micturation																								NG		
90	3254	Kamala	16	F	burning micturation																								NG		
91	3255	Rathina	56	F	lower abdominal pain	M	IP		K.pneumoniae	R	S	-	R	S	R	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
92	3256	Kutti	1	MCH	fever																								NG		
93	3257	Krishnan	14	M	lower abdominal pain																								NG		
94	3258	Raja	50	M	lower abdominal pain																								NG		
95	3259	Pooja	64	F	dysuria																								NG		
96	3260	Rathina	13	F	fever																								NG		
97	3291	Sekar	75	M	catheterized ,fever																								NG		
98	3292	Rani	18	F	lower abdominal pain	M	IP		E.coli	R	S	R	R	S	R	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
99	3293	Kani	63	F	fever																								NG		
100	3294	James	63	M	catheterized ,fever																								NG		

101	3295	James	46	M	lower abdominal pain	M	IP		C.koseri	S	S	-	S	S	S	S	-	-	S	S	S	S	S	R	S	S			SG		NON ESBL
102	3296	Kattan	67	M	urgency	M	IP		Proteus	S	S	S	R	-	R	R	R	R	R	S	S	S	S	S	S	S			SG		NON ESBL
103	3297	Selvi	28	F	dysuria																										
104	3298	Ambika	53	F	urgency	OG		OP	E.coli	R	R	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S S S**	ESBL
105	3312	Baby	68	F	dysuria																								NG		
106	3313	Bala	47	M	lower abdominal pain	M	IP		Kleb pneumoniae	R	R	R	R	R	R	S	R	R	S	R	R	S	S	R	S	R			SG	S S S S	ESBL
107	3314	Kumar	62	M	dysuria																								NG		
108	3315	Chinnu	21days	MCH	fever	P	IP		E.coli	R	R	R	R	S	R	S	R	R	S	R	R	S	S	R	S	S			SG	**S S S**	ESBL
109	3316	Mega	41	M	dysuria																								NG		
110	3317	Janani	11	FCH	lower abdominal pain																								NG		
111	3318	Dhanya	19	F	urgency																								NG		
112	3319	Jothi	10	FCH	dysuria																								NG		
113	3320	Vijaya	14	M	lower abdominal pain	M	IP		Keeb pneumoniae	R	S	-	R	R	R	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
114	3371	Sethu	70	M	flank pain burning micturation																								NG		
115	3372	Sethupathy	61	M	catheterized ,fever																								NG		
116	3373	Anush	9	FCH	urgency																								NG		
117	2274	Vannan	69	M	dysuria																								NG		
118	2275	Vani	66	F	urgency	OG		OP	Klebpneumoniae	S	S	-	R	S	R	S	S	S	S	S	S	S	S	R	S	S			SG		NON ESBL
119	3376	Shree	29	F	dysuria	S	IP		S.saprophyticus	-	-	R	R	s	s	-	-	-	S	-	-	-	-	-	-	-	R		SG		
120	3377	Kohi	54	F	urgency																								NG		
121	3378	Sound	69	M	dysuria																								NG		
122	3379	Kohila	8	FCH	dysuria	P	IP		Kleb pneumoniae	R	S	-	R	S	R	S	R	R	S	R	R	R	S	R	R	R			SG		NON ESBL
123	3380	Sankara	59	M	urgency																								NG		
124	3381	Anu	28	F																									NG		
125	3382	Ragu	28	M	lower abdominal pain																								NG		

126	3383	Badhri	2	MCH	fever	P	IP		E.coli	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S			SG					NON ESBL
127	3384	Raja	65	M	flank pain burning micturation	S	IP		Proteus	R	S	R	R	-	R	S	R	R	R	R	R	S	S	R	S	S			SG	**S**	**S**	**S**	**S**	ESBL
128	3385	Pitchai	64	F	urgency																								NG					
129	3421	kala	16	F	dysuria																								NG					
130	3422	Saranya	44	F	urgency	S		OP	Klebpneumoniae	S	S	-	R	S	S	S	R	R	R	S	S	S	S	S	S	S			SG					NON ESBL
131	3423	Gowri	16	F	catheterized ,fever	M	IP		Enterobacter	S	S	-	S	R	R	S	-	-	-	S	S	S	S	R	R	S			SG					NON ESBL
132	3424	Prakash	67	M	urgency																								NG					
133	3425	Lalith	2	MCH	fever																								NG					
134	3426	Veeram	63	F	urgency																								NG					
135	3427	Veeram	70	M	flank pain burning micturation	S	IP		E.coli CoNS	S -	S -	R S	R S	S R	R R	R -	R -	R -	S S	R -	R -	R -	S -	R -	S -	S -	S -		SG		**S**	**S**	**S**	ESBL
136	3428	Kattan	74	M	dysuria																								NG					
137	3429	Chellam	1	MCH	fever																								NG					
138	3430	Ponni	18	F	urgency																								NG					
139	3471	Rajini	41	M	urgency																								NG					
140	3472	Prakash	40	M	lower abdominal pain																								NG					
141	3473	Chellam	2	FCH	fever	p	IP		E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG					NON ESBL
142	3474	Ponnu	27	F	dysuria	OG	IP		S,saprophyticus	-	-	S	S	S	R	-	-	-	S	-	-	-	-	-	-	-	R		SG					
143	3475	Kannu	60	F	dysuria	M	IP		CoNS	-	-	R	R	R	S	-	-	-	S	-	-	-	-	-	-	-	S		SG					
144	3476	Ravi	58	M	urgency	S	IP		E.coli	R	S	R	R	R	R	S	R	R	S	S	S	S	S	S	S	S			SG					NON ESBL
145	3477	Gnana	67	F	dysuria																								NG					
146	3478	Mani	72	M	catheterized ,fever																								NG					
147	3479	Devar	58	M	urgency																								NG					
148	3480	Pavai	29	F	urgency																								NG					
149	3521	Nagan	42	M	urgency	STD		OP	E.coli	S	S	R	R	S	S	S	R	R	S	R	R	R	S	S	S	S			SG		**S**	**S**	**S**	ESBL

150	3522	Malai	68	M	urgency	U		OP	Ckoseri	R	S	-	R	R	R	S	-	-	S	S	S	S	S	R	S	S			SG		NON ESBL
151	3523	Arasu	39	M	urgency																								NG		
152	3524	Ganse	69	M	flank pain burning micturation																								NG		
153	3525	Naga	41	F	dysuria	OG	IP		E.coli	R	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
154	3526	Nagan	69	M	urgency																								NG		
155	3527	Kannan	48	M	dysuria																								NG		
156	3528	Selvai	5	FCH	fever																								NG		
157	3529	Sam	1	FCH	fever	P	IP		E.coli	S	S	R	S	S	S	S	R	R	S	R	R	S	S	S	S	S			SG	**S** **S** **S**	ESBL
158	3641	Kannu	68	F	urgency	M	IP		**E.coli**	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	S	S			SG	S S S S	ESBL
159	3644	Chiinu	29	M	lower abdominal pain																								NG		
160	3645	Palani	60	M	urgency																								NG		
161	4646	Pavai	40	F	urgency																								NG		
162	3647	Pavithra	47	F	urgency																								NG		
163	3648	Suresh	65	M	catheterized ,fever	STD		OP	E.coli	R	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
164	3649	Divya	39	F	urgency	M	IP		E.coli	S	S	R	R	R	R	S	R	R	S	R	R	S	S	R	S	R			SG	**S** **S** **S** **S**	ESBL
165	3650	Soloman	68	M	fever	U	IP		E.coli	R	S	R	R	S	R	S	S	S	S	S	S	S	S	R	S	S			SG		NON ESBL
166	3693	Suganya	66	F	urgency																								NG		
167	3694	Sundhar	58	M	urgency																								NG		
168	3695	Dinesh	1	MCH	fever																								NG		
169	3696	Ranmesh	59	M	urgency																								NG		
170	3697	Swathi	3	MCH	fever	P	IP		NFGNB	S	S	R	S	R	R	S	R	R	R	S	R	S	S	R					SG		
171	3698	Meena	42	F	lower abdominal pain																								NG		
172	3699	Saroja	48	F	dysuria	OG	IP		E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
173	3700	Raju	65	M	fever																								NG		

174	3708	Suresh	9	FCH	fever	P		OP	E.coli	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S.			SG					NON ESBL
175	3709	Nambi	66	M	catheterized ,fever																								NG					
176	3810	Jithan	2	MCH	fever																								NG					
177	3811	kumutha	38	F	dysuria																								NG					
178	3812	Divya	59	F	urgency																								NG					
179	3813	Vishali	8	FCH	fever	P	IP		E.coli, NFGNB	S R	S S	R R	R R	S S	R R	S S	R S	R S	S S	R S	R S	S S	S S	R S	S S	S S			SG		**S**	**S**	**S**	ESBL
180	3814	Bharath	39	M	urgency	M	IP		Klebpneumoniae	R	S	R	R	S	R	R	R	R	R	R	R	R	S	R	R	R			SG		S	S	S	ESBL
181	3815	Mant	48	M	lower abdominal pain																								NG					
182	3816	Sumo	4	MCH	fever																								NG					
183	3817	Gaya	37	F	dysuria																								NG					
184	3818	ponni	46	F	urgency	OG	IP		E.coli	R	S	R	R	S	S	S	R	R	R	S	S	S	S	S	S	S			SG					NON ESBL
185	3819	Chandra	4	FCH	fever																								NG					
186	3867	Mahesh	59	M	urgency	S	IP		Enterococci	S(HLG)		S	-	S	S	-	-	-	-	-	-	-	-	-	-	-	-	S	SG					
187	3868	pannu	64	F	dysuria																								NG					
188	3869	Baby	6	FCH	dysuria	P	IP		E.coli	R	S	R	R	R	R	R	R	R	R	S	S	S	S	S	S	S			SG					NON ESBL
189	3870	Kavya	36	F	fever																								NG					
190	3871	Bharathi	65	F	urgency																								NG					
191	3872	Praddep	69	M	dysuria																								NG					
192	3873	Pandi	70	M	urgency	S	IP		E.coli	R	R	R	R	S	R	R	R	R	S	R	R	S	S	R	R	S			SG	**S**	**S**	**S**	**S**	ESBL
193	3874	Kannan	14	M	dysuria																								NG					
194	3875	Jithu	7	FCH	dysuria																								NG					
195	3876	Bakkya	39	F	dysuria	OG		OP	S.sapro K pneu	R	S	R	S R	R R	S R	R	R	R	S R	R	R	S	S	R	S	R	R -		SG	S	S	S	S	ESBL
196	3877	Selvi	8	FCH	lower abdominal pain	P		OP	E.coli	S	S	S	S	S	R	S	S	S	S	S	S	S	S	S	S	S			SG					NON ESBL
197	3879	Priya	18	F	fever																													
198	3880	Suresh	63	M	fever	S	IP		E.coli	R	S	R	S	S	R	R	R	R	S	R	R	R	S	R	S	S			SG	**S**	**S**	**S**	**S**	ESBL

199	3895	Suriya	11	MCH	lower abdominal pain	P		OP	Kleb pneu	S	S	-	R	S	S	S	R	R	S	R	R	R	S	S	S	S			SG		NON ESBL
200	3896	Mari	59	F	fever	OG	IP		CONS	-	-	S	R	R	R	-	-	-	S	-	-	-	-	-	-	-	S		SG		
201	3897	Mohammad	43	M	dysuria	M		OP	E.coli	S	S	R	R	S	R	R	R	R	R	S	S	R	S	R	S	S			SG		NON ESBL
202	3898	Beham	63	F	fever																								NG		
203	3890	Bavi	38	F	dysuria																								NG		
204	3900	Kavin	9	MCH	fever	P	IP		NFGNB	S	S	R	R	R	R	R	R	R	R	S	R	S	S	R					SG		
205	3911	Bavith	38	F	fever	S	IP		NFGNB	R	S	R	R	\R	R	R	R	R	R	S	S	S	S	R					SG		
206	3912	Arasu	62	M	catheterized ,fever	S	IP		Ecoli	R	R	R	R	S	R	S	R	R	S	R	R	S	S	R	S	S			SG	**S** **S** **S**	ESBL
207	3913	Siva	29	M	fever																								NG		
208	3914	Prathap	47	M	dysuria	M	IP		E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
209	3915	Monica	8	FCH	fever																								NG		
210	3916	Ravi	48	M	lower abdominal pain																								NG		
211	3918	Sudha	3	FCH	fever																								NG		
212	3967	Priya	28	F	dysuria																								candida		
213	3968	Sundar	65	M	fever																								NG		
214	3969	Selvan	67	M	catheterized ,fever																								NG		
215	3970	Ranji	43	F	dysuria	OG	IP		E.coli	R	R	R	R	S	R	S	R	R	S	R	R	R	S	R	S	R			SG	**S** **S** **S** **S**	ESBL
216	3971	Rajam	64	F	lower abdominal pain	OG		OP	Kleboxytoca	S	S	-	R	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
217	3972	Dev	46	M	dysuria	STD		OP	K.pneumoniae	S	S	-	R	R	R	R	R	R	S	R	R	R	S	R	S	S			SG		NON ESBL
218	3973	Dilip	4	FCH																									NG		
219	3974	Ajith	28	M	lower abdominal pain																								NG		
220	3985	Jothika	6	FCH	fever																								NG		
221	3986	Karthi	38	M	dysuria	S	IP		E.coli	R	S	R	R	S	R	R	R	R	R	R	R	S	S	R	S	S			SG		NON ESBL
222	3987	Kali	40	F	dysuria	OG	IP		Enterococci	S(HLG)	-	S	-	S	S	-	-	-	-	-	-	-	-	-	-	-		S	SG		
223	3988	Meena	44	F	fever																								NG		

224	3989	Kumari	19	F	urgency																								NG		
225	4000	Sugu	7	FCH	dysuria	P	IP		S.aureus	-	-	S	S	S	S	-	-	-	S	-	-	-	-	-	-	-	S		SG		
226	4004	Manna	46	F	dysuria																								NG		
227	4005	Lallu	2	MCH	fever	P	IP		S.aureus	-	-	R	S	S	S	-	-	-	R	-	-	-	-	-	-	-	S		SG		
228	4006	saroja	68	M	catheterized ,fever	M	IP		E.coli	R	S	R	R	S	R	S	R	S	R	R	R	R	S	R	R	S			SG	**S S S**	ESBL
229	4007	mani	9	MCH	lower abdominal pain																								NG		
230	4008	Chitu	2	MCH	dysuria																								NG		
231	4009	Kannan	19	F	dysuria	M	IP		NFGNB	S	S	R	R	R	R	S	R	R	R	R	R	R	S	R					SG		
232	4010	Divya	8	FCH	lower abdominal pain	P	IP		CoNS Enterococci	S(HLG)	-	R S	S -	R S	R S	-	-	-	S -	-	-	-	-	-	-	-	S -	S	SG		
233	4012	Ponni	70	F	dysuria	OG	IP		E.coli	R	R	R	R	S	R	S	R	R	S	R	R	R	S	R	R	S			SG	**S S S S**	ESBL
234	4014	Sam	2	MCH	fever																								NG		
235	4016	Kamal	68	M	dysuria	S	IP		E.coli	R	S	R	R	S	R	S	R	R	R	R	R	R	S	R	R	R			SG		NON ESBL
236	4017	Amala	24	F	lower abdominal pain	S	IP		K.oxytoca	R	S	R	R	S	R	S	R	R	S	R	R	S	S	R	S	R			SG	S S S S	ESBL
237	4018	Kala	9	FCH	fever	P	IP		NFGNB	S	S	R	R	R	R	R	R	R	R	R	R	R	S	R	S	S			SG		
238	4019	Nambi	58	M	lower abdominal pain																								NG		
239	4020	Rama	47	F	fever	OG	IP		K.pneumoniae	R	S	-	R	S	R	S	R	S	S	S	S	S	S	S	S	S			SG		NON ESBL
240	4034	Suria	46	M	lower abdominal pain																								NG		
241	4035	Kumar	66	M	lower abdominal pain																								NG		
242	4036	Kaniya	64	M	lower abdominal pain																								NG		
243	4037	Suriya	11	MCH	fever																								NG		
244	4038	Pavia	28	F	lower abdominal pain																								candida		

245	4039	Nalayini	4	FCH	lower abdominal pain	P	IP		E.coli	S	S	R	R	S	S	S	R	R	S	R	R	S	S	R	S	S			SG	**S S S**	ESBL
246	4040	Kattu	68	M	catheterized ,fever																								NG		
247	4041	Matti	58	M	lower abdominal pain																								NG		
248	4042	Dilip	4	FCH	fever																								NG		
249	4054	Chinna	59	F	fever																								NG		
250	4055	Alagu	7	FCH	fever																								NG		
251	4056	Beham	68	F	catheterized ,fever																								NG		
252	4057	Devan	8	MCH	fever																								NG		
253	4058	Rosy	29	F	burnig micturation	M	IP		E.coli	S	S	S	S	S	R	S	S	S	S	S	S	S	S	R	S	S			SG		NON ESBL
254	4059	Bala	29days	MCH	suprapubic aspirate																								NG		
255	4060	Rosline	7	FCH	burnig micturation																								NG		
256	4074	shan	68	M	catheterized ,fever	U	IP		E.coli	S	S	R	R	S	R	S	R	R	R	S	S	S	S	R	S	S			SG		NON ESBL
257	4075	mathi	6	FCH	burnig micturation																								NG		
258	4076	Santhanam	46	M	burnig micturation																								NG		
259	4077	Selvi	40	F	lower abdominal pain	M	IP		Proteus	R	S	R	R	-	R	R	R	R	R	S	S	S	S	R	S	S			SG		NON ESBL
260	4078	Sundar	38	M	burnig micturation	STD		OP	K.pneumoniae	R	S	R	R	S	R	S	R	R	S	R	R	S	S	R	S	S			SG	S S S	ESBL
261	4079	Anil	1	MCH	fever	P	IP		E.coli	S	S	R	R	S	S	S	R	R	S	R	R	S	S	R	S	S			SG	**S S S**	ESBL
262	4108	Janani	11	FCH	burnig micturation	P		OP	E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
263	4109	Dev	29	M	fever	M	IP		E.coli	R	S	R	R	S	R	R	R	R	S	R	R	S	S	R	S	S			SG	**S S S S**	ESBL
264	4110	Shoba	27	F	fever																										
265	4112	Shathi	30	F	lower abdominal pain	S	IP		NFGNB	R	R	R	R	R	R	R	R	R	R	S	S	S	S	R					SG		
266	4113	Palani	67	F	catheterized ,fever																								NG		
267	4114	Naveen	2	MCH	fever																								NG		

268	4115	Kanayiram	70	M	catheterized ,fever	U	IP		E.coli	S	S	R	R	S	S	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
269	4119	Kaveen	4	MCH	lower abdominal pain																								NG		
270	4145	maru	66	M	burnig micturation																								NG		
271	4146	Megala	8	FCH	fever	P	IP		E.coli	R	S	R	R	S	S	S	R	R	S	R	R	S	S	R	S	S			SG	**S S S**	ESBL
272	4147	Deva	58	M	fever																								NG		
273	4148	Samuth	29	F	lower abdominal pain																								NG		
274	4149	Sanmathi	7	FCH	fever																								NG		
275	4150	Sami	68	M	burnig micturation	S		OP	K.oxytoca	R	R	-	R	S	R	R	R	R	R	S	S	S	S	S	S	S			SG		NON ESBL
276	4156	Selvi	7	FCH	lower abdominal pain	P	IP		E.coli	S	S	R	R	S	R	R	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
277	4157	Boomi	38	M	burnig micturation																								NG		
278	4158	Kala	18	F	lower abdominal pain																								NG		
279	4159	Kavi	5	FCH	flank pain	P	IP		CoNS, Enterococci	S	-	S S	S -	S S	S S	-	-	-	S -	-	-	-	-	-	-	-	S -	S	SG		
280	4160	Ammu	35	F	burnig micturation	S		OP	E.coli	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
281	4167	s\Sathaya	8	FCH	flank pain																								NG		
282	4168	Savi	57	F	catheterized ,fever																								NG		
283	4169	Ravi	8	MCH	flank pain																								NG		
284	4170	Malayan	65	F	burnig micturation	OG	IP		E.coli	S	S	R	R	R	R	R	R	R	R	R	R	R	S	S	S	S			SG		NON ESBL
285	4178	Devi	19	F	flank pain																								NG		
286	4179	Kannan	46	M	burnig micturation	M	IP		E.coli	S	S	R	R	S	R	R	R	R	R	S	S	S	S	S	S	S			SG		NON ESBL
287	4180	Ponnam	40	M	flank pain																								NG		
288	4196	rethu	10	FCH	flank pain																								NG		

289	4197	Kavitha	38	F	burnig micturation																								NG		
290	4198	sai	27	F	lower abdominal pain																								NG		
291	4199	Naga	48	F		OG	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	S	S	R	S	S			SG	**S S S S**	ESBL
292	4207	Malai	69	M	dysuria																								NG		
293	4208	Andi	35	M	lower abdominal pain	M	IP		CoNs	-	-	R	R	S	-	-	-	S	-	-	-	-	-	-	-	-	R		SG		
294	4209	Arasi	40	F	dysuria	OG	IP		E.coli	S	S	R	R	S	S	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
295	4210	Roopa	11	FCH	dysuria																								NG		
296	4212	Jothika	38	F	dysuria	N	IP		Enterococci	S(HLG)	-	S	-	S	S	-	-	-	-	-	-	-	-	-	-	-	-	S	SG		
297	4213	Kala	46	F	dysuria																										
298	4215	Manu	5	FCH	dysuria	P	IP		S.aureus	-	-	S	S	S	S	-	-	-	S	-	-	-	-	-	-	-	S -		SG		
299	4216	Chinna	64	M	dysuria	M		OP	E.coli	S	S	R	S	S	S	S	S	S	S	S	S	S	S	S	S	S			SG		NON ESBL
300	4217	Palani	6	MCH	dysuria																								NG		
301	4218	Priya	4	FCH	fever																								NG		
302	4219	Pathran	8	MCH	lower abdominal pain																								NG		
303	4220	Andi	18	M	fever																								NG		
304	4221	Ravi	8	MCH	fever																								NG		
305	4226	Solai	40	F	fever																								NG		
306	4227	Mari	64	F	fever																								NG		
307	4228	Mani	38	M	fever	M	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	R	S			SG	**S S S**	ESBL
308	4229	Sara	21	F	lower abdominal pain																								NG		
309	4230	Selva	39	M	dysuria																								NG		
310	4236	Sarasan	9	MCH	dysuria																								NG		
311	4235	Vasanthi	19	F	dysuria																								NG		
312	4237	Kavya	7	FCH	lower abdominal pain	P	IP		K.pneumoniae	R	R	R	R	S	R	S	R	R	S	R	R	S	S	R	S	S			SG	S S S S	ESBL
313	4239	Priya	8	FCH	flank pain																								NG		
314	4245	Kavin	18	MCH	burnig micturation																								NG		
315	4246	Selvi	27	F	flank pain																								NG		

316	4247	Selsi	7	FCH	lower abdominal pain																								NG					
317	4248	Ammatha	63	F	lower abdominal pain	OG	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	R	S			SG		**S**	**S**	**S**	ESBL
318	4252	Malayan	68	M	catheterized ,fever	M	IP		CoNS	-	-	R	R	R	R	-	-	-	S	-	-	-	-	-	-	-	S		SG					
319	4253	Pappu	8	FCH	dysuria	P	IP		K.pneumoniae	R	S	-	R	S	R	S	R	R	S	S	S	S	S	R	S	S			SG					NON ESBL
320	4254	Pandi	47	M	dysuria																								NG					
321	4255	Ammu	10	FCH	dysuria																								NG					
322	4256	Palani	38	M	dysuria	S	IP		E.coli	S	S	R	R	S	R	S	R	R	R	R	R	R	S	R	R	R			SG					NON ESBL
323	4257	Kannath	26	F	fever																								NG					
324	4258	Selvi	44	F	fever																								NG					
325	4265	Roopa	9	FCH	lower abdominal pain	P	IP		S.aureus	S	-	S	S	S	S	-	S	-	S	-	-	-	-	S	-	-			SG					
326	4266	Vijayan	11	MCH	burnig micturation																								NG					
327	4267	Malai	67	M	catheterized ,fever	U	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S**	**S**	**S**	**S**	ESBL
328	4268	Kali	32	F	burnig micturation	M	IP		S.saprophyticus	-	-	S	R	S	S	-	-	-	S	-	-	-	-	-	-	-	S		SG					
329	4269	Arasi	17	F	fever																													
330	4270	Naveen	9	MCH	dysuria	P		OP	K.oxytoca	S	S	-	R	R	R	S	R	R	S	S	S	S	S	S	S	S			SG					NON ESBL
331	4276	Andi	68	M	dysuria	U	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S**	**S**	**S**	**S**	ESBL
332	4277	Ponnan	58	M	dysuria																								NG					
333	4278	Sound	38	M	burnig micturation	M		OP	E.coli	S	S	R	S	R	S	S	R	S	S	S	S	S	S	S	S	S			SG					NON ESBL
334	4279	Kamala	35	F	dysuria																								candida					
335	4280	Eswar	67	M	catheterized ,fever																								NG					
336	4284	Rama	19	F	dysuria																								NG					
337	4285	Ramachand	60	M	dysuria																								NG					
338	4286	Chandru	7	MCH	dysuria																								NG					
339	4287	Mandra	33	F	urgency	M	IP		E.coli	R	S	R	R	S	R	S	R	R	R	S	S	S	S	S	S	S			SG					NON ESBL

340	4288	Manikam	43	M	burnig micturation	M	IP		S.aureus	-	-	R	R	R	R	-	-	-	S	-	-	-	-	-	-	-	S		SG		
341	4294	Seetha	48	F	dyuria																								NG		
342	4295	Megala	64	M	urgency	M	IP		E.coli	R	S	R	R	S	R	S	R	R	R	S	S	S	S	S	S	S			SG		NON ESBL
343	4296	Sen	6	MCH	urgency																								NG		
344	4298	Kavya	29	F	urgency																								NG		
345	4301	amman	65	F	urgency	OG	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S S S S**	ESBL
346	4302	Deva	66	M	catheterized ,fever																								NG		
347	4306	Pandi	70	M	frequency																								NG		
348	4307	Palani	39	M	frquency	M		OP	E.coli	S	S	R	S	S	S	S	R	R	R	R	R	S	S	R	R	R			SG		NON ESBL
349	4308	Manickam	64	M	catheterized ,fever																								NG		
350	4309	Arasi	31	F	burnig micturation																								NG		
351	4310	Veeram	54	M	dyuria																								NG		
352	4314	Ponni	58	F	burnig micturation																								NG		
353	4315	Kanmani	30	F	burnig micturation	M	IP		E.coli	R	S	R	R	R	R	S	R	R	R	R	R	R	S	R	R	R			SG		NON ESBL
354	4316	Selva	9	MCH	burnig micturation																								NG		
355	4317	Kavin	8	MCH	fever																								NG		
356	4319	Sela	39	F	urgency																								NG		
357	4320	Mani	46	M	burnig micturation																								NG		
358	4324	Devi	47	F	frequency																								NG		
359	4325	Pavi	65	M	urgency	S		OP	E.coli	S	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
360	4327	Kumari	64	F	urgency																										
361	4328	Kaviya	29	F	urgency	M	IP		E.coli	R	S	R	R	S	R	S	R	R	S	R	R	R	S	R	S	S			SG	**S S S**	ESBL
362	4329	Ganesh	70	M	urgency	M	IP		E.coli	R	S	R	R	S	S	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESBL
363	4330	Krishnan	67	M	urgency																								NG		
364	4331	Veni	58	F	urgency																								NG		

365	4335	Nalini	29	F	burnig micturation	M		OP	Kpneu moniae	R	S	-	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESB L
366	4336	Jeya	58	F	frequency																										
367	4337	Ananthi	17	F	frequency	M	IP		E.coli	R	S	R	R	S	R	R	R	R	S	R	R	S	S	R	S	S			SG	**S S S S**	ESB L
368	4338	Somu	66	M	catheterized ,fever																								NG		
369	4339	Sundari	18	F	frequency																								NG		
370	4342	Mani	57	M	frequency																								NG		
371	4343	Kandan	66	M	urgency	N	IP		E.coli	R	S	R	R	S	S	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESB L
372	4344	Selvan	38	M	urgency	M		OP	E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESB L
373	4345	Selvi	59	F	burning micturation																								NG		
374	4346	Kaniya	48	F	frequency																								NG		
375	4347	Sugu	28	F	burning micturation																								NG		
376	4348	Nagy	19	F	frequency																								NG		
377	4349	Nagam	68	F	urgency	M	IP		E.coli	R	S	R	R	S	R	R	R	R	S	R	R	S	S	R	S	R			SG	**S S S S**	ESB L
378	4350	Pavi	68	F	urgency																								NG		
379	4351	Banu	69	F	urgency																								NG		
380	4352	Kavin	2	MCH	fever																								NG		
381	4353	Pappan	58	M	flank pain																								NG		
382	4355	Uma	30	F	burning micturation	M	IP		CoNS NFGN B	S	S	S R	S R	R R	R R	R	R	R	S S	S	S	S	S	S			S -		SG		NON ESB L
383	43357	Mahesh	2	MCH	fever	P	IP		E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESB L
384	4358	Kumar	69	M	burning micturation																										
385	4359	Kalan	65	M	fever	M	IP		E.coli	R	S	R	R	S	R	R	R	R	S	R	R	S	S	R	S	R			SG	**S S S S**	ESB L
386	4360	Dhana	38	F	flank pain																								NG		
387	4361	Lakshmi	32	F	urgency																								NG		
388	4362	Pavithra	48	F	urgency																								NG		
389	4365	Maniya	58	F	fever																								NG		
390	4366	Rathiya	65	F	flank pain	U	IP		Proteus	R	R	R	R	-	R	S	R	R	S	S	S	S	S	R	S	S			SG		NON ESB L

391	4367	Ranji	32	F	burning micturation	OG		OP	E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL
392	4368	Manna	68	M	urgency	N	IP		E.coli	R	S	R	R	S	R	S	R	R	R	S	S	S	S	S	S	S			SG		NON ESBL
393	4369	Mali	34	F	urgency																								NG		
394	4370	Ponnu	64	F	urgency																								NG		
395	4371	Kala	42	F	flank pain	OG	IP		E.coli	R	S	R	R	R	R	S	R	R	S	S	R	S	S	R	S	S			SG		NON ESBL
396	4372	Selvi	28	F	dysuria																								NG		
397	4373	Vani	26	F	urgency																								NG		
398	4374	Anu	38	F	urgency	OG	IP		E.coli	R	S	R	R	S	R	R	R	R	S	R	R	R	S	R	S	R			SG	**S S S S**	ESBL
399	4375	Dhanu	34	F	urgency																								NG		
400	4376	Mohan	43	F	burning micturation	OG	IP		E.coli	R	S	R	R	S	R	S	R	R	S	S	S	S	S	S	S	S			SG		NON ESBL

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Course : PG in MD, MICROBIOLOGY

Period of Study : 2014-2017

College : MADURAI MEDICAL COLLEGE

Research Topic : A STUDY ON COMPARISON OF DIFFERENT PHENOTYPIC METHODS FOR THE DETECTION OF EXTENDED SPECTRUM BETA LACTAMASE AMONG ENTEROBACTERIACEAE IN URINARY TRACT INFECTION IN A TERTIARY CARE CENTRE.

Ethical Committee as on : 11.01.2016

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

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