"ASSOCIATION OF URINARY TRACT INFECTION AS A RISK FACTOR FOR PREECLAMPSIA: A CROSS SECTIONAL STUDY"

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LIST OF ABBREVIATIONS

UTI	–	Urinary Tract Infection
PE	–	Preeclampsia
ASB	–	Asymptomatic Bacteriuria
OR	–	Odds Ratio
CI	–	Confidence Interval
STROBE	–	Strengthening the Reporting of Observational study in Epidemiology
SIRS	–	Systemic Inflammatory Response
sFlt-1	–	Soluble fms-like Tyrosine kinase 1
sEng	–	Soluble endoglin
EVT	–	Extravascular trophoblasts
TGF- B	–	Tumor necrosis factor Beta
PlGF	–	Placental growth factor
VEGF	–	Vascular endothelial growth factor
NO	–	Nitric- oxide
FGR	–	Fetal growth restriction
IL-6	–	Interleukin-6
TNF- α	–	Tumor necrosis factor alpha
STBM's	–	Syncytiotrophoblast microparticles
PGI2	–	Prostacyclin
RAS	–	Renin-angiotensin system

ABSTRACT

Background

Preeclampsia (PE) in pregnancy is a serious cause of maternal and perinatal morbidity. Urinary tract infection (UTI) is a common source of inflammatory burden on the mother in pregnancy, as inflammation plays a crucial role in the pathogenesis of PE when the body cannot compensate for this extra inflammatory burden, PE becomes more likely. With appropriate screening and treatment of UTI, this morbidity can be significantly reduced.

Objective

To find an association between urinary tract infection and Preeclampsia.

Materials & Methods

This prospective case-control study which was conducted between Mach 2021 to February 2022 at KLE's Dr. Prabhakar Kore Charitable Hospital, Belagavi. Pregnant women with singleton pregnancy between 11-20 weeks of gestation attending antenatal OPD or admitted in the hospital at were advised to give midstream, clean catch urine samples on the day of enrollment for Urine culture testing. Based on the result of this women were divided into cases who had positive findings on urine culture and into controls who did not isolate any organism on culture.

Result:

505 women were screened for UTI and out of them 110 were diagnosed with UTI with a prevalence rate of 21.7% in our institute. A total of 239 participants were enrolled in the study out of which, 110 were cases diagnosed with UTI and 129 women belonged control group without UTI. All 239 participants in the study were followed up till delivery to look for pregnancy outcomes like development of gestational hypertension, preeclampsia, eclampsia, HELLP syndrome. Out of the 110 participants in UTI group 21.82% (24) participants developed hypertension in pregnancy. Whereas out of the 129 participants in the control group, only 9.3% (12) participants developed hypertension in pregnancy. Suggesting that the incidence of hypertension in pregnancy was more in the UTI group than in the control group with a $P=0.007^{MC}$ which is statistically significant. (14) 58.33% participants from the UTI group developed gestational hypertension and (10) 41.67% participants developed Preeclampsia which was statistically comparable to the control group in which (10) 66.67% participants developed Gestational hypertension and (4) 33.33% developed PE. Thus, there was no statistically significant association between development of UTI and PE.

Conclusion

There was a significant association between UTI and development of hypertension in pregnancy.

Keywords

Preeclampsia, Urinary Tract Infection, Hypertension in pregnancy, Gestational Hypertension.

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INTRODUCTION

Pre-eclampsia (PE) is a multi-system vascular syndrome of pregnancy which is characterized by new onset hypertension and proteinuria or new onset hypertension with end-organ dysfunction with or without proteinuria, occurring after 20 weeks of gestation.[1]

The additional signs and symptoms suggestive of end organ damage that might be associated with PE include visual disturbances, nausea, vomiting, headache, epigastric pain, thrombocytopenia, and abnormal liver functions. These clinical and biochemical manifestations are a result of the microangiopathy at the level of various end organs like the brain, liver, kidney and placenta.[1]

In India, the incidence of PE is approximated to be 8-10% among pregnant women. According to a study conducted in India, the prevalence of hypertensive disorders of pregnancy was found to be 7.8% and 5.4% for preeclampsia.[2]

The etiology of PE is considered to be multi-factorial. It is postulated that the placental insufficiency and reduced blood flow in the intervillous spaces of the placenta in early pregnancy leads to the release of anti-angiogenic factors into the maternal circulation. The pathophysiology of preeclampsia is primarily due to incomplete modification of the uterine spiral arteries (uteroplacental arteries). This abnormality hampers the development of the placental vasculature thus causing placental under perfusion/ hypoxia/ ischemia. This leads to release of anti-angiogenic factors into the maternal circulation which causes endothelial injury. [3]

Any factor that increases the maternal systemic inflammatory response (SIRS), such as any infection, adding to the overall inflammatory burden and hence can lead to the development of preeclampsia.[1]

The association between infection or inflammation and the development of preeclampsia could be at the initiation of preeclampsia (by increasing the of acute uteroplacental atherosis) and/or it can cause potentiation of the disease (by amplifying the existing maternal systemic inflammatory response). It is however unclear, whether inflammation occurs before development of preeclampsia or is a consequence of preeclampsia itself. [4]

Although some studies have demonstrated that the inflammatory response in preeclampsia exceeds to that in any normal pregnancy. Some studies have found increase in the levels of inflammatory cytokines, such as prostaglandin E2 (PGE2), tumor necrosis factor (TNF)-a, interleukin 1 (IL-1), interleukin 6 (IL-6), soluble phospholipase A2, activated clotting and complement pathways, as well as C-reactive protein (CRP) in women with preeclampsia as compared to normotensive women.[5]

Over the years studies have shown that there could be some association between various maternal infections and adverse pregnancy outcomes such as Preeclampsia, Intra uterine fetal death (IUFD), fetal growth restriction (FGR), preterm labour and birth. These infections include UTI, human immunodeficiency virus (HIV) infection, periodontal disease, chlamydia, malaria, bacterial vaginosis and group B streptococcal infections. In their recent systematic review and meta-analysis Conde-Agudelo et al (9) showed that there is an association between Preeclampsia and UTI, and between Preeclampsia and periodontal disease in pregnancy.[5,6]

Others have demonstrated the role of infections in increasing sFlt-1 levels in nulliparous women and in inhibition of extra-villous trophoblastic invasion thus causing disruption in normal placentation and placental function.[7]

However, it is difficult to prove a direct causal relationship between preeclampsia and maternal infections as Karmon and Sheiner (8) have pointed out. Von Dadelszen et al (9) suggested that infection could damage the arterial wall through direct endothelial injury, local inflammation or acute atherosis.

Urinary Tract Infection (UTI) is one of the most common infections during pregnancy. UTI stands the second most common complication after anemia during pregnancy. Urinary tract Infection includes infections of both the lower urinary tract (urethra and bladder) and upper urinary tract (kidney). In women, the physiological and anatomical changes that occur in the urinary tract during pregnancy, as well as immune system changes during pregnancy, makes the women more susceptible to developing urinary tract infection and thus increases the incidence of both symptomatic and Asymptomatic bacteriuria. Making UTI one of the most common infections (including ASB) during pregnancy. [10]

Keeping in mind the inflammatory changes involved in the development of preeclampsia, UTI can cause an activation of the systemic inflammatory response and if the burden exceeds the maternal capacity, Preeclampsia becomes more likely.[11]

OBJECTIVES

Objective:

To find an association between urinary tract infection and development of preeclampsia.

REVIEW OF LITERATURE

In historic times, pre-eclampsia and eclampsia were not classified as pregnancy disorders. The earliest descriptions of pre-eclampsia were published in 1637 by Francois Mauriceau, who was considered as a pioneer in obstetrics. He attributed the development of eclamptic seizures to either abnormal blood flow in lochia or due to intrauterine fetal death.[12]

In the early 18th century, for the first time Bossier de Sauvages differentiated between eclamptic seizures and epileptic seizures and gave his opinion on the cause of these convulsions, he theorized that the eclamptic seizures are acute in nature and would resolve once the precipitating event was removed.[12]

In 1821, Dr Thomas Denman drew the conclusion that as the uterus grew in size as the pregnancy advanced, it increased the pressure upon the descending blood vessels and this caused regurgitation of blood in the brain, overloading the cerebral vessels leading to cerebral oedema and subsequent convulsions. In 1843, Dr Robert Johns drew a connection between the antecedental occurrence of premonitory symptoms such as headache, transient loss of vision, severe epigastric pain, and the swelling of the hands, arms, neck and face in the later months of pregnancy and further development of puerperal convulsions in these patients. In 1906, Horn first utilised magnesium sulphate to manage pre-eclampsia and eclampsia.[13]

Trying to explore more in the pathogenesis of preeclampsia, in 1960s placental bed biopsies were taken and examined under a microscope and it was found that the trophoblast cells of the placenta failed to adequately invade maternal spiral arteries.[14]

In 1989, Dr Roberts and colleagues described pre-eclampsia to be a multifaceted disorder, characterised by endothelial dysfunction, excessive activation of coagulation cascade, along with blood pressure abnormalities and loss of fluid from intravascular spaces.[15]

National High Blood Pressure Education Program Working Group Report on High Blood Pressure in Pregnancy first presented the concept of Hypertensive disorders of pregnancy in 1988.[16]

Blood pressure in healthy pregnancies decreases during the first trimester, reaching its lowest point by mid-pregnancy, and typically returns to pre-pregnancy levels during the third trimester. In India, preeclampsia individually showed a prevalence of 8-10 % and incidence of 4.6%. [4]

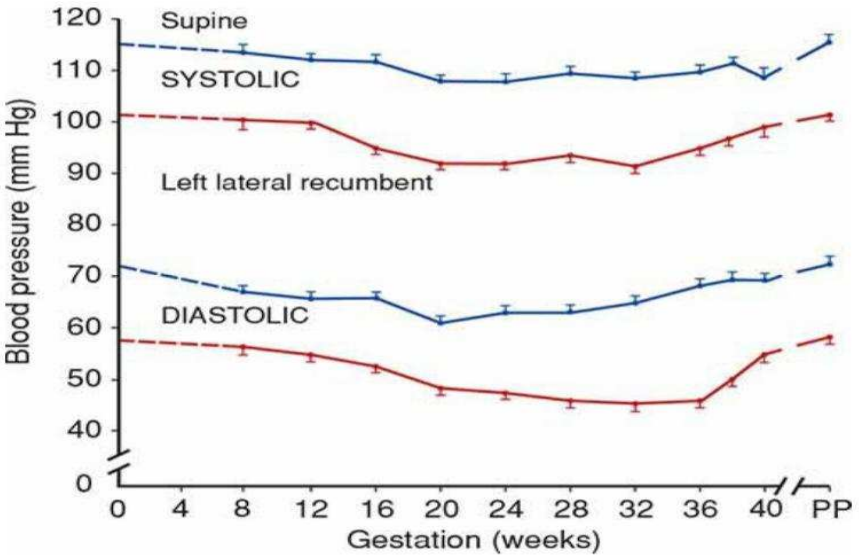


Figure 1: Sequential changes (±SEM) in blood pressure throughout pregnancy in 69 women in supine (*blue lines*) and left lateral recumbent positions (*red lines*). PP postpartum.

(Adapted from Williams Obstetrics, 25th edition)

The major hypertensive disorders that occur in pregnant women are: Gestational Hypertension/ Preeclampsia / Eclampsia / HELLP Syndrome (Hemolysis, Elevated liver enzymes, Low Platelets)/ Chronic Hypertension/ Preeclampsia superimposed on chronic hypertension.

Despite the devastating effects of pre-eclampsia, the precise aetiology of this condition remains largely elusive. In recent years extensive research has been concentrated in this area and as a result a number of hypotheses and theories have emerged. Currently two main theories of the pathogenesis of pre-eclampsia: The two-stage disorder theory and the continuum theory. Both the theories are centred around placenta as the primary source of the condition. This is also supported by the fact that the only definitive treatment of pre-eclampsia is the removal of placenta.[18]

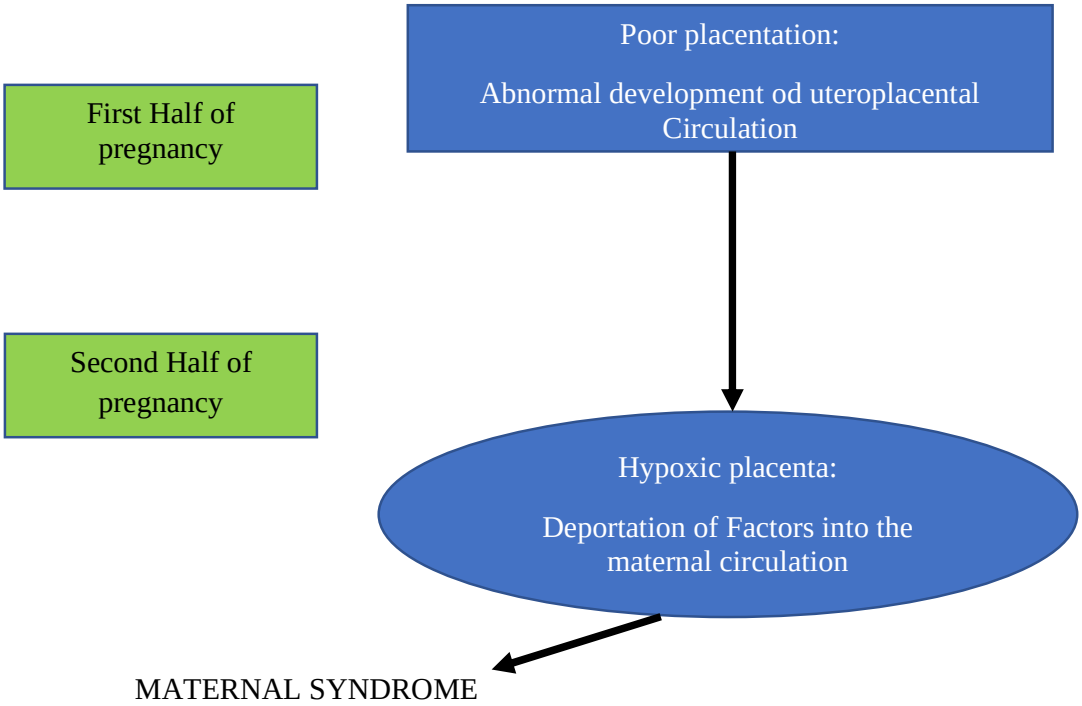
"Two-stage disorder" theory - Redman and co-workers (2015)

In this theory it is postulated that the disease occurs in 2 stages.

1. Stage 1: which is asymptomatic, there is relative reduction in placental perfusion as a result of abnormal placentation and/ or maternal disease at the microvascular level leading to reduced placental perfusion, ischaemia and hypoxia.
2. Stage 2 - modified by pre-existing maternal conditions that are also manifested by endothelial cell activation or inflammation

Deficient placentation in stage 1 is probably due to abnormal immune interaction between paternal antigens on the trophoblast and the maternal decidual NK lymphocytes.[19]

This then triggers release of circulating anti-angiogenic factors from the placental trophoblasts and maternal vascular endothelium causing endothelial dysfunction. These events lead to further exaggeration of the basal SIRS of normal pregnancy. The exaggerated SIRS (systemic inflammatory response syndrome) results in widespread endothelial activation, damage and dysfunction causing profound disturbances in blood flow, vascular function and arterial blood pressure. This is referred to as Stage 2 or the Maternal clinical syndrome, characterized hypertension, proteinuria, eclampsia, HELLP syndrome etc.[20]



- Hypertension
- Proteinuria
- Eclampsia
- HELLP syndrome

One of the two anti-angiogenic factors strongly implicated in the transformation of the disease process from Stage 1 to Stage 2 is the soluble fms-like tyrosine kinase-1 (sFlt-1) which is a receptor for vascular endothelial growth factor (VEGF) and placental growth factor (PlGF). It binds these two growth factors such that even though their total serum levels increase during pregnancy, their free form is drastically reduced in patients with Preeclampsia. This has a negative impact on placental angiogenesis.[21]

The other circulating factor gaining recognition in the linkage between Stage 1 and Stage 2 in the soluble form of endoglin (sEng) which is a co-receptor for transforming growth factor- β (TGF- β) types 1 and 3. This impact negatively on TGF- β angiogenic function and its nitric- oxide (NO) dependent vasodilation effect.[22]

The Continuum Theory

In this theory it is believed that the normal pregnancy is characterised by an inflammatory response and it is therefore postulated that pre-eclampsia is primarily due to an exaggeration of the normal physiological inflammatory response (SIRS) which has progressed to a point of decompensation. It is suggested that SIRS could be due to an infectious insult occurring in pregnancy.[23]

Therefore, in this model Preeclampsia is seen as an extreme end of a continuum of SIRS of normal pregnancy which has resulted in decompensation of different body systems thus creating the clinical maternal syndrome.[23]

Certain medical conditions are accompanied by low-grade systemic inflammation even in non- pregnant women. These conditions include diabetes mellitus, chronic arterial disease, chronic hypertension and obesity. If these conditions exist in a pregnant woman, then the normal SIRS of pregnancy is further elevated and

it is therefore not surprising that such women are at a greater risk of developing preeclampsia.[24]

Development of Placenta and its role in the pathogenesis of preeclampsia

After implantation, extra-villous trophoblast cells (EVT) migrate into the lumens and the smooth muscle walls of the spiral arteries (terminal branches of the uterine arteries supplying the placenta) and convert them into flaccid conduits, with disappearance of the smooth muscle wall resulting in very low resistance to blood flow.[25]

Different degrees of trophoblast invasion occur with ongoing pregnancy, but remodelling of the spiral arteries could be deficient. This causes impaired placental perfusion and the associated oxidative stress in the placenta contributes to the development of pre- eclampsia. Optimum placental perfusion is dependent on the conversion of the spiral arteries, involving loss of smooth muscle and the elastic lamina from the vessel wall. This conversion is associated with a 5-10-fold dilation of the vessel.[14]

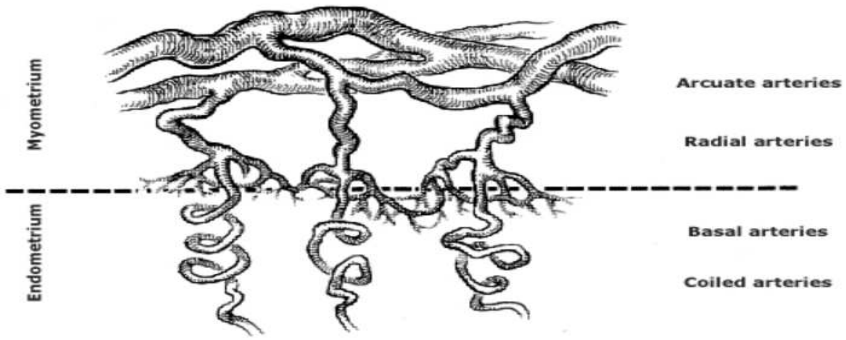


Figure 2. Parts of myometrial arcuate arteries from which myometrial radial arteries course toward the endometrium. There are found larger endometrial coiled arteries and smaller endometrial basal arteries.

The spiral arteries present in the placental bed (around 100–150 in total) are 200–300 μm diameter. In normal pregnancies, the cytotrophoblast cells of the developing placenta migrate through the decidua and myometrium to invade both the endothelium and highly muscular tunica media of the maternal spiral arteries present within the decidua and myometrium. Remodelling of uteroplacental arteries, is the physiological change which converts these small muscular arterioles to high capacitance vessels of low resistance, facilitating great amount of blood flow to the placenta. Remodelling of spiral arteries begins in the late first trimester and is mostly completed by 18- 20 weeks of gestation, though the exact gestational age is unclear.[27]

In comparison to normal pregnancy, in preeclampsia, cytotrophoblast cells infiltrate only the decidual portion of the spiral arteries but fail to penetrate the myometrial segment. Failure of this, leads to pregnancy complications, like Preeclampsia and FGR (Fetal growth restriction). If invasion of spiral arteries does not take place, blood enters the intervillous space in a turbulent way and damages the villous architecture and the anchoring villi, creating cystic lesions containing trophoblast tissue. [28]

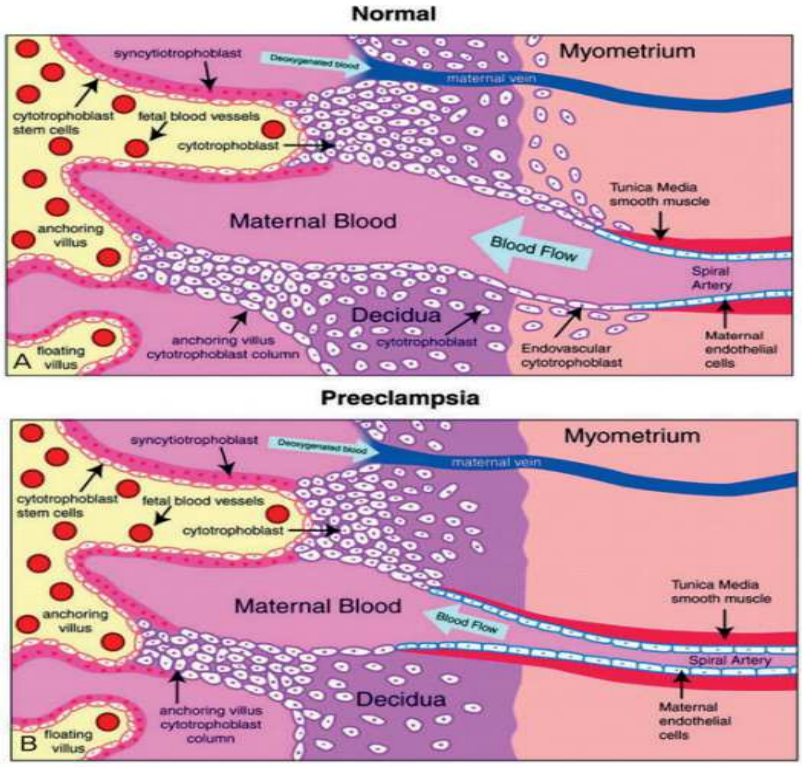


Figure 3**.** Abnormal placentation in preeclampsia. In normal pregnancies, extravillous cytotrophoblasts of fetal origin invade the uterine spiral arteries of the decidua and myometrium. These invasive cytotrophoblasts replace the endothelial layer of the maternal spiral arteries, transforming them from small, high-resistance vessels into large-caliber vessels (A). However, in preeclampsia, this transformation is incomplete. Cytotrophoblast invasion of the spiral arteries is limited to the superficial decidua and does not reach the myometrium (B).

Incomplete remodelling of spiral arteries changes the uteroplacental perfusion from a constant low-pressure flow to a more pulsatile flow at higher pressure. This process injures the chorionic villi, hydrodynamically and biochemically via ischemia-reperfusion.[30]

Another consequence of this changed blood flow due to incomplete remodeling is a local increase in the pro-inflammatory cytokines, chemokines and anti-angiogenic molecules in the placenta. It sets into motion a whole cascade of events starting with release of sFlt-1 and sEng which inhibit angiogenic functions of VEGF, PlGF and TGF- β. There is also associated release of cytokines e.g., TNF- α

and (IL-6) interleukin-6 and trophoblastic debris i.e., Syncytiotrophoblast microparticles (STBM's) and lipid peroxidase, both leading to SIRS and widespread endothelial activation, damage and dysfunction.[31]

This in turn causes systemic vasoconstriction due to raised endothelial production of endothelin, thromboxane and reactive oxygen radicals; reduced endothelial production of nitric acid and prostacyclin (PGI_2) which are natural vasodilators and increased vascular sensitivity to angiotensin II (which is suppressed in normal pregnancy despite high serum levels of angiotensin II). The end result of all the above events is the multi- system, multi-organ disorder i.e., Preeclampsia.[31]

In normal pregnancy during trophoblast differentiation, the invading trophoblasts alter their adhesion molecule expression from those specific to epithelial cells (integrin alpha 6/ beta1, alphav/beta5, and E-cadherin) to those specific to endothelial cells (integrin alpha1/beta1, alphav/beta3, and VE-cadherin), this change is referred to as pseudo-vasculogenesis. Trophoblasts obtained from placenta of women diagnosed with preeclampsia do not show this upregulation of adhesion molecules or pseudo-vasculogenesis.[32\]

Role of maternal infection in the pathogenesis of Preeclampsia:

Over the years studies have shown that there could be some association between various maternal infections and adverse pregnancy outcomes. A literature search retrieved two systematic reviews looking at the role of maternal infections and Preeclampsia. These reviews provided opposing results. Conde et al (33) conducted the review in 2007 to explore the association between maternal infections and preeclampsia. Their review included 49 studies and found a significant association

between urinary tract infections as well as periodontal disease and the development of preeclampsia. In contrast, Rustveld et al (6) reviewed 16 studies to investigate relationship between maternal infections and Preeclampsia. Their findings showed that some studies were significantly associated while others were not as far as viral and bacterial infections and Preeclampsia is concerned.

Maternal infections increased the generalized SIRS seen in pregnancy. It has been demonstrated that widespread endothelial dysfunction is one of the main aspects of a SIRS.[34]

This inflammatory response is detected in normal pregnancy as well, where it is not intrinsically different from that seen in pre-eclampsia except that it is milder. It can be said that pre-eclampsia develops when the systemic inflammatory processes cause one or other maternal systems to decompensate. In other words, the disorder is not a separate entity but simply the extreme end of a range of maternal systemic inflammatory responses engendered by pregnancy itself.[33]

Any factor that would increase the maternal systemic inflammatory responses in pregnancy would predispose the women to pre-eclampsia. There are three possible such factors, based on evidence: a large placenta; an abnormal stimulus from a small placenta; or an excessive maternal sensitivity to such stimuli.[34]

First, with respect to placental bulk, it is a clinically known fact that pre-eclampsia is predominantly a disorder of the third trimester, when the placenta reaches its greatest size.

The placenta grows larger with multiple pregnancies, which also increase the likelihood of developing pre-eclampsia. Pre-eclampsia is also more likely to develop with bigger, rather than smaller, hydatidiform moles. [34]

Second, the most severe form of preeclampsia is that which develops with intense fetal growth retardation, often associated with abnormally small placenta. Here, it can be said that there is an alteration in the quality of the inflammatory stimulus generated by the placenta that is suffering from oxidative stress.[34]

Third, the idea that women may have increased sensitivity to the inflammatory stimulus of a normal pregnancy has already been developed by Ness and Roberts (1996) who proposed that the maternal and placental susceptibilities 'converge at the point of the endothelium.[35]

The inflammatory theory extends this theory by recognizing that endothelial activation is intrinsic to inflammatory responses, which also involve activation of leukocytes.[36]

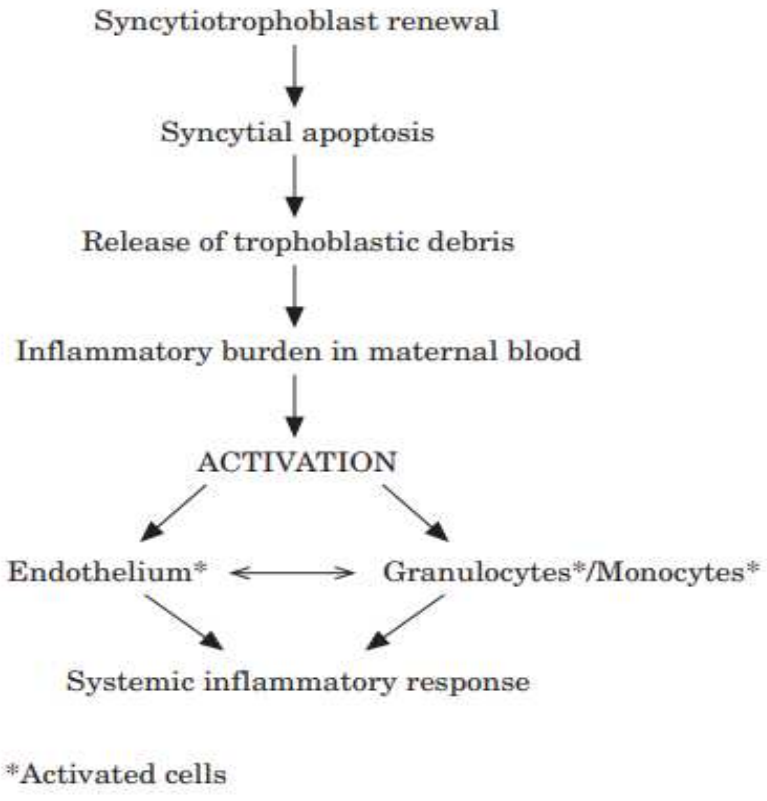


Figure 4. Inflammatory activation leading to SIRS.

In pregnant women, UTI is one of the most common maternal infections, which can potentially cause activation of the maternal systemic inflammatory response and cause endothelial injury; this in turn can lead to development of placental hypoxia and eventually lead to the development of Preeclampsia.[38]

Urinary tract infections have been described since ancient times. The first documented description of urinary tract infection is in the "Ebers Papyrus" dated to 1550 BC. Urinary tract infection was described by the Egyptians as "sending forth heat from the bladder".[39]

In a study, the prevalence of urinary tract infection in pregnancy was found to range from 3.3% to 56% with sample sizes of 200 to 1537 pregnant women.[40]

Escherichia coli is the most commonly isolated pathogen in UTI in pregnancy which is shown in various studies.[41-44] The other organisms include Klebsiella, Staphylococcus aureus, Citrobacter and Pseudomonas aeruginosa this, the prevalence of asymptomatic bacteriuria ranged from 2 % to 25%.[45-48]

There are several studies which describes the association of UTI with pregnancy complications. Untreated urinary tract infection is associated with preterm delivery, low birth weight infants, stillbirths and neonatal death.[49]

We believe that infection may be important in the pathogenesis of preeclampsia, both in terms of its initiation (by increasing the risk of acute uteroplacental atherosis) and/or its potentiation (by amplifying the maternal systemic inflammatory response).[50]

Inflammatory cytokines are said to play an important role in the pathogenesis of preeclampsia. Supporting this, it has been found that cytokines cause elevation of

tumor necrosis factor alpha (TNF-alpha) and interleukin-6 (IL-6) plasma levels in preeclamptic women. The blood pressure regulatory mechanisms (eg, renin-angiotensin system [RAS] and sympathetic nervous system) interact with these pro-inflammatory cytokines, which in turn affect the angiogenic and endothelium-derived factors regulating endothelial function causing increase in blood pressure. [51-52]

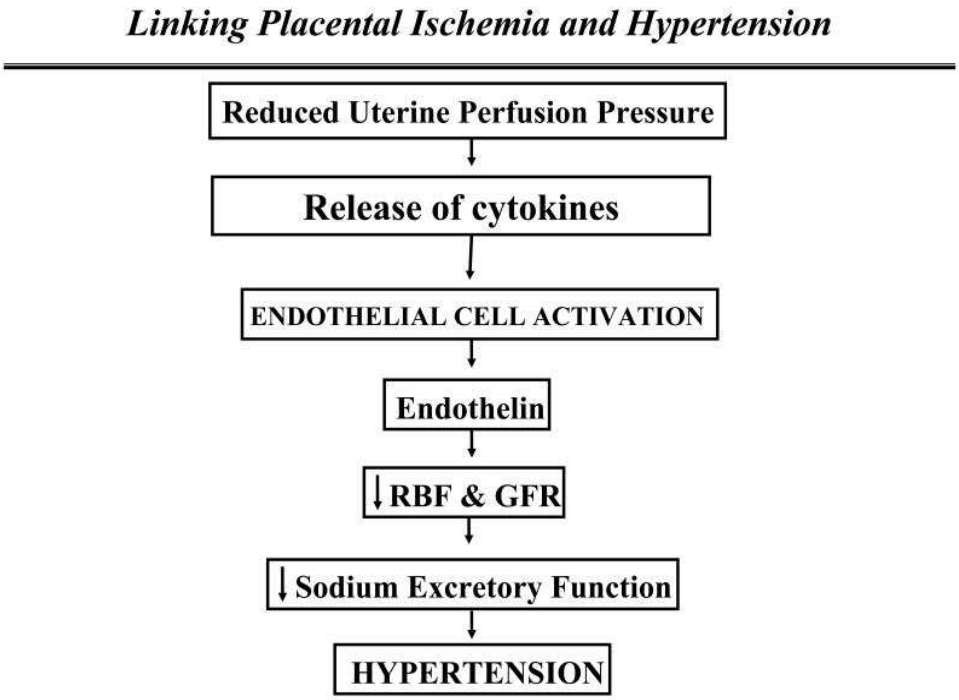


Figure 5. Placental Ischaemia and Hypertension in Pregnancy.

OTHER STUDIES:

1. In a study by Joseph Hill et al. 300 pregnant women at term were enrolled in a case control study. In this study, 100 primigravida diagnosed with preeclampsia were considered as case group and total 200 women as controls which consisted 100 primigravida who delivered by caesarean section for either abnormal lie, cephalopelvic disproportion or fetal distress and 100 multigravida who were not in labour and underwent repeat elective caesarean section. At term, 19% of preeclamptic patients had significant bacteriuria whereas bacteriuria occurred in 3% of the primigravida and 6% of the multigravida control subjects. There was a significant difference in the frequency of bacteriuria between preeclamptic and control patients with a $P<0.005$.[53]

2. In a systemic review and metanalysis by Conde- Agudelo et al. conducted on observational studies that examined the relationship between maternal infections and preeclampsia. An initial search (from inception to June 2007 was performed in MEDLINE, EMBASE, CINAHL and LILACS to identify potentially eligible studies, 88 studies were considered relevant. Forty-nine studies met the inclusion criteria. 40 studies including a total of 182,308 women provided data for the metanalyses. Out of these, 17 studies involving a total of 7317 women provided data on the relationship between preeclampsia and urinary tract infection. Twelve studies reported an association between urinary tract infection and increased risk of preeclampsia. Five studies found no association. Overall, women with urinary tract infection during pregnancy were 57% (95% CI, 45-70) more likely to develop preeclampsia than women without urinary tract infection. [34]

3. In 2013 Minassian conducted, a nested case-control study using the data from the UK General Practise Research Database was evaluated to examine the association between maternal infections and preeclampsia. Primiparous women aged at least 13 years and registered with a practising practise between January 1987 to October 2007 were eligible for inclusion. Cases were patients with preeclampsia (n= 1533) were individually matched up-to 20 controls (n= 14236) who primiparous women without preeclampsia. After adjusting for maternal age, pre-gestational hypertension, diabetes, renal disease and multifetal gestation, the odds of preeclampsia were increased in patients with Urinary tract infection with adjusted odds ratio of 1.22; 1.03-1.45.[1]

4. In 2016, Sarah et al conducted a study including women aged ≥18 years presenting for prenatal care < 15 weeks were recruited and followed up prospectively from the initiation of prenatal care through delivery at 3 tertiary care academic centres: Brigham and Women's Hospital and Beth Israel Deaconess Medical Centre in Boston, MA and the Hospital of the University of Pennsylvania in Philadelphia, PA from October 2007 to June 2009. A total of 2607 gestations with delivery ≥ 24 weeks gestation were enrolled. 129 (4.9%) with diagnosed UTIs and 235 with Preeclampsia (8.8%). Women with UTI had higher rates of preeclampsia compared to those without UTI (31% vs 7.8%, p< 0.0001). [54]

5. In 2017 Joshua Kaduman et al conducted a 1:2 matched case-control study (by age and gravidity) was conducted at the Bugando Medical Centre and Sekou Toure Regional Referral Hospital, Mwanza City, Tanzania. In this study 393 women were enrolled out of which 131 women with preeclampsia were considered as cases involving and 262 without preeclampsia as controls. The proportion of pregnant women with significant bacteria was 28% (110/393). In this study it was concluded

that pregnant women with preeclampsia had 7.7 odds of having significant bacteriuria than those without preeclampsia with a p value <0.001. The predominant bacteria species isolated from these 110 pregnant women were: Escherichia Coli, Klebsiella spp, and other gram negative Enterobacteriaceae. [55]

6. In a metanalyses conducted by Lin Yan et al, observational studies upto October 2017 were extracted from Medline, PubMed, Cochrane Library and Embase databases. Nineteen studies qualified the inclusion criteria. Overall, the pooled Odds ratio was 1.31 (95%; 1.22-1.40) which indicates UTI in pregnancy as a risk factor for development of PE. [11]

7. A case-control study was conducted by Taghavi et al. in 2019 at Bahar hospital in Shahroud. All eligible patients were divided into two equal groups, 92 pregnant women with a diagnosis of preeclampsia and 92 pregnant women were selected as controls. History of UTI in first trimester was assessed as a risk factor. It was found that 37 (40.2%) patients in the case group and 29 (31.5%) patients in the control group had UTI which was significantly higher in the case group with $p < 0.048$. It was concluded that UTI increases the risk of preeclampsia with OR= 1.86.[5]

8. In 2021 Dr. Fawzy et al conducted a study was conducted which included 80 pregnant women who presented to El shatby University Hospital antenatal care unit. Out of which 40 women who were diagnosed with preeclampsia were included in the case group and 40 healthy pregnant women were taken as controls. This study found no significant association between bacteriuria and development of preeclampsia.[56]

METHODOLOGY

The study was conducted in the department of Obstetrics and Gynaecology, KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi from March 2021 to February 2022.

Study design

Cross sectional study.

Study duration and period

One year, March 2021 to February 2022.

Place

KLE'S Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi, a teaching hospital affiliated to Jawaharlal Nehru Medical College, Belagavi.

Source of data

Pregnant women between gestational age of 11-20 weeks attending antenatal OPD or admitted at KAHER's Dr. Prabhakar Kore Charitable Hospital.

Sample size

The minimum sample size formula based on prevalence rate is, n = 136.

$$n = \frac{z_{\alpha}^{2} P(1-P)}{d^2}$$

where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

$z\alpha$ is linked with the level of significance. For 5% level of the significance $z\alpha = 1.96$.

With P = 31.1% and d = 25% of P = 7.78%, the sample size is 136. (3)

Ethical clearance

Prior to the commencement, study was approved by the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belagavi.

Selection Criteria

Inclusion criteria

- Pregnant women with a singleton pregnancy
- Gestation age between 11-20 weeks of gestation

Exclusion criteria

- Unreliable dates
- Known case of renal disorders
- Known case of liver disorders
- Smoking, Alcoholism
- Known case of chronic hypertension
- Antibiotic therapy usage in the last 72 hours
- Non consenting women.

Informed Consent

All the participants fulfilling the selection criteria were explained about the purpose of the study and a written informed consent in their own vernacular language was obtained before enrolment.

Method of collection of data:

In antenatal clinic

All the pregnant women between 11-20 weeks of gestation who visit at the teaching hospital attached to KAHER's J N Medical College, Belagavi be were screened. The women fulfilling the selection criteria were enrolled in the study after obtaining their informed consent. The baseline information and the obstetric details were collected.

Patients were advised to give midstream, clean catch urine samples on the day of enrollment using wide mouthed, sterile containers provided by the hospital. Patients were guided on how to collect the urine sample.

Specimen Transportation

The collected urine samples (Label with the patient's name, OPD number, time and date of collection) were transported at room temperature in a box to the culture lab in the Department of Microbiology within four hours of voiding the sample.

In Microbiology lab

The samples were then subjected to urine routine microscopy and culture. Microscopic test included examining the centrifuged urine deposit at high magnification for pus cells, red cells, urine protein, epithelial cells, casts and crystals. Pus cells $\geq$ 5/ high power field was considered significant for infection.

The samples were then cultured and after overnight incubation on blood agar and Mac Conkey's agar at 37^0 C for 12-14 hours, colony counts yielding bacterial growth of $\geq 10^5$/ml were taken as significant in both symptomatic and asymptomatic pregnant women and were sent for gram staining. Novobiocin sensitivity was done for Gram positive cocci to identify Staphylococcus saprophyticus. Gram negative bacilli were processed for IMViC reaction.

Here, urinary tract infection was defined as the presence of at least 10^5 organisms per millilitre of urine in an asymptomatic patient or symptomatic patients.

Women with both positive and negative culture reports were followed up till delivery to look for following outcomes: **Preeclampsia, Eclampsia, Gestational Hypertension, HELLP syndrome.**

Functional definitions used for the purpose of this study:

1. Chronic hypertension was defined as a blood pressure measurement of 140/90 mm Hg or more on two occasions before 20 weeks of gestation or persisting 12 weeks post-partum.
2. Gestational Hypertension: New onset of hypertension with systolic blood pressure ≥140 mmHg and/ or diastolic blood pressure ≥90 mmHg on at least 2 occasions 4 hours apart after 20 weeks of gestation in a previously normotensive woman.
3. **Preeclampsia:**
    - Blood Pressure: Greater than or equal to 140 mm Hg systolic or greater than or equal to 90 mm Hgdiastolic on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previously normal blood pressure.

Greater than or equal to 160 mm Hg systolic or greater than or equal to 110 mm Hgdiastolic, hypertension can be confirmed within a short interval (minutes) to facilitate timely antihypertensive therapy

And

- Proteinuria: Greater than or equal to 300 mg per 24-hour urine collection (or this amountextrapolated from a timed collection)

or

Protein/creatinine ratio greater than or equal to 0.3*

Dipstick reading of 1+ (used only if other quantitative methods not available)

Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following:

- Thrombocytopenia: Platelet count less than 100,000/microliter
- Renal Insufficiency: Serum creatinine concentrations greater than 1.1 mg/dL or a doubling of the serumcreatinine concentration in the absence of other renal disease
- Impaired Liver Function: Elevated blood concentrations of liver transaminases to twice normal concentration
- Pulmonary edema
- Cerebral or visualsymptoms

* Each measured as mg/dL.

4. **Preeclampsia with Severe features: Any of these findings in a patient with preeclampsia:**
    - Systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg on 2 occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)

- Thrombocytopenia (platelet count <100,000/microL)
- Impaired liver function as indicated by liver transaminase levels at least twice the normal concentration or severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, or both
- Progressive renal insufficiency (serum creatinine concentration >1.1 mg/dL [97 micromol/L] or a doubling of the serum creatinine concentration in the absence of other renal disease)
- Pulmonary edema
- Persistent cerebral or visual symptoms

5. Eclampsia: Eclampsia is defined by new-onset tonic-clonic, focal, or multifocal seizures in the absence of other causative conditions such as epilepsy, cerebral arterial ischemia and infarction, intracranial hemorrhage, or drug use.
6. HELLP syndrome:
    - Hemolysis
    - Elevated Liver Enzymes
    - Low Platelet Count (Platelet <1,00,000/microL)

Statistical Analysis:

- Since the study is of observational study the plan of analysis will be as follows.
- For the continuous quantitative variables mean and standard deviation will be calculated. For the purpose of comparison if the data is divided into two groups with respect to certain qualitative characteristic, the continuous variables will be compared using suitable tools of statistics like student's unpaired t test. The pre and post treatment measures will be compared using student's paired t test
- Discrete variables will be represented by median.
- The categorical data will be expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and demographic characteristics will be tested using Chi-square test, test of proportion or Fisher's exact test.
- For discrete variables nonparametric tests will be used.
- Apart from the above suitable tools like ANOVA, correlation, regression etc., will be used according to the need.
- Suitable graphs will be used to depict the comparison. For all the tests the value of p less than 5% (0.05) will be considered significant.

RESULTS

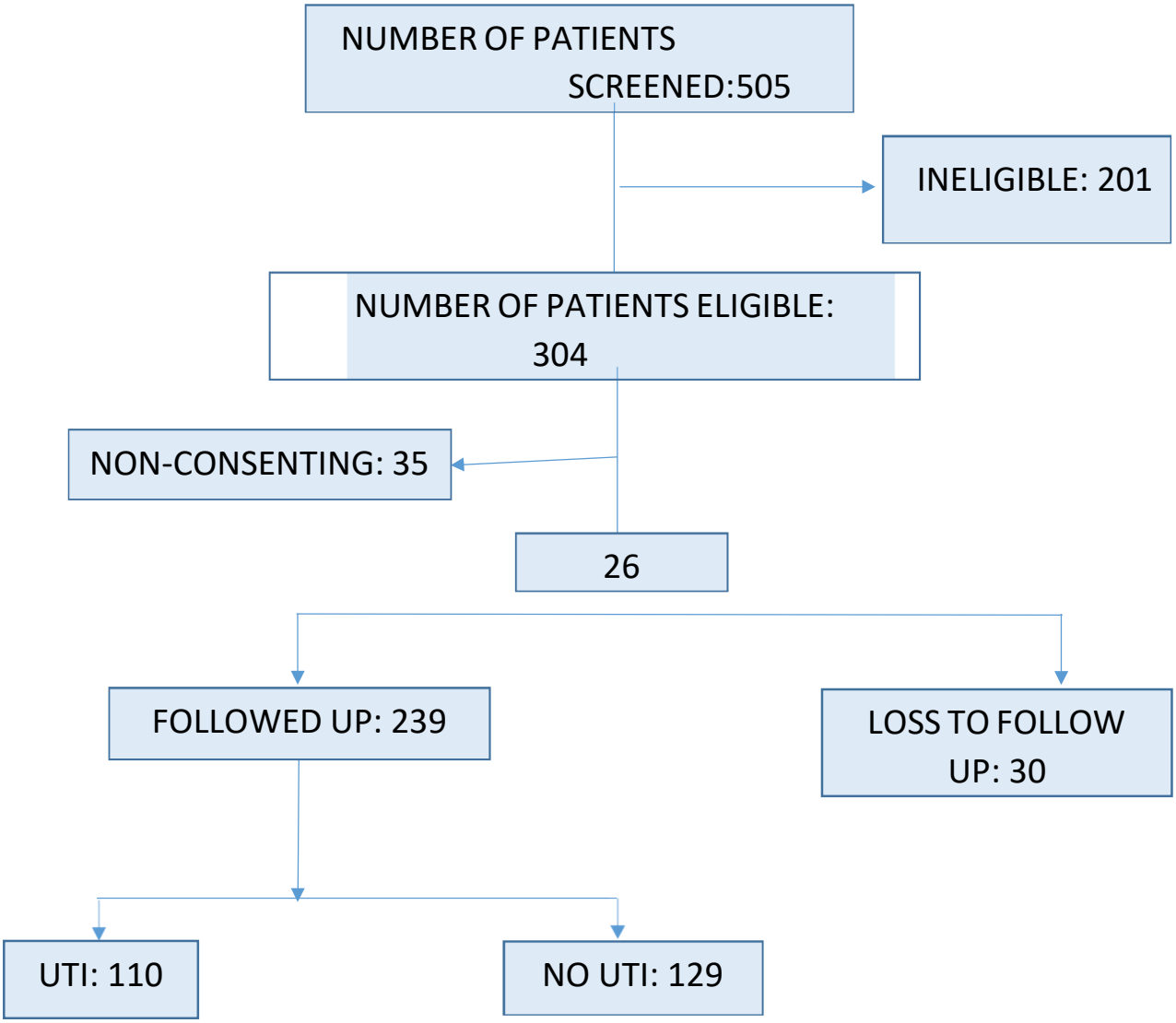
The study was conducted in the Department of Obstetrics and Gynaecology at the teaching hospital attached to KAHER's J N Medical College, Belagavi from 1st March 2021 to 31st February 2022.

A total of 505 antenatal women with singleton pregnancy between 11-20 weeks of gestation were screened. Among whom, 35 women did not consent. Out of the remaining 470 women, 201 women were ineligible including 112 women with contaminated samples. The remaining 239 women were enrolled in the study. Out of the 239 women enrolled, 46.02% (110) women had significant bacteriuria. 53.98% (129) women had no growth in culture.

110 out of the 505 women had significant bacteriuria which constitutes 21.7%. Out of 110, 60% (66) women were symptomatic and 40% (44) women were asymptomatic.

239 women who were enrolled in the study were followed up till delivery for following outcomes: Gestational Hypertension, Preeclampsia, Eclampsia, HELLP Syndrome.

CONSORT

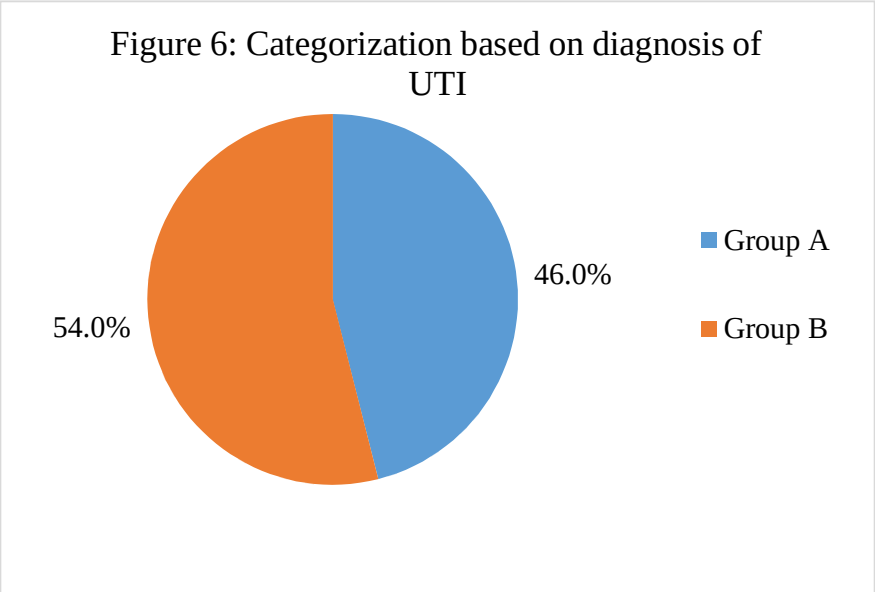

NUMBER OF PATIENTS SCREENED:505
INELIGIBLE: 201
NUMBER OF PATIENTS ELIGIBLE: 304
NON-CONSENTING: 35
26
FOLLOWED UP: 239
LOSS TO FOLLOW UP: 30
UTI: 110
NO UTI: 129

INELIGIBLE Candidates: 201

- Multifetal pregnancy- 24
- Contaminated Sample- 112
- Unreliable Dates- 34
- Usage of Antibiotics in last 72 hours- 14
- Pyelonephritis - 1
- Known Case of Liver disorder- 0
- MTP- 4
- Blighted Ovum- 1
- Known case of chronic hypertension- 4
- H/O PIH in previous pregnancy- 7

Table 1: Categorization based on diagnosis of UTI (N=239)

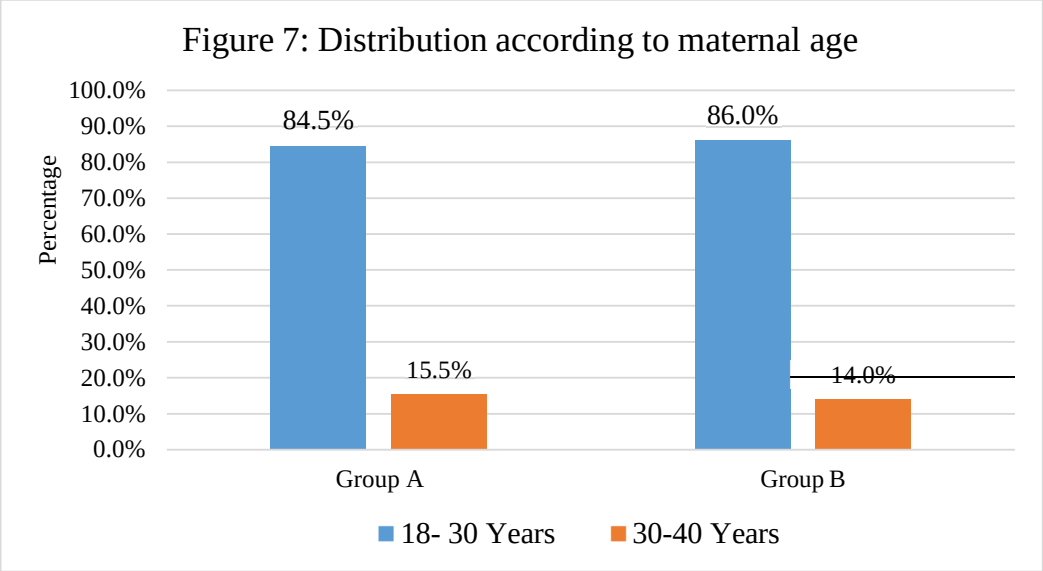
Study Group	n (%)
Cases (with UTI) Group A	110 (46.03%)
Controls (without UTI) Group B	129 (53.97%)



In present study, 110 (46.03%) participants were diagnosed with UTI based on positive urine culture findings and categorized as cases whereas, 129 (53.97%) participants who did not have any growth on urine culture were categorized as controls.

Table 2: Distribution according to maternal age.

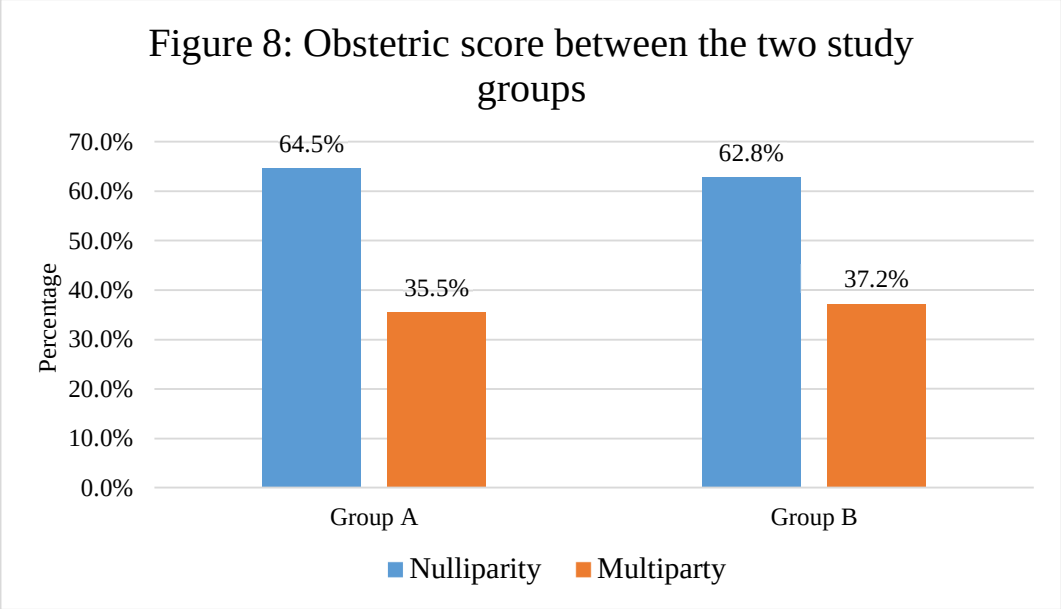
Parameters		Study Group		Chi square	P value
		Cases (N=110)	Controls (N=129)		
Age Group	18-30 Years	93 (84.55%)	111 (86.05%)	0.107	0.744
	30-40 Years	17 (15.45%)	18 (13.95%)		



Most of the participants in both the groups belonged to 18-30 years of age and there was no statistically significant difference in the maternal age between the two study groups.

Table 3: Obstetric score in the study population.

Parameters		Study Group		Chi square	P value
		Cases (N=110)	Controls (N=129)		
Obstetrics Score	Nulliparity	71 (64.55%)	81 (62.79%)	0.079	0.779
	Multiparty	39 (35.45%)	48 (37.21%)		

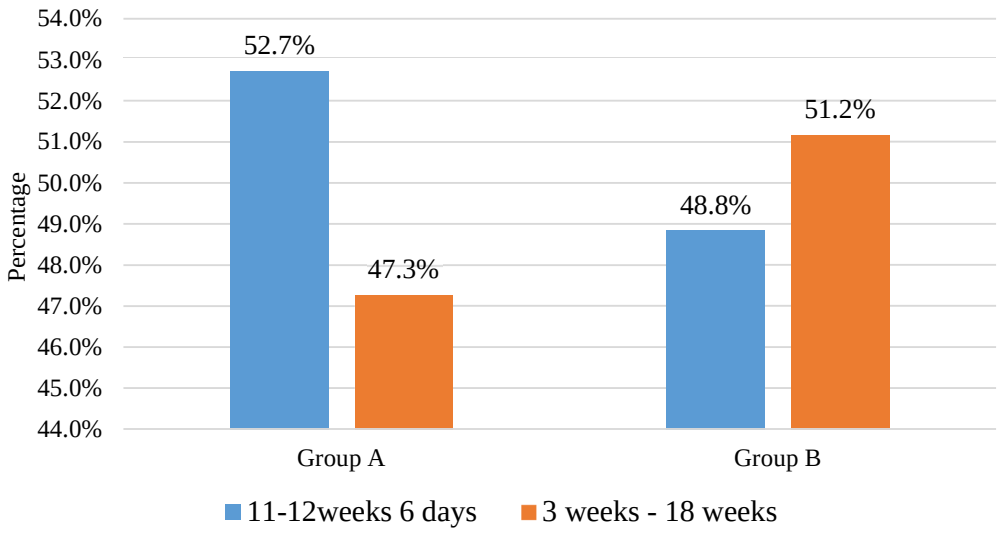


Most of the participants included in the study were nulliparous but the parity was matched between the two groups.

Table 4: Gestational age at enrolment of the two groups.

Gestational age at diagnosis	Study Group		Chi square	P value
	Cases (n=110)	Controls (n=129)		
11-12 Weeks 6 Days	58 (52.73%)	63 (48.84%)	0.359	0.549
13 Weeks - 18 Weeks	52 (47.27%)	66 (51.16%)		

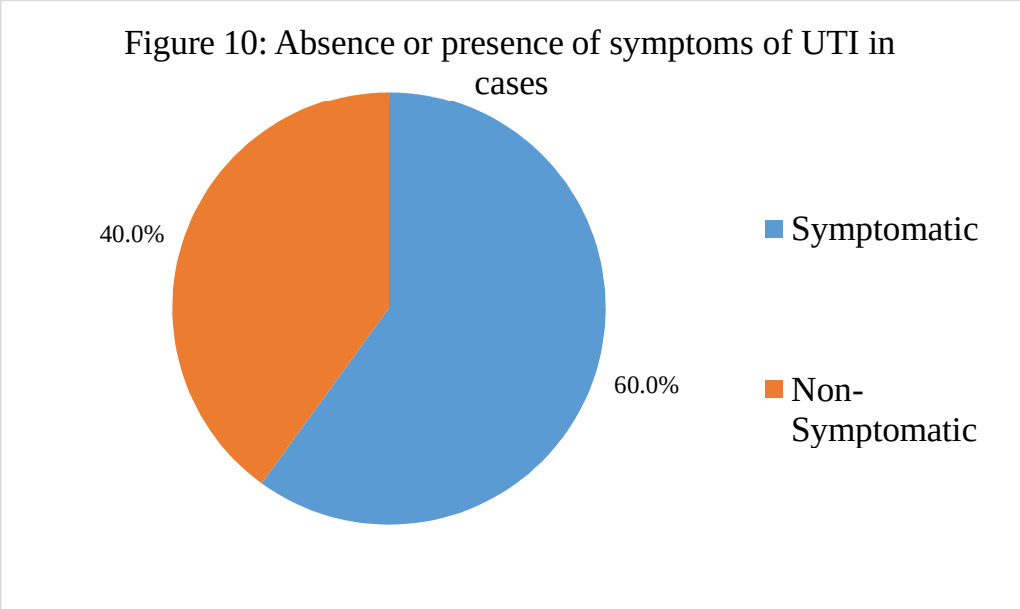
Figure 9: Gestational age at enrolment of all participants



All participants were enrolled between 11-18 weeks of gestation.

Table 5: Symptoms of UTI in Cases (n=110)

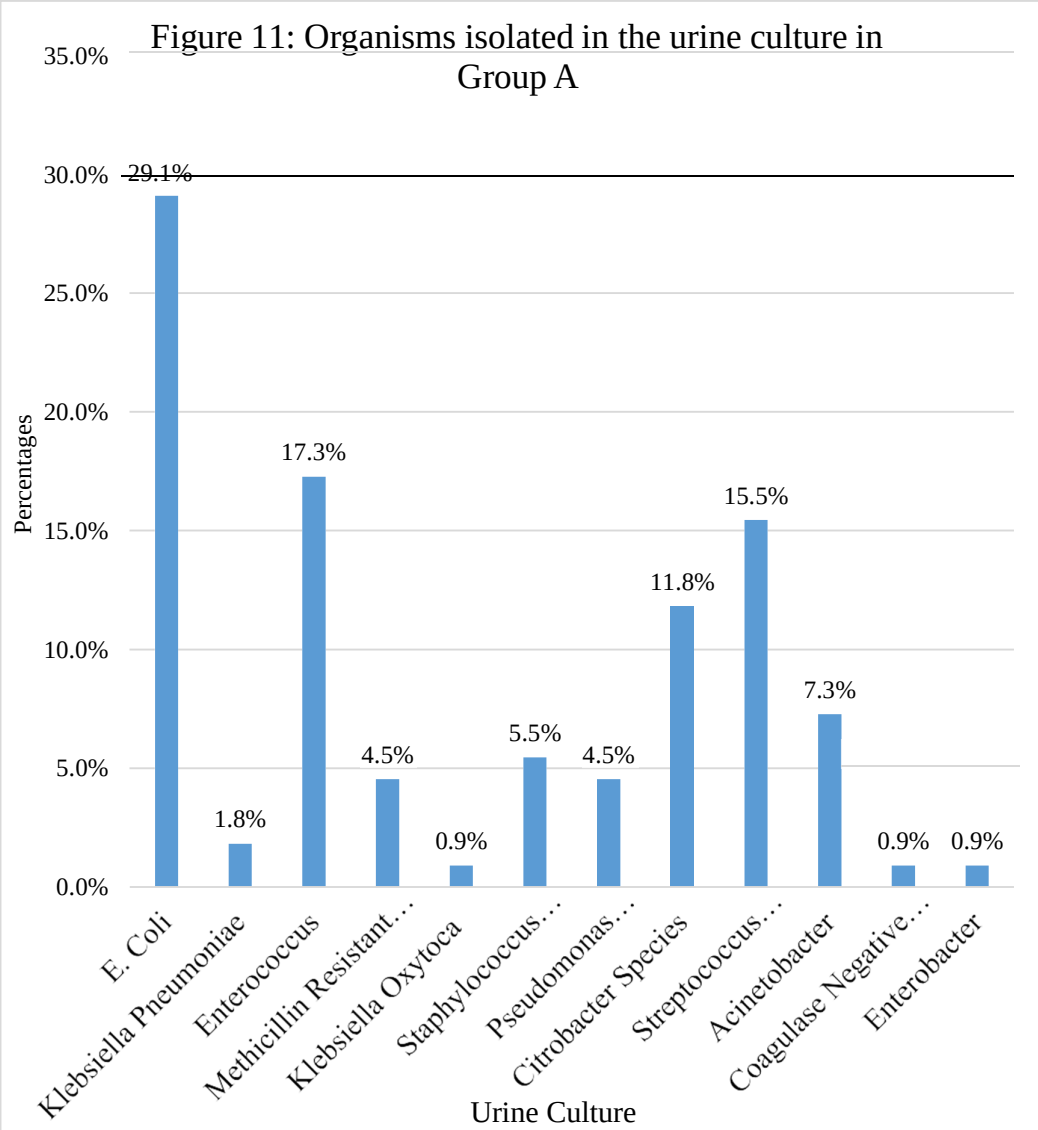
Symptomatic	n	%
Symptomatic	66	60.00%
Non-Symptomatic	44	40.00%



In our study, 60% (66) of the participants with UTI were symptomatic and 40% (44) participants had asymptomatic bacteriuria.

Table 6: Organisms isolated in urine culture in the cases (n=110)

Urine Culture	n	%
E. Coli	32	29.09%
Klebsiella Pneumoniae	19	17.27%
Enterococcus	17	15.45%
Methicillin Resistant Staphylococcus Aureus	13	11.82%
Klebsiella Oxytoca	8	7.27%
Staphylococcus Aureus	6	5.45%
Pseudomonas Aeruginosa	5	4.55%
Citrobacter Species	5	4.55%
Streptococcus Agalactiae	2	1.82%
Acinetobacter	1	0.91%
Coagulase Negative Staphylococcus Aureus	1	0.91%
Enterobacter	1	0.91%

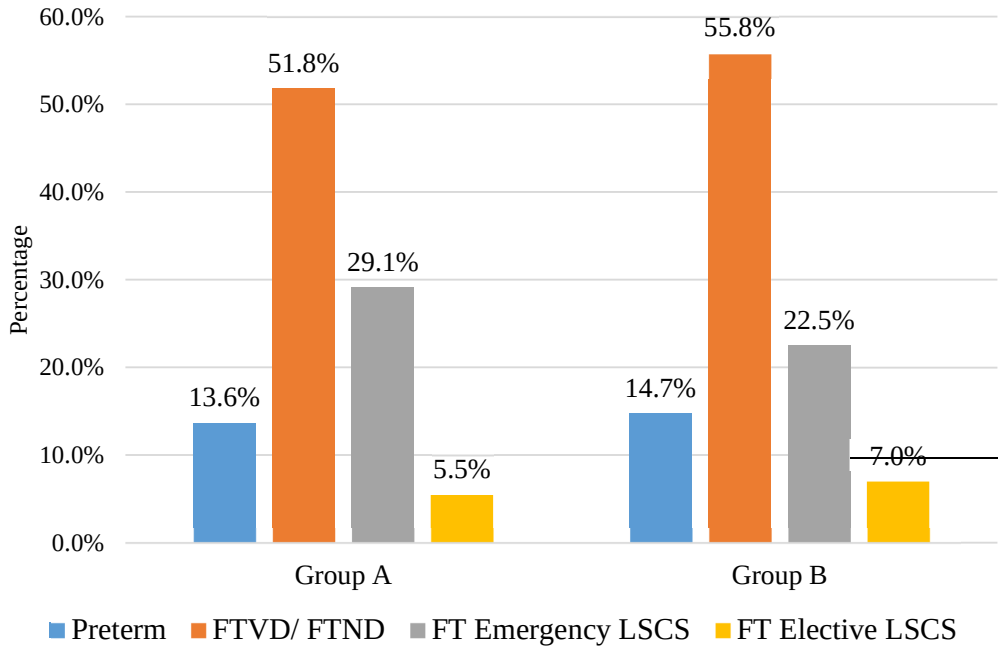


In the present study, E. coli was the most common organism affecting 29.09% (32) of the participants. Other organisms which were isolated included Klebsiella Pneumoniae, Enterococcus, Methicillin Resistant Staphylococcus Aureus.

Table 7: Mode of delivery (N=239)

Parameters		Study Group		Chi square	P value
		Cases (n=110)	Controls (n=129)		
Mode of delivery	Preterm	15 (13.64%)	19 (14.73%)	1.461	0.691
	FTVD/ FTND	57 (51.82%)	72 (55.81%)		
	FT Emergency LSCS	32 (29.09%)	29 (22.48%)		
	FT Elective LSCS	6 (5.45%)	9 (6.98%)		

Figure 12: Mode of Delivery



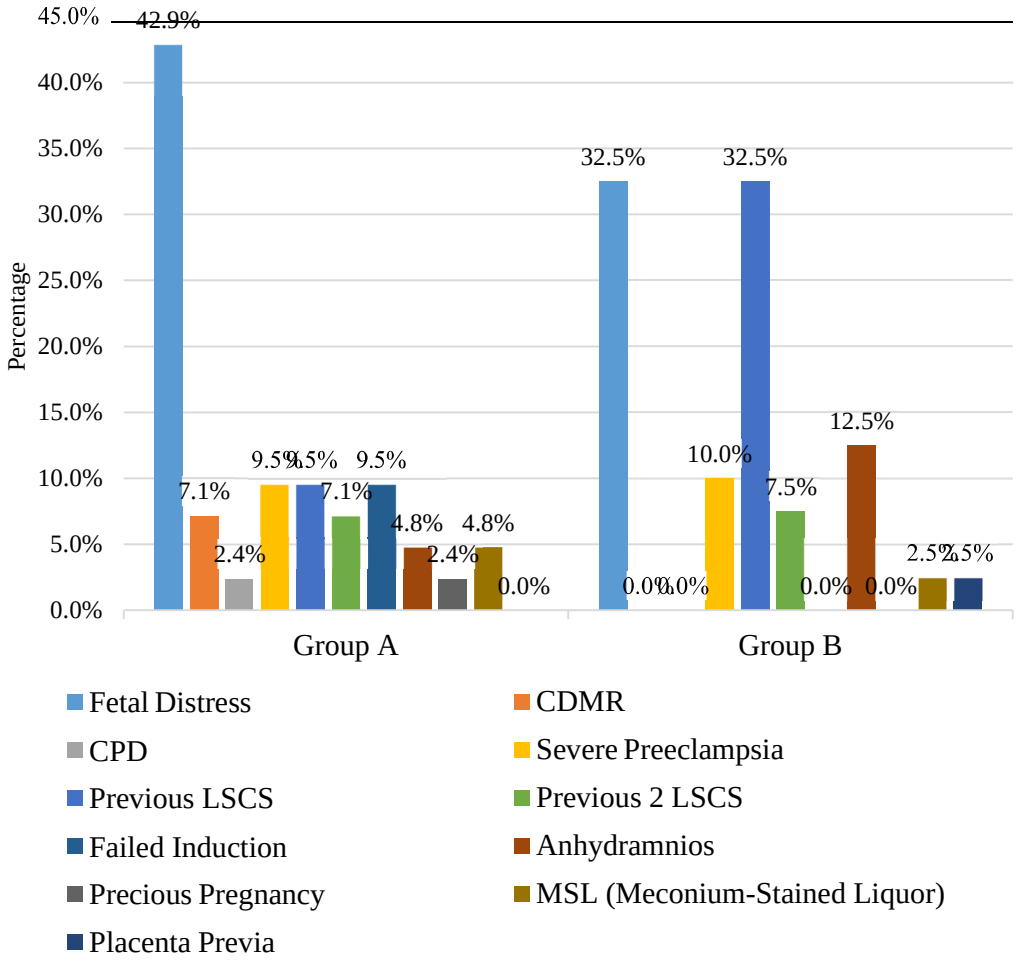
In present study, 15 participants from the cases and 19 participants from the controlshad a preterm delivery. Whereas, the rest of all study participants had a full-term delivery either vaginally or by caesarean section.

There was no statistically significant difference between the caesarean section rate between the UTI cases and controls.

Table 8: Indications of caesarean section in the two study groups. (n=82)

Indications of Section	Study Group		Chi square	P value
	Cases (n=42)	Controls (n=40)		
Fetal Distress	18 (42.86%)	13 (32.5%)	17.15	0.071
Caesarean delivery at maternal request (CDMR)	3 (7.14%)	0 (0%)		
Cephalopelvic disproportion (CPD)	1 (2.38%)	0 (0%)		
Preeclampsia	4 (9.52%)	4 (10%)		
Previous LSCS	4 (9.52%)	13 (32.5%)		
Previous 2 LSCS	3 (7.14%)	3 (7.5%)		
Failed Induction	4 (9.52%)	0 (0%)		
Anhydramnios	2 (4.76%)	5 (12.5%)		
Precious Pregnancy	1 (2.38%)	0 (0%)		
Thick Meconium-Stained Liquor (MSL)	2 (4.76%)	1 (2.5%)		
Placenta Previa	0 (0%)	1 (2.5%)		

Figure 13: Indications of caesarean delivery

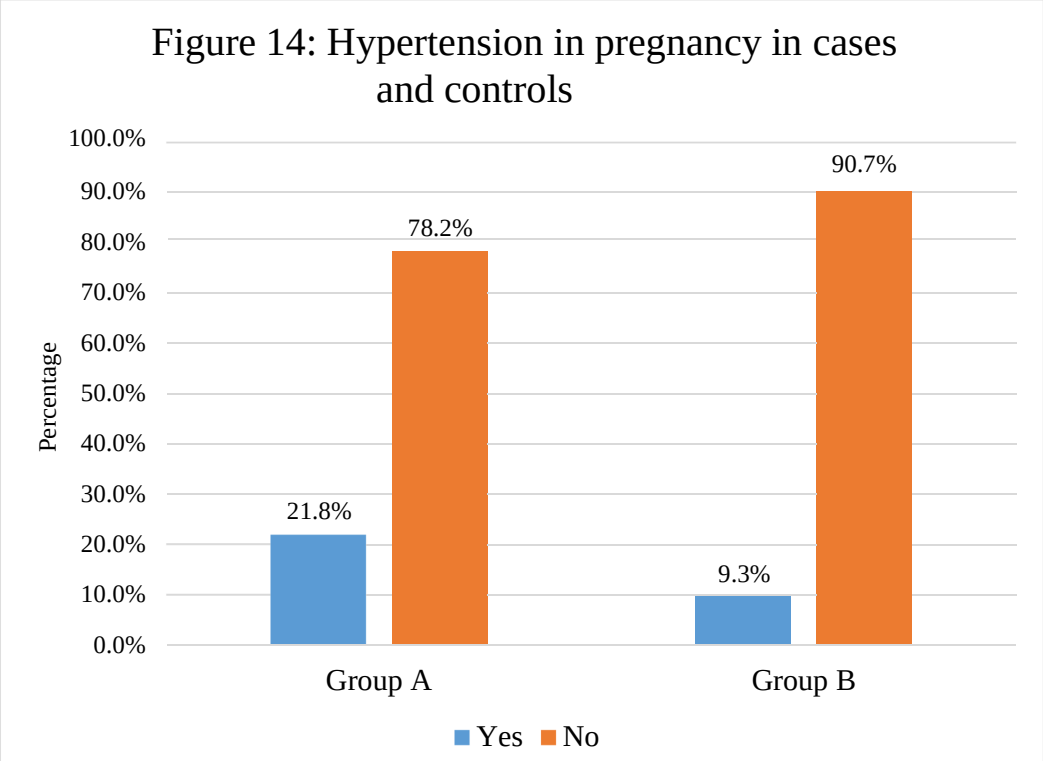


A comparison between the indications of caesarean delivery in the two study groups were comparable. The most common indication of caesarean delivery in both the groups was Fetal Distress i.e., 42.86% and 32.5% respectively.

As shown, both case and control group had 4 caesarean deliveries due to Preeclampsia and there was no statistically significant difference between the two. In the control group, 32.5% caesarean delivery were by repeat LSCS whereas only 9.5% of caesarean delivery were by repeat LSCS in the case group.

Table 9: Development of hypertension in pregnancy in cases and controls (N=239)

Hypertension in pregnancy	Study Group		Chi square	P value
	Cases (n=110)	Controls (n=129)		
Yes	24 (21.82%)	12 (9.3%)	7.269	0.007
No	86 (78.18%)	117 (90.7%)		



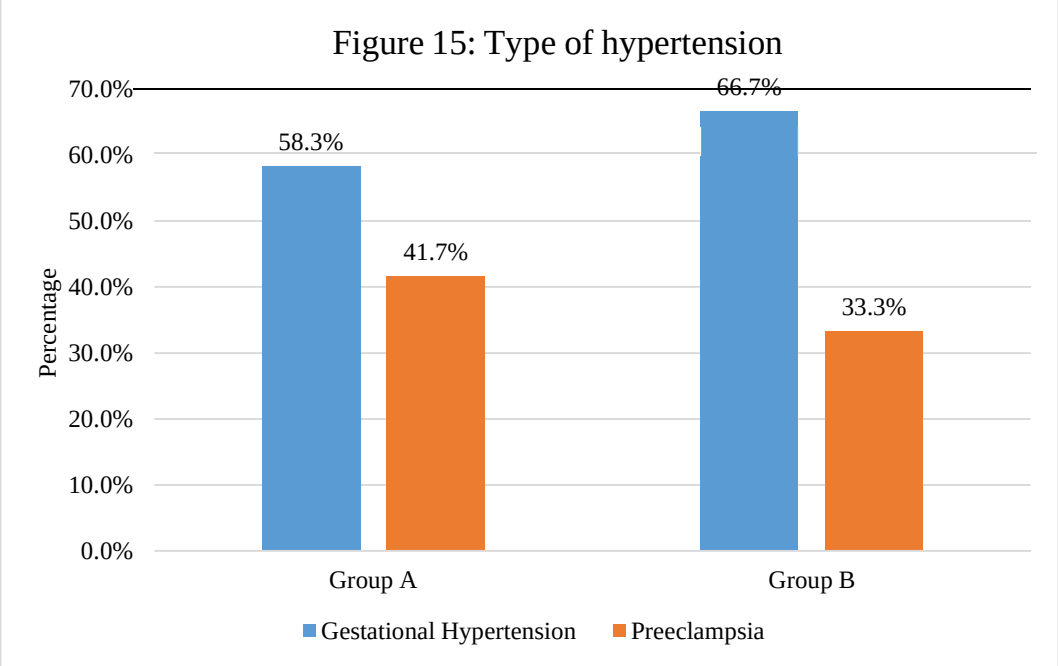
In the present study, total 239 enrolled participants in which 110 were cases who were diagnosed with UTI between 11-20 weeks of gestation and 129 were controls

who were enrolled in the study at comparable gestational age but were not diagnosed with UTI.

In our study, 21.8% of the cases and only 9.3% of the controls developed new onset hypertension in pregnancy. Thus, it can be concluded that the association between UTI and development of new onset of hypertension in pregnancy is statistically significant.

Table 10: Type of hypertension amongst the cases and controls (n=36)

Type Of Hypertension	Study Group		Chi square	P value
	Cases (n=24)	Controls (n=12)		
Gestational Hypertension	14 (58.33%)	8 (66.67%)	0.234	0.727
Preeclampsia	10 (41.67%)	4 (33.33%)		



In the total participants who developed hypertension in pregnancy in the two comparative groups, 58.3% (14) of the cases and 66.6% (8) of the controls developed gestational hypertension respectively.

Similarly, out of the total 14 participants who collectively developed preeclampsia, only 10 cases and 4 controls developed preeclampsia.

The P value for this comparison was 0.727 making it statistically insignificant.

DISCUSSION

PE in pregnancy is a serious cause of maternal and perinatal morbidity. UTI is a common source of inflammatory burden on the mother in pregnancy, as inflammation plays a crucial role in the pathogenesis of PE when the body cannot compensate for this extra inflammatory burden, PE becomes more likely. With appropriate screening and treatment of UTI, this morbidity can be significantly reduced.

This cross-sectional study was carried out in the department of Obstetrics and Gynaecology, KLES Dr. Prabhakar Kore Hospital, Belagavi.

In present study 505 women between 11-20 weeks of gestation were screened for UTI and out of them 110 were diagnosed with UTI with a prevalence rate of 21.7% in our institute. Sarah Rae Easter et al.[4] conducted a study between 2007-2009 wherein the prevalence of UTI was found to be only 4.7%. The huge difference in the prevalence of UTI between the two settings could be due to the difference between the socio-economic conditions of the two places where the studies were conducted.

In 2017, Joshua et al.[5] reported a prevalence of UTI in pregnant women as 28% in Tanzania and the net result of the study conducted by Fawzy et al.[56] in 2021 showed an incidence of asymptomatic bacteriuria of 30% in Egyptian pregnant women. The result of both these studies is comparable to the prevalence rate found in our setting probably owing to the similarity in the socio-economic conditions of these places with India.

In Indian study conducted by Shashi Kant et al.[57] in Ballabgarh, Haryana the prevalence of UTI in women was 33.3% which is comparable to the prevalence found in the present study.

In the present study, 239 participants were enrolled out of which, 110 (46.03%) were cases diagnosed with UTI and 129 (53.97%) women belonged control group without UTI. All 239 participants in the study were followed up till delivery to look for pregnancy outcomes like development of gestational hypertension, preeclampsia, eclampsia, HELLP syndrome.

In the present study, the maternal age of participants was comparable in both the study groups and ranged from 18-30 years of age. In studies by Sarah Rae et al.[4] (18-50y), Joshua et al. [55] (16-42y), Taghavi et al.[5] (18-35y), Fawzy et al.[56] the maternal age of the two groups were similarly matched to ensure no confounding effect due to advanced maternal age being an independent risk factor for developing PE.

With regard to the obstetric score, in the present study there was no difference noted between the development of PE between the nulliparous and multiparous women. In contrast to this, Sarah Rae et al. [4] concluded that there were increased odds of developing PE after diagnosis of UTI during pregnancy in nulliparous women. Similar to the findings of this study, Taghavi et al.[5] and Joshua et al.[55] also did not find any significant association between parity and development of PE in their studies.

Gestational age at the time of diagnosis of UTI in the present study was comparable between both the study groups.

The commonest organism isolated in the present study were- E. coli (29.09%), Klebsiella Pneumoniae (17.27%). Similar to this, in 1986, Joseph A. Hill et al.[53] concluded that the most common organism isolated in their study was E. Coli (50%) and in 2017, Joshua Kaduma et al. [55] also found that E. coli (45.5%) and klebsiella spp (23.6%) were the most common organisms that were isolated from bacteriuric pregnant women. In 2021, Fawzy et al.[56] also reported that E. coli was the most common organism isolated in their study. Another study, by Akerele et al[51] in Benin found that the most prevalent organism isolated was klebsiella which was inconsistent with the findings of the previous studies.

For the first time in 1936, Peters et al.[1] suggested an association between Bacteriuria and pre-eclampsia. Later, Stuart et al. later noted that preeclampsia was 4 times more common in women with bacteriuria than those without bacteriuria. In the present study, out of the 110 participants in UTI group 21.82% (24) participants developed hypertension in pregnancy. Whereas out of the 129 participants in the control group, only 9.3% (12) participants developed hypertension in pregnancy. Suggesting that the incidence of hypertension in pregnancy was more in the UTI group than in the control group with a $P = 0.007^{MC}$ which is statistically significant.

In the present study, (14) 58.33% participants from the UTI group developed gestational hypertension and (10) 41.67% participants developed Preeclampsia which was statistically comparable to the control group in which (10) 66.67% participants developed Gestational hypertension and (4) 33.33% developed Preeclampsia.

Thus, there was no statistically significant association between development of UTI and PE.

In 1986, Joseph A. Hill Et al.[53] quoted the incidence of bacteriuria preeclamptic patients was 19% and 3-6% in the control group which showed a significant difference in frequency of bacteriuria at term between preeclamptic patients and control patients with a P< 0.005. In a large population-based study from 1987- 2007 by Caroline Minassian et al. [1] it was concluded that there was a significant association between development of UTI and preeclampsia with an adjusted odds ratio 1.2; 1.03-1.45. In 2007, Agustin Conde-Agudelo et al.[34] concluded through his metanalyses that women with UTI during pregnancy were 57% more likely to develop PE than women without UTI with a odds ratio of 1.57.

In 2009, Sarah et al. [4] concluded in her study that women with UTI (31%) in pregnancy has higher rates of PE compared to those without UTI (7.8%) with P<0.0001. Babak Izadi et al. conducted a study in Iran and found a significant association between UTI and severe PE with a P value of <0.05.

Between 2019-2020, Taghavi et al. [5] conducted a study on Iranian pregnant women and found a significant association between development of UTI in first trimester and development of PE with an increase in the chance of its occurrence by 1.8 times.

In 2021, Fawzy et al.[56] reported that the incidence of asymptomatic bacteriuria was higher in the preeclampsia group (37%) than in the control group (22%) but it had no statistical significance between the association of bacteriuria and PE, consistent with the finding of our study.

CONCLUSION

The prevalence of urinary tract infection among pregnant women was 21.7% in our study. UTI was significantly associated with development of new onset hypertension in pregnancy, however no significant association was found between UTI and development of Preeclampsia. A multicentre study with a greater sample size could help us throw better light on the nature of this association.

SUMMARY

The present study was a case control study, conducted in the Department of Obstetrics and Gynaecology at the teaching hospital attached to KAHER's J N Medical College, Belagavi over a period of one year. The objective of the study was to find an association between UTI and development of PE.

Patient population included 239 pregnant women with singleton pregnancy between 11-20 weeks of gestation who were segregated into

- Cases (110)- Women who were diagnosed with UTI on Urine culture.
- Controls (129)- Women who did not have UTI.

239 women who were enrolled in the study were followed up till delivery for following outcomes: Gestational Hypertension, Preeclampsia, Eclampsia, HELLP Syndrome. Data regarding demographic characteristics, investigations and outcome had been collected in the form of a structured proforma and analysed statistically.

The key findings of this study have been summarized as follows:

- The age of the participants ranged from 18-30 years with mean age of 26.4 ± 3.4 years. There was no correlation between age and outcomes.
- Majority of women included in the study were nulliparous. There was no significant association between parity and outcomes.
- Most women enrolled in the study who belonged to the UTI group, were symptomatic (66%).
- The most common organism isolated from the urine culture in the present study were, E. coli (29%), Klebsiella pneumoniae (17%), Enterococcus (15%) etc.

- Most patients in both the groups delivered via full term vaginal delivery. Only 13% cases and 14% of controls had a preterm delivery. 29% of cases underwent emergency LSCS in cases group and 22% in the control group.
- The most common indication for caesarean delivery in both the groups was fetal distress i.e., 42% in the cases and 32.5% in controls. There was no significant association between the indications of caesarean delivery between both the groups.
- In the present study, 21% of cases and only 9.3% of the controls developed new onset of hypertension in pregnancy. This association was found to be significant with P value of 0.007.

14 patients in case group and 8 patients in the control group developed gestational hypertension. 10 patients in the case group and 4 in the control group developed preeclampsia. This association between the two groups was not found to be significant.

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ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

"THE ASSOCIATION BETWEEN URINARY TRACT INFECTION AS A RISK FACTOR FOR PREECLAMPSIA: A CROSS-SECTIONAL STUDY"

Principal Investigator:
REG NO. BJ0120005
Post Graduate student
Dept. of Obstetrics & Gynaecology
J.N Medical College, Belagavi.

Co-Investigator:
Dr. _____
Professor
Dept. of Obstetrics Gynaecology
J.N Medical College, Belagavi.

Mrs. _____ we are requesting you to enroll yourself in the study titled: "The association between urinary tract infection as a risk factor for preeclampsia: A cross-sectional study" conducted by Dr. _____, Post Graduate in M.S Obstetrics and Gynaecology under the guidance of Dr_____, Professor at Department of Obstetrics & Gynaecology, J.N. Medical College, KAHER, Belagavi.

The Purpose of this study is to see for the association between urinary tract infection and preeclampsia. I will be the investigator for our study. The study is not being funded. I am going to give you information about this research project. Before you decide, you can talk to anyone you feel comfortable with about the research

PURPOSE OF THE STUDY:

Preeclampsia is one of the most common antenatal complications of pregnancy. Inspite of many previous studies, the pathogenesis of preeclampsia is not known. One of the proposed theories behind the pathogenesis of preeclampsia states that increased

systemic inflammation in a pregnant women could predispose her towards development of preeclampsia.

Urinary tract infection being one of the most common systemic inflammatory disorders of pregnancy could potentially be a risk factor in the development of preeclampsia.

I am doing this study to find an association between urinary tract infection as a risk factor for preeclampsia.

TYPE OF THE STUDY

This is a cross-sectional study. In this study participants will be screened at their first visit for urinary tract infection via Urine routine and microscopy and via urine dipstick tests. Urine culture and sensitivity will be done as a confirmatory test for patients who test positive

Patients with urinary tract infection will be followed up till delivery for the development of preeclampsia.

PARTICIPANT SELECTION

We are inviting all women with singleton pregnancy and who are not a known case of preeclampsia who will be attending the outpatient department of OBG for their routine antenatal checkup at KAHER's Dr Prabhakar Kore Hospital, Belagavi.

VOLUNTARY PARTICIPATION

Your participation in the research is voluntary. It is your choice whether to participate or not. Your decision whether to participate in the study or not will not change present or future health care services offered to you and will not affect your relationship with

Dr. Prabhakar Kore Hospital or J.N Medical College. If you choose not to participate in the study, you will still be offered the treatment necessary for you. If you decide to participate in the study then you are free to withdraw at anytime.

PROCEDURE INVOLVED:

After selection of patients for the study and obtaining informed consent, patients will be evaluated as per history, general physical examination, routine antenatal investigations and ultrasonography. Urine routine microscopy will be done as a part of routine antenatal investigations. Apart from this urine dipstick tests will be done in the outpatient department. Patients who come positive for either of these tests will be advised to send urine samples for culture and sensitivity for confirmation. The patients will then be followed up till delivery for the development of preeclampsia.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No financial incentives are being offered to enrolled patients. It is purely done with the idea of research and all the cost of the study will be borne by the investigator.

PRIVACY & CONFIDENTIALITY:

The only people who will know that you are the research subject will be the members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission except:

1. In Emergency to protect your rights and welfare.
2. If required by law.

AUTHORIZATION TO PUBLISH RESULTS:

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential. Results of the study will be used to improve maternal outcome.

RIGHT TO REFUSE OR WITHDRAW FROM THE STUDY:

You do not have to participate in the study if you do not wish to. You can withdraw at any time from the study. There will be no penalty for withdrawal. Your treatment and care in this hospital will not change irrespective of whether you agree to participate or not. You can be removed from the study if necessary.

INSTITUTIONAL / SPONSOR'S POLICY:

In the event of any injury related to the study, treatment will be made available through KAHER, Belagavi. There is no compensation or payment for such medical treatment by the law. If you are injured you may contact Dr. _____, Post graduate student, Department of Obstetrics and Gynaecology, KAHER or by ph. No- _____.

CONTACT DETAILS:

In case you have any questions related to the study, in future or in case of study related problems you can contact,

REG NO. BJ0120005

Post graduate student,

Department of Obstetrics and Gynaecology,

KLE University's Jawaharlal Nehru Medical College,

Belagavi.

Ph. No: 9049729900.

1. Dr. _____

Dept. of Obstetrics and Gynaecology,

KLE University's Jawaharlal Nehru Medical College,

Belagavi.

Ph No: 9164693333.

If you have any queries about your rights as a study participant, you may contact

Dr. Roopa M Bellad,

Professor, Department of Paediatrics,

Chairman of J.N.Medical College,

Institutional Ethics Committee on Human Subjects Research,

Phone no-0831 2473777 ext-1527 at J.N Medical College, Belagavi.

CONSENT STATEMENT:

I, _____voluntarily agree for participating in this study. By signing this consent form I am not giving any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read to the form in my own vernacular language, including the risks and the benefits and having all my questions answered.

Participant Name: _____

Signature or the Left Thumb Print of Participant: _____

Investigators Name: _____

Signature of Investigator: _____

Witness Name: _____Signature: _____

Date: _____

ANNEXURE II – SCREENING FORM

Screening number: ☐☐☐☐ OP number: ☐☐☐☐☐☐

Date of screening (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

First name: ☐☐☐☐☐☐☐☐☐

Middle name: ☐☐☐☐☐☐☐

Last name: ☐☐☐☐☐☐☐☐☐

Husband's name: ☐☐☐☐☐☐☐☐

Age (years): ☐☐

Address:

_____

Phone number: ☐☐☐☐☐☐☐☐☐☐

Registered	
Unregistered	

Eligibility:

INCLUSION CRITERIA:

Singleton Pregnancy Yes ☐ No ☐

Diagnosed with UTI Yes ☐ No ☐

EXCLUSION CRITERIA:

Known case of chronic hypertension:	Yes	☐	No	☐
Known case of renal disease:	Yes	☐	No	☐
Known case of liver disease:	Yes	☐	No	☐
Known case of preeclampsia:	Yes	☐	No	☐
Known case of Eclampsia:	Yes	☐	No	☐
History of alcoholism:	Yes	☐	No	☐
History of smoking:	Yes	☐	No	☐
No History of antimicrobials therapy in the last 72 hours:	Yes	☐	No	☐
Is she eligible?	**Yes**	☐	**No**	☐

ANNEXURE III - PROFORMA

STUDY TITLE: THE ASSOCIATIONG BETWEEN URINARY TRACT INFECTION AND PREECLAMPSIA.

Screening Id: ☐☐☐☐ Study Id: ☐☐☐☐

Age: ☐☐

Date of enrollment: ☐☐☐☐☐☐

Obstetric history:

Married Life (years): ☐☐

Obstetric score: G ☐ P ☐ L ☐ A ☐ D ☐

Menstrual history:

Menarche:

Last Menstrual Period (LMP):

Expected Date of Delivery (EDD): USG 1st trimester EDD:

Gestational Age:

Past History:

History of Diabetes/ Tuberculosis/ Hypertension/ Asthma/ Epilepsy:

☐ ☐ ☐ ☐ ☐

Personal History:

Diet: Appetite:

Bowel and Bladder: Habits: Smoking/ Alcoholism/ Tobacco:

General physical examination

Pulse rate (beats per minute): bpm

Blood pressure (mmHg): / mm of Hg

Pedal oedema:

INVESTIGATIONS:

DATE		
Urine Routine & Microscopy: Albumin- Nitrite- RBC- WBC- Epithelial Cells- Bacteria-		
Urine Culture: Organism isolated		

Diagnosis of Urinary Tract Infection: Yes ☐ No ☐

Date of Diagnosis: Gestational Age:

Treatment given for UTI

IF yes, details: _____

AT THE TIME OF DELIVERY

Development of Gestational Hypertension Yes ☐ No ☐

Development of preeclampsia Yes ☐ No ☐

Development of eclampsia Yes ☐ No ☐

Date of Delivery:

Gestational age at the time of delivery: WEEKS DAYS

Mode of delivery:

If, LSCS, indication _____

Antihypertensive treatment given- Yes ☐ No ☐

If yes, details:

_____

ANNEXURE IV

KEY TO MASTERCHART

1. AGE

18- 30 YEARS: 1

>30-40: 2

2. PARITY

NULLIGRAVIDA: 1

MULTIGRAVIDA: 2

3. TRIMESTER

T1: 1

T2: 2

4. SYMPTOMATIC/ NON-SYMPTOMATIC

SYMPTOMATIC: 1

NON-SYMPTOMATIC: 2

5. ORGANISM ISOLATTED

E. COLI- 1

SPREPTOCOCCUS AGALACTIAE -2

KLEBSIELLA PNEUMONIAE-3

PSEUDOMONAS AEROGUINOSA- 4

ACINETOBACTER- 5

STAPHYLOCOCCUS AUREUS- 6

CITROBACTER SPECIES- 7

METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS - 8

ENTEROCOCCUS- 9

KLEBSIELLA OXYTOCA - 10

Coagulase Negative Staphylococcus Aureus - 11

ENTEROBACTER: 12

SALMONELLA: 13

6. DELIVERY:

PRETERM: 1

FTVD/ FTND: 2

FT EMERGENCY LSCS: 3

FT ELECTIVE LSCS: 4

7. HYPERTENSION:

YES: 1

NO: 2

8.TYPE OF HYPERTENSION:

GESTATIONAL HYPERTENSION: 1

PREECLAMPSIA: 2

ECLAMPSIA: 3

9. INDICATION FOR SECTION:

FETAL DISTRESS: 1

CDMR (CESAREAN DELIVERY AT MATERNAL REQUEST) : 2

CPD (CEPAHLO PELVIC DISPROPORTION) : 3

SEVERE PREECLAMPSIA : 4

PREVIOUS LSCS: 5

PREVIOUS 2 LSCS: 6

FAILED INDUCTION: 7

ANAMNIOS: 8

PRECIOUS PREGNANCY: 9

MSL (MECONIUM STAINED LIQUOR) : 10

PLACENTA PREVIA : 11

S. NO	OP NO/ IP NO.	AGE	OBSTETRIC SCORE	GESTATIONAL AGEAT DIAGNOSIS	DATE OF TEST	SYMPTOMATIC	URINE CULTURE	DELIVERED/ NOT DELIVERED	MODE OF DELIVER	GESTATIONAL AGE AT DELIVERY	INDICATION OF SECTION	HYPERTENSION	TYPE OF HYPERTENSION
1	1041958	21	G2P1L1	12 WKS 1 DAY	03-03-2021	Symptomatic	Escheriachia Coli	Delivered	PTD	36 weeks 6 days		No	
2	5995468	21	PRIMIGRAVIDA	12 WEEKS 6 DAYS	31-03-2021	Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	37 weeks 2 days	Fetal Distress	Yes	Gestational Hypertension
3	6053084	22	PRIMIGRAVIDA	16 WKS 2 DAY	08-04-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	38 weeks		Yes	Gestational Hypertension
4	1047593	26	G2A1	12 WEEKS 1 DAY	12-04-2021	Symptomatic	Escheriachia Coli	Delivered	FT Elective LSCS	39 weeks	CDMR	No	
5	3826759	26	G2A1	12 WEEKS 1 DAY	12-04-2021	Non Symptomatic	Streptococcus agalactiae	Delivered	PTD	36 weeks		No	
6	6026794	22	PRIMIGRAVIDA	15 WKS 1 DAY	22-04-2021	Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	39 weeks 4 days	CPD	Yes	Gestational Hypertension
7	6052802	23	PRIMIGRAVIDA	12 wks 2 day	24-04-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	38 wks 1 day	Fetal distress	No	
8	6069977	26	G2A1	12 Wks 3 days	24-04-2021	Non Symptomatic	Pseudomonas aeruguinosa	Delivered	FTND	37 wks 2 days		No	
9	6018991	25	G3A2	12 wks 6 days	29-04-2021	Symptomatic	Acinetobacter Species	Delivered	FTVD	38 wks 3 days		No	
10	1051348	30	G2P1D1	12 wks 2 days	03-05-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	39 wks 5 days	Fetal distress	Yes	Preeclampsia
11	6026453	29	G3A2	12 WKS 2 DAYS	06-05-2021	Non Symptomatic	Escheriachia Coli	Delivered	PT Emergency LSCS	35 wks 6 days	Severe PE with uncontrolled HTN	Yes	Severe Preeclampsia
12	1051563	20	PRIMIGRAVIDA	13 WKS 4 DAYS	11-05-2021	Symptomatic	Staphylococcus Aureus	Delivered	FTND	38 WEEKS 3 DAYS		NO	
13	5921087	22	PRIMIGRAVIDA	13 WKS 6 DAYS	13-05-2021	Symptomatic	Citrobacter Species	Delivered	FTND	40 WEEKS		NO	
14	6091822	32	G3P1L1	12 WKS 2 DAYS	24-05-2021	Non Symptomatic	STAPHYLOCOCCUS SPECIES	Delivered	FTVD	40 WEEKS 5 DAYS		No	
15	6022981	28	G2P1L0	12 WKS 1 DAY	24-05-2021	Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	38 weeks 4 days	CDMR with precious pregnancy	No	
16	1039314	22	G2P1L1	13 WKS 2 DAY	24-05-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	38 weeks 3 days		No	
17	6091170	25	G2P1L1	11 WKS 4 DAY	28-05-2021	Non Symptomatic	Escheriachia Coli	Delivered	PTD	34 WEEKS 5 DAY		YES	PREECLAMPSIA
18	6096062	22	PRIMIGRAVIDA	16 WKS	01-06-2021	Non Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	39 weeks	Fetal distress	No	
19	6101649	27	G3A2	13 WEEKS 6 DAYS	05-06-2021	Non Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	41 WEEKS 1 DAY	Fetal distress	No	
20	3469679	32	G3P2L2	13 WEEKS 3 DAYS	07-06-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	39 WEEKS 1 DAY		No	
21	3951607	29	G2P1L1	13 WEEKS 2 DAYS	15-06-2021	Symptomatic	Enterococcus Species	Delivered	FT Emergency LSCS	39 weeks 5 days	Previous LSCS in Labour	No	

22	1054797	18	PRIMIGRAVIDA	12 WEEKS 1 DAY	15-06-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	40 weeks 6 days		No	
23	4767583	26	G2A1	13 WEEKS 2 DAY	15-06-2021	Symptomatic	STAPHYLOCOCCUS SPECIES	Delivered	FT Emergency LSCS	37 weeks 3 days	Fetal distress	No	
24	6072415	23	PRIMIGRAVIDA	12 WEEKS 3 DAYS	15-06-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	39 WEEKS 2 DAYS	Pathological Trace	No	
25	1055161	18	PRIMIGRAVIDA	13 WEEKS 3 DAYS	15-06-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FTND	39 WEEKS 1 DCAY		Yes	Gestational Hypertension
26	6052802	23	G2A1	11 WEKS 6 DAYS	21-06-2021	Symptomatic	Enterococcus species	Delivered	FTND	39 weeks 4 days		No	
27	1056658	23	G2P1L1	14 WEEKS	24-06-2021	Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	38 weeks 5 day	Previous LSCS	No	
28	6049505	21	PRIMIGRAVIDA	11 WEEKS 6 DAYS	28-06-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	38 WEEKS 3 DAYS		NO	
29	6125818	22	PRIMIGRAVIDA	11 WEEKS 2 DAYS	30-06-2021	Symptomatic	Escheriachia Coli	Delivered	PTD	36 WEEKS 2 DAYS		NO	
30	6129645	32	G3A2	12 WEEKS 6 DAYS	06-07-2021	Symptomatic	CITOBACTER	Delivered	FT Elective LSCS	37 weeks 1 day	CDMR	Yes	Preeclampsia
31	6118503	18	G2A1	12 WEEKS 1 DAY	07-07-2021	Non Symptomatic	STAPHYLOCOCCUS SPECIES	Delivered	FTND	40 WEEKS		NO	
32	6107162	31	G2P1L1	12 WEEKS 6 DAYS	09-07-2021	Symptomatic	KLebsiella Oxytoca	Delivered	FTND	39 weeka 1 day		No	
33	1058481	31	G2P1L1	13 WEEKS 4 DAY	12-07-2021	Symptomatic	KLebsiella Pneumoniae	Delivered	FTND	37 weeks		No	
34	6141580	24	PRIMIGRAVIDA	13 WEEKS 4 DAYS	14-07-2021	Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	39 WEEKS 1 DAY	Fetal distress	NO	
35	1058945	20	G2P1L1	13 WEEKS 5 DAYS	14-07-2021	Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	40 WEEKS 0 DAYS	Previous LSCS	NO	
36	1059060	20	G2A1	13 WEEKS 6 DAYS	14-07-2021	Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	PTD	35 WEEKS 5 DAYS		NO	
37	6061548	24	G2A1	13 WEEKS 5 DAYS	15-07-2021	Symptomatic	Enterococcus species	Delivered	FTVD	41 WEEKS 5 DAYS		NO	
38	6142596	30	G2A1	16 WEEKS 5 DAYS	16-07-2021	Non Symptomatic	Klebsiella Oxytoca	Delivered	FT Emergency LSCS	40 WEEKS 5 DAYS	PERSISTENT FETAL TACHYCARDIA	NO	
39	1059100	29	G2A1	13 WEEKS 6 DAYS	16-07-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	39 weeks 4 days	Fetal distress	NO	
40	6144013	20	PRIMIGRAVIDA	13 WEEKS 6 DAYS	30-07-2021	Symptomatic	Enterococcus species	Delivered	FTND	39 weeka 1 day		No	
41	6091181	20	PRIMIGRAVIDA	14 WEEKS 5 DAYS	31-07-2021	Symptomatic	KLebsiella Pneumoniae	Delivered	FTND	39 WEEKS 2 DAYS		No	
42	6135298	32	G4P1L1D1A1	15 WEEKS 6 DAYS	04-08-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	40 weeks 1 day		Yes	Gestational Hypertension
43	1062081	20	PRIMIGRAVIDA	11 WEEKS 6 DAYS	21-08-2021	Symptomatic	Coagulase Negative Staphylococcus Aureus	Delivered	FT Emergency LSCS	38 weeks 2 days	Fetal Distress	No	
44	6167350	25	G2P1L1	16 WEEKS 4 Days	09-08-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FTVD	39 WEEKS 2 DAYS		No	
45	1063498	19	PRIMIGRAVIDA	13 WEEKS 4 DAYS	09-08-2021	Non Symptomatic	Enterococcus Faecalis	Delivered	FTND	39 weeks 5 days		No	

46	6053862	32	G4P2L1A1D1	13 WEEKS 4 DAYS	09-08-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTVD	40 Weeks 4 days		No	
47	1063110	32	G4P2L2A1	14 WEEKS 6 DAYS	11-08-2021	Symptomatic	Enterococcus Species	Delivered	FT Elective LSCS	38 weeks 2 days	Previous 2 LSCS	No	
48	3167839	30	G3P1L1A1	14 WEEKS 4 DAYS	18-08-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FTND	39 WEEKS 3 DAYS		NO	
49	1065335	27	G2P1L1	14 WEEKS 4 DAYS	21-08-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	39 weeks 3 days		No	
50	6126278	20	PRIMIGRAVIDA	16 weeks 4 days	25-08-2021	Non Symptomatic	Citrobacter Species	Delivered	FTND	39 WEEKS 4 DAYS		NO	
51	6061020	24	G2A1	12 WEEKS 6 DAYS	25-08-2021	Non Symptomatic	Klebsiella Oxytoca	Delivered	FTVD	37 weeks 5 days		Yes	Gestational Hypertension
52	6174887	21	PRIMIGRAVIDA	13 WEEKS 1 DAY	26-08-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FTVD	39 WEEKS 5 DAYS		NO	
53	6193528	25	G2P1L1	16 WEEKA 1 DAY	29-08-2021	Symptomatic	Klebsiella Oxytoca	Delivered	FT Emergency LSCS	38 weeks 1 day	Severe Preeclampsia with uncontrolled HTN	Yes	Severe Preeclampsia
54	5282657	28	G2P1L1	11 WEEKS 4 DAYS	08-09-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	39 WEEKS		NO	
55	6208988	23	PRIMIGRAVIDA	12 WEEKS 1 DAY	09-09-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	PTD	35 WEEKS		NO	
56	6076150	24	PRIMIGRAVIDA	12 WEEKS 2 DAYS	13-09-2021	Symptomatic	Klebsiella Oxytoca	Delivered	FT EMERGENCY LSCS	39 WEEKS	MSL WITH UNFAVOURABLE CERVIX	NO	
57	1069914	28	G3P1L1A1	12 WEEKS 1 DAY	18-09-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	38 WEEKS		NO	
58	6126200	21	PRIMIGRAVIDA	12 WEWEKS 1 DAY	22-09-2021	Non Symptomatic	Methicillin Resistant Stayphylococcus Aureus	Delivered	FT EMERGENCY LSCS	38 weeks 1 day	FAILED INDUCTION	NO	
59	1070203	24	G2A1	12 WEEKS	23-09-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	PTD	34 WEEKS 4 DAY		NO	
60	1070609	31	G4P2L2	13 WEEKS 1 DAY	24-09-2021	Symptomatic	Enterococcus faecium	Delivered	FT Elective LSCS	38 weeks 1 day	Previous 2 LSCS	No	
61	1070203	24	G2P1L1	11 WEEKS 4 DAYS	24-09-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	38 weeks 1 day	Fetal Distress	No	
62	6229439	30	g3p2l2	14 WEEKS 1 DAY	26-09-2021	Symptomatic	Pseudomonas aeruguinosa	Delivered	FTND	39 WEEKS 1 DAY		No	
63	6048699	32	G3P1L1A1	14 WEEKS 3 DAYS	29-09-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	39 WEEKS 2 DAYS		Yes	Gestational Hypertension
64	1072743	33	G4P2L2A1	11 WEEKS 6 DAYS	04-10-2021	Symptomatic	Enterobacter Cloacae	Delivered	FT Emergency LSCS	39 weeks 1 day	Fetal Disttress	No	
65	6195365	23	PRIMIGRAVIDA	14 WEEKS 6 DAYS	05-10-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	37 weeks 5 days		No	
66	4224419	26	G2A1	12 WEEKS 3 DAYS	06-10-2021	Symptomatic	Enterococcus Faecium	Delivered	PTD	36 weeks 5 days		No	
67	1072610	25	G3P1L1A1	15 WEEKS 1 DAY	12-10-2021	Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	38 weeks 3 days	Fetal Disttress	No	
68	1074483	20	PRIMIGRAVIDA	12 weeks 3 days	19-10-2021	Symptomatic	Enterococcus Faecium	Delivered	FTND	39 weeks 1 day		No	
69	6250985	23	g2a1	12 weeks 1 day	19-10-2021	Symptomatic	Pseudomonas aeruguinosa	Delivered	FT Emergency LSCS	39 weeks 1 day	Fetal distress	No	
70	6247406	19	PRIMIGRAVIDA	11 weeks 3 days	23-10-2021	Symptomatic	Escheriachia Coli	Delivered	PTD	36 weeks 1 day		No	

71	61703030	25	G2A1	12 weeks 4 days	23-10-2021	Symptomatic	Enterococcus Species	Delivered	FT Emergency LSCS	38 weeks 6 days	Failed Induction	YES	Preeclampsia
72	1076605	23	G3P2L2	13 WEEKA 1 DAY	23-10-2021	Non Symptomatic	Enterococcus Species	Delivered	FTVD	38 weeks 5 days		No	
73	1071851	25	G2A1	12 WEEKS	26-10-2021	Non Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	40 weeks 2 days	Anamnios	No	
74	6262146	24	PRIMIGRAVIDA	13 WEEKS 3 DAYS	26-10-2021	Symptomatic	Escheriachia Coli	Delivered	FTND	39 weeks 1 day		Yes	Gestational Hypertension
75	1077206	32	G4P2L2A1	12 WEEKS 3 DAYS	26-10-2021	Symptomatic	KLebsiella Pneumoniae	Delivered	FTVD	39 weeks 3 days		Yes	Gestational Hypertension
76	6228820	30	G2P1L1	11 WEEKS 4 DAYS	27-10-2021	Non Symptomatic	Staphylococcus Aureus	Delivered	FTVD	37 weeks		No	
77	1076770	21	PRIMIGRAVIDA	15 WEEKS 2 DAYS	28-10-2021	Non Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	39 weeks 1 day	Fetal Distress	No	
78	6152337	26	G2A1	12 WEEKS 5 DAYS	28-10-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	38 WEEKS 6 DAYS		NO	
79	5096708	28	G2P1L1	13 WEEKS 1 DAY	28-10-2021	Symptomatic	Klebsiella Pneumoniae	Delivered	FTVD	39 weeka 1 day		Yes	Gestational Hypertension
80	6186307	32	G4P1L1A2	12 WEEKS	31-10-2021	Non Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	PT Emergency LSCS	36 weeks 5 days	Previous LSCS in Labour	No	
81	1078009	30	G3P1L1A1	11 WEEKS 2 DAYS	01-11-2021	Symptomatic	Escheriachia Coli	Delivered	PTD	36 Weeks 1 day		No	
82	1076698	19	PRIMIGRAVIDA	14 WEEKS 2 DAYS	02-11-2021	Non Symptomatic	Staphylococcus aureus	Delivered	FT Emergency LSCS	40 weeks 5 days	Anamnios	No	
83	1077280	24	G2P1L0	12 WEEKS 3 DAYS	09-11-2021	Non Symptomatic	Escheriachia Coli	Delivered	FTND	39 weeks		No	
84	6241262	24	G3P1L1A1	13 WEEKS 3 DAYS	09-11-2021	Non Symptomatic	Enterococcus Species	Delivered	FTVD	39 weeks		No	
85	1079217	20	PRIMIGRAVIDA	12 WEEKS 1 DAY	09-11-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FTVD	37 WEEKS 5 DAYS		NO	
86	6102920	32	G3P2L1D`	12 WEEKS 4 DAYS	10-11-2021	Non Symptomatic	Klebsiella Oxytoca	Delivered	FTND	38 weeks 5 days		No	
87	6220945	21	PRIMIGRAVIDA	12 WEEKS 1 DAY	10-11-2021	Non Symptomatic	CITROBACTER SPECIES	Delivered	FTVD	39 weeks		No	
88	6073219	24	G2A1	13 WEEKS 5 DAY	11-11-2021	Symptomatic	Enterococcus Species	Delivered	FT Emergency LSCS	37 weeks 4 days	Severe Preeclampsia with Uncontrolled HTN	Yes	Severe Preeclampsia
89	1080750	31	G2A1	13 weeks 6 days	17-11-2021	Non Symptomatic	Pseudomonas aeruguinosa	Delivered	FTVD	38 weeks 4 days		Yes	Gestational Hypertension
90	1079990	22	PRIMIGRAVIDA	11 WEEKS 4 DAYS	19-11-2021	Symptomatic	CITROBACTER SPECIES	Delivered	PT Emergency LSCS	35 weeks 1 day	Severe Preeclampsia with Uncontrolled HTN	Yes	Preeclampsia
91	6290276	22	PRIMIGRAVIDA	12 WEEKS 2 DAYS	20-11-2021	Non Symptomatic	STAPHYLOCOCCUS AUREUS	Delivered	FTVD	38 weeks 5 days		No	
92	1081446	25	G2P1L1	11 WEEKS 5 DAYS	20-11-2021	Symptomatic	Escheriachia Coli	Delivered	FTND	39 weeks		No	
93	6221593	21	PRIMIGRAVIDA	12 WEEKS 1 DAY	27-11-2021	Symptomatic	KLebsiella Oxytoca	Delivered	FTND	39 weeks 6 days		No	

94	1080480	40	G4P2L1A1D1	12 WEEKS 4 DAYS	19-11-2021	Symptomatic	KLebsiella Oxytoca	Delivered	FT Elective LSCS	38 weeks 1 day	Previous 2 LSCS	No	
95	1080020	31	G2A1	12 WEEKS	20-11-2021	Non Symptomatic	Klebsiella Pneumoniae	Delivered	FT Emergency LSCS	39 weeks 3 days	Fetal Distress	No	
96	6127686	23	PRIMIGRAVIDA	13 WEEKS 3 DAYS	04-12-2021	Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FT Emergency LSCS	39 weeks 4 days	FAILED INDUCTION	no	
97	1083370	20	PRIMIGRAVIDA	12 WEEKS 3 DAYS	04-12-2021	Symptomatic	Entercoccus Species	Delivered	PTD	34 weeks 1 days		No	
98	1082442	19	PRIMIGRAVIDA	12 WEEKS 1 DAY	09-12-2021	Non Symptomatic	Klebsiella pneumoniae	Delivered	FTND	37 weeks 4 days		No	
99	1084791	24	PRIMIGRAVIDA	13 WEEKS	10-12-2021	Symptomatic	Enterococcus Species	Delivered	FTND	40 weeks 1 day		No	
100	6278883	26	PRIMIGRAVIDA	11 WEEKS 5 DAYS	10-12-2021	Symptomatic	Escheriachia Coli	Delivered	FTVD	38 weeks 4 days		No	
101	1084248	22	PRIMIGRAVIDA	13 WEEKS 1 DAY	10-12-2021	Symptomatic	Streptococcus species	Delivered	FT Emergency LSCS	40 weeks	MSL with unfavourable cervix	no	
102	6167411	22	PRIMIGRAVIDA	14 WEEKS 5 DAYS	11-12-2021	Non Symptomatic	Klebsiella species	Delivered	FTND	39 WEEKS		NO	
103	1084362	20	PRIMIGRAVIDA	11 WEEKS 3 DAYS	11-12-2021	Non Symptomatic	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS	Delivered	FTVD	39 WEEKS		NO	
104	1086581	21	PRIMIGRAVIDA	15 WEEKS 4 DAYS	19-12-2021	Symptomatic	Escheriachia Coli	Delivered	FT Emergency LSCS	40 weeks 4 days	FAILED INDUCTION	Yes	Gestational Hypertension
105	1096716	28	G2A1	14 WEEKA 1 DAY	22-02-2022	Symptomatic	Escheriachia Coli	Delivered	FTVD	38 weeks 3 days		Yes	Gestational Hypertension
106	1103168	25	G2P1L1	14 WEEKS 1 DAY	29-03-2022	Non Symptomatic	Pseudomonas aeruguinosa	Delivered	PT Emergency LSCS	36 WEEKS 4 DAYS	Severe PREECLAMPSIA WITH FETAL DISTRESS	Yes	Severe Preeclampsia
107	5973650	22	G2A1	12 WEEKS 1 DAY	16-02-2021	Symptomatic	Enterococcus Species	Delivered	FTVD	39 WEEKS 6 DAYS		Yes	Gestational Hypertension
108	6284328	21	PRIMIGRAVIDA	11 WEEKS 5 DAYS	15-02-2022	Non Symptomatic	STAPHYLOCOCCUS AUREUS	Not Delivered				No	
109	1098808	29	G3A2	14 WEEKS35 DAYS	01-03-2022	Symptomatic	Escheriachia Coli	Delivered	FT Elective LSCS	38 weeks 3 days	Precious Pregnancy	No	
110	6438693	19	PRIMIGRAVIDA	11 WEEKS 6 DAYS	08-03-2022	Non Symptomatic	Enterococcus Species	Delivered	FTND	39 WEEKS		No	

S. NO	AGE	OBSTETRIC SCORE	GESTATIONAL AGE AT TEST	UTI: DIAGNOSED/ NOT DIAGNOSED	DATE OF TEST	SYMPTOM ATIC	DELIVERED/ NOT DELIVERED	MODE OF DELIVER	GESTATIONAL AGE AT DELIVERY	INDICATION OF SECTION	HYPERTENSI ON	TYPE OF HYPERTENSION
1	25	G2P1L1	11 WEEKS 6 DAYS	NO	04-07-2021	NO	Delivered	FTND	38 weeks 1 day		No	
2	31	G4P2L2A1	12 WEEKS 1 DAY	NO	01-07-2021	NO	Delivered	FTVD	39 WEEKS 5 DAYS		No	
3	21	PRIMIGRAVIDA	13 WEEKS 4 DAYS	NO	02-07-2021	NO	Delivered	FT Emergecy LSCS	40 weeks 1 day	Anamnios	No	
4	24	G2A1	15 WEEKS 1 DAY	NO	04-07-2021	NO	Delivered	FTVD	37 WEEKS 5 DAYS		NO	
5	22	PRIMIGRAVIDA	16 WEEKS 4 DAYS	NO	14-07-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
6	29	G3P2L2	15 WEEKS 6 DAYS	NO	07-07-2021	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
7	24	G2P1L1	14 WEEKS 3 DAYS	NO	17-07-2021	NO	Delivered	FT EMERGENCY LSCS	40 weeks 1 day	FETAL DISTRESS	NO	
8	26	G2P1L1	16 WEEKS 4 DAYS	NO	28-07-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
9	24	G2A1	12 WEEKS 5 DAYS	NO	28-07-2021	NO	Delivered	FT EMERGENCY LSCS	37 weeks 4 days		No	
10	22	PRIMIGRAVIDA	13 WEEKS 5 DAYS	NO	29-07-2021	NO	Delivered	FTVD	38 WEEKS 2 DAYS		NO	
11	32	G4P2L2A1	11 WEEKS 5 DAYS	NO	01-08-2021	NO	Delivered	FT Elective LSCS	38 weeks 1 day	Previous LSCS	No	
12	27	G2P1L1	14 WEEKS 4 DAYS	NO	26-07-2021	NO	Delivered	FTVD	38 WEEKS 4 DAYS		YES	GESTATIONAL HYPERTENSION
13	30	G3P1L1A1	11 WEEKS 3 DAYS	NO	27-07-2021	NO	Delivered	FT Emergency LSCS	37 weeks 1 day	FETAL DISTRESS	No	
14	26	G2P1L1	14 WEEKS 1 DAY	NO	26-07-2021	NO	Delivered	FTND	39 WEELS 4 DAYS		NO	
15	24	PRIMIGRAVIDA	11 WEEKS 1 DAY	NO	04-08-2021	NO	Delivered	FTVD	39 WEEKS 4 DAYS		NO	
16	20	PRIMIGRAVIDA	12 WEEKS 2 DAYS	NO	05-04-2021	NO	Delivered	FT Emergency LSCS	38 weeks 6 days	Anamanios	No	
17	28	G2P1L1A1	15 WEEKS 1 DAY	NO	06-04-2021	NO	Delivered	PTD	36 WEEKS 1 DAY		NO	
18	22	PRIMIGRAVIDA	13 WEEKS 4 DAYS	NO	08-04-2021	NO	Delivered	FTND	37 WEEKS 1 DAY		NO	
19	24	G2A1	11 WEEKS	NO	27-04-2021	NO	Delivered	FTND	38 WEEKS 5 DAYS		NO	
20	27	G2P1L1	12 WEEKS 1 DAY	NO	09-04-2021	NO	Delivered	FTVD	39 WEEKS 6 DAYS		NO	
21	31	G4P2L1	14 WEEKS 1 DAY	NO	11-04-2021	NO	Delivered	FT Emergecy LSCS	39 weeks	Previous LSCS	No	
22	22	PRIMIGRAVIDA	16 WEEKS 1 DAY	NO	10-04-2021	NO	Delivered	FT Emergency LSCS	37 weeks	Preeclampsia with Fetal	no	
23	21	PRIMIGRAVIDA	14 WEEKS 2 DAYS	NO	11-05-2021	NO	Delivered	FTVD	38 WEEKS 5 DAYS		NO	
24	29	G3P2L2	15 WEEKS 1 DAY	NO	21-05-2021	NO	Delivered	FTND	39 WEEKS		NO	
25	27	G2P1L1	14 WEES 2 DAYS	NO	11-05-2021	NO	Delivered	FT Emergency LSCS	37 weeks 4 days		No	
26	22	PRIMIGRAVIDA	13 WEEKS 1 DAY	NO	12-05-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
27	24	G2A1	13 WEEKS 4 DAYS	NO	21-05-2021	NO	Delivered	FT Elective LSCS	38 weels 3 days	Previous LSCS	no	
28	39	G4P2L2A1	12 WEEKS	NO	22-06-202	NO	Delivered	FT Elective LSCS	39 WEEKS	PREVIOUS 2 LSCS	No	
29	26	G2P2L2	11 WEEKS 4 DAYS	NO	24-06-2021	NO	Delivered	FT EMERGENCY LSCS	37 WEEKS 4 DAYS	FETAL DISTRESS	NO	
30	20	PRIMIGRAVIDA	12 WEEKS 6 DAYS	NO	28-06-2021	NO	Delivered	FTND	38 WEEKS 1 DAY		NO	
31	22	PRIMIGRAVIDA	12 WEEKS 3 DAYS	NO	02-06-2021	NO	Delivered	FTVD	38 WEEKS 5 DAYS		YES	GESTATIONAL HYPERTENSION
32	20	PRIMIGRAVIDA	11 WEEKS 5 DAYS	NO	26-06-2021	NO	Delivered	FTVD	40 WEEKS 1 DAY		NO	
33	26	G3A2	11 WEEKS 1 DAY	NO	27-06-2021	NO	Delivered	FTND	37 WEEKS		No	
34	25	G2P1A1	13 WEEKS	NO	27-08-2021	NO	Delivered	FTND	37 WEEKS 5 DAYS		NO	
35	20	PRIMIGRAVIDA	14 WEEKS 1 DAY	NO	24-06-2021	NO	DELIVERED	FTVD	38 weeks 1 day		NO	
36	25	G2A1	13 WEEKS 4 DAYS	NO	02-05-2021	NO	Delivered	FTND	39 WEEKS		NO	
37	26	G2P1L1	13 WEEKS 4 DAYS	NO	04-05-2021	NO	Delivered	PT EMERGENCY LSCS	35 WEEKS 1 DAY	VIOUS LSCS IN LABO	NO	
38	27	G2P1L1	12 WEEKS 5 DAYS	NO	06-07-2021	NO	Delivered	FTND	37 weeks 4 days		NO	
39	23	PRIMIGRAVIDA	11WEEKS 4 DAYS	NO	08-06-2021	NO	Delivered	PTD	36 WEEKS 4 DAYS		NO	
40	24	G2P1L1	12 WEEKS 2 DAYS	NO	18-11-2021	NO	Delivered	FT EMERGENCY LSCS	38 weeks 1 day	FETAL DISTRESS	No	
41	22	PRIMIGRAVIDA	14 WEEKS 6 DAYS	NO	04-08-2021	NO	Delivered	PTD	36 WEEKS		NO	

42	23	G2A1	12 WEEKS 1 DAY	NO	02-08-2021	NO	Delivered	FTVD	38 WEEKS 3 DAYS		YES	GESTATIONAL HYPERTENSION
43	22	PRIMIGRAVIDA	11 WEEKS 1 DAY	NO	16-08-2021	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
44	20	PRIMIGRAVIDA	12 WEEKS	NO	03-08-2021	NO	Delivered	PT EMERGENCY LSCS	34 WEEKS 2 DAYS	Preeclampsia with Fetal	YES	Preeclampsia
45	25	G2P1L1	11 WEEKS 6 DAYS	NO	10-08-2021	NO	Delivered	FTND	38 weeks 1 day		No	
46	21	PRIMIGRAVIDA	12 WEEKS 1 DAY	NO	09-08-2021	NO	Delivered	FTVD	39 WEEKS 5 DAYS		NO	
47	21	PRIMIGRAVIDA	13 WEEKS 4 DAYS	NO	04-08-2021	NO	Delivered	FT Emergecy LSCS	40 weeks 1 day	Anamnios	No	
48	24	G2A1	15 WEEKS 1 DAY	NO	10-08-2021	NO	Delivered	FTVD	37 WEEKS 5 DAYS		NO	
49	22	PRIMIGRAVIDA	16 WEEKS 4 DAYS	NO	09-08-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
50	29	G3P2L2	15 WEEKS 6 DAYS	NO	05-06-2021	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
51	24	G2P1L1	14 WEEKS 3 DAYS	NO	24-11-2021	NO	Delivered	FT EMERGENCY LSCS	40 weeks 1 day	FETAL DISTRESS	YES	GESTATIONAL HYPERTENSION
52	26	G2P1L1	16 WEEKS 4 DAYS	NO	23-11-2021	NO	Delivered	PTD	33 WEEKS 2 DAYS		No	
53	24	G2A1	12 WEEKS 5 DAYS	NO	22-11-2021	NO	Delivered	FT EMERGENCY LSCS	37 weeks 4 days		NO	
54	22	PRIMIGRAVIDA	13 WEEKS 5 DAYS	NO	12-11-2021	NO	Delivered	FT EMERGENCY LSCS	38 WEEKS 2 DAYS	PLACENTA PREVIA	NO	
55	32	G4P2L2A1	11 WEEKS 5 DAYS	NO	09-11-2021	NO	Delivered	FT Elective LSCS	38 weeks 1 day	Previous LSCS	NO	
56	27	G2P1L1	14 WEEKS 4 DAYS	NO	11-11-2021	NO	Delivered	PTD	34 WEEKS 2 DAYS		NO	
57	30	G3P1L1A1	11 WEEKS 3 DAYS	NO	24-11-2021	NO	Delivered	FT Emergency LSCS	37 weeks 1 day	FETAL DISTRESS	NO	
58	26	G2P1L1	14 WEEKS 1 DAY	NO	11-11-2021	NO	Delivered	FTND	39 WEELS 4 DAYS		NO	
59	24	PRIMIGRAVIDA	11 WEEKS 1 DAY	NO	12-11-2021	NO	Delivered	FTVD	39 WEEKS 4 DAYS		NO	
60	20	PRIMIGRAVIDA	12 WEEKS 2 DAYS	NO	09-11-2021	NO	Delivered	FT Emergency LSCS	38 weeks 6 days	Anamanios	NO	
61	28	G2P1L1A1	15 WEEKS 1 DAY	NO	11-11-2021	NO	Delivered	PTD	35 WEEKS 2 DAYS		NO	
62	22	PRIMIGRAVIDA	13 WEEKS 4 DAYS	NO	22-11-2021	NO	Delivered	FTND	37 WEEKS 1 DAY		YES	GESTATIONAL HYPERTENSION
63	24	G2A1	11 WEEKS	NO	12-11-2021	NO	Delivered	FTND	38 WEEKS 5 DAYS		NO	
64	27	G2P1L1	12 WEEKS 1 DAY	NO	15-11-2021	NO	Delivered	FTVD	39 WEEKS 6 DAYS		NO	
65	31	G4P2L1	14 WEEKS 1 DAY	NO	15-11-2021	NO	Delivered	FT Emergecy LSCS	39 weeks	Previous LSCS	NO	
66	22	PRIMIGRAVIDA	16 WEEKS 1 DAY	NO	15-11-2021	NO	Delivered	FT Emergency LSCS	37 weeks	Preeclampsia with Fetal	YES	Preeclampsia
67	21	PRIMIGRAVIDA	14 WEEKS 2 DAYS	NO	14-11-2021	NO	Delivered	FTVD	38 WEEKS 5 DAYS		NO	
68	29	G3P2L2	15 WEEKS 1 DAY	NO	16-11-2021	NO	Delivered	FTND	39 WEEKS		NO	
69	27	G2P1L1	14 WEES 2 DAYS	NO	21-11-2021	NO	Delivered	FT Emergency LSCS	37 weeks 4 days		NO	
70	22	PRIMIGRAVIDA	13 WEEKS 1 DAY	NO	25-11-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
71	24	G2A1	13 WEEKS 4 DAYS	NO	25-11-2021	NO	Delivered	FT Elective LSCS	38 weels 3 days	Previous LSCS	NO	
72	39	G4P2L2A1	12 WEEKS	NO	25-11-2021	NO	Delivered	FT Elective LSCS	39 WEEKS	PREVIOUS 2 LSCS	NO	
73	26	G2P2L2	11 WEEKS 4 DAYS	NO	28-11-2021	NO	Delivered	FT EMERGENCY LSCS	37 WEEKS 4 DAYS	FETAL DISTRESS	NO	
74	20	PRIMIGRAVIDA	12 WEEKS 6 DAYS	NO	29-11-2021	NO	Delivered	FTND	38 WEEKS 1 DAY		NO	
75	22	PRIMIGRAVIDA	12 WEEKS 3 DAYS	NO	30-11-2021	NO	Delivered	FTVD	38 WEEKS 5 DAYS		YES	GESTATIONAL HYPERTENSION
76	20	PRIMIGRAVIDA	11 WEEKS 5 DAYS	NO	31-11-2021	NO	Delivered	FTVD	40 WEEKS 1 DAY		NO	
77	26	G3A2	11 WEEKS 1 DAY	NO	02-12-2021	NO	Delivered	FTND	37 WEEKS		NO	
78	25	G2P1A1	13 WEEKS	NO	04-12-2021	NO	Delivered	FTND	37 WEEKS 5 DAYS		NO	
79	20	PRIMIGRAVIDA	14 WEEKS 1 DAY	NO	04-12-2021	NO	DELIVERED	FTVD	38 weeks 1 day		NO	
80	25	G2A1	13 WEEKS 4 DAYS	NO	04-12-2021	NO	Delivered	FTND	39 WEEKS		NO	
81	26	G2P1L1	13 WEEKS 4 DAYS	NO	06-12-2021	NO	Delivered	PT EMERGENCY LSCS	35 WEEKS 1 DAY	VIOUS LSCS IN LABO	NO	
82	27	G2P1L1	12 WEEKS 5 DAYS	NO	07-12-2021	NO	Delivered	FTND	37 weeks 4 days		NO	
83	23	PRIMIGRAVIDA	11WEEKS 4 DAYS	NO	12-12-2021	NO	Delivered	PTD	36 WEEKS 4 DAYS		NO	

84	24	G2P1L1	12 WEEKS 2 DAYS	NO	12-12-2021	NO	Delivered	FT EMERGENCY LSCS	38 weeks 1 day	FETAL DISTRESS		
85	22	PRIMIGRAVIDA	14 WEEKS 6 DAYS	NO	13-12-2021	NO	Delivered	PTD	36 WEEKS		NO	
86	23	G2A1	12 WEEKS 1 DAY	NO	14-12-2021	NO	Delivered	FTVD	38 WEEKS 3 DAYS		YES	GESTATIONAL HYPERTENSION
87	22	PRIMIGRAVIDA	11 WEEKS 1 DAY	NO	14-12-2021	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
88	20	PRIMIGRAVIDA	12 WEEKS	NO	15-12-2021	NO	Delivered	PT EMERGENCY LSCS	34 WEEKS 2 DAYS	Preeclampsia with Fetal	YES	SEVERE PREECLAMPSIA
89	19	PRIMIGRAVIDA	14 WEEKS 2 DAYS	T1	16-12-2021	NO	DELIVERED	FTVD	38 weeks 1 day		NO	
90	24	G2P1L0	12 WEEKS 3 DAYS	T1	17-12-2021	NO	Delivered	FTND	39 WEEKS		NO	
91	24	G3P1L1A1	13 WEEKS 3 DAYS	T1	18-12-2021	NO	Delivered	PT EMERGENCY LSCS	35 WEEKS 1 DAY	VIOUS LSCS IN LABO	NO	
92	20	PRIMIGRAVIDA	12 WEEKS 1 DAY	T1	18-12-2021	NO	Delivered	FTND	37 weeks 4 days		NO	
93	32	G3P2L1D`	12 WEEKS 4 DAYS	T1	19-12-2021	NO	Delivered	PTD	36 WEEKS 4 DAYS		NO	
94	21	PRIMIGRAVIDA	12 WEEKS 1 DAY	T1	21-12-2021	NO	Delivered	FT EMERGENCY LSCS	38 weeks 1 day	FETAL DISTRESS	NO	
95	24	G2A1	13 WEEKS 5 DAY	T1	22-12-2021	NO	Delivered	PTD	36 WEEKS		NO	
96	31	G2A1	13 weeks 6 days	T1	22-12-2021	NO	Delivered	FTVD	38 WEEKS 5 DAYS		NO	
97	22	PRIMIGRAVIDA	11 WEEKS 4 DAYS	T1	22-12-2021	NO	Delivered	FTND	39 WEEKS		NO	
98	22	PRIMIGRAVIDA	12 WEEKS 2 DAYS	T1	23-12-2021	NO	Delivered	FT Emergency LSCS	37 weeks 4 days		NO	
99	25	G2P1L1	11 WEEKS 5 DAYS	T1	23-12-2021	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
100	21	PRIMIGRAVIDA	12 WEEKS 1 DAY	T1	26-12-2021	NO	Delivered	FT Elective LSCS	38 weels 3 days	Previous LSCS	NO	
101	40	G4P2L1A1D1	12 WEEKS 4 DAYS	T1	26-12-2021	NO	Delivered	FT Elective LSCS	39 WEEKS	PREVIOUS 2 LSCS		
102	31	G2A1	12 WEEKS	T1	28-12-2021	NO	Delivered	FT EMERGENCY LSCS	37 WEEKS 4 DAYS	FETAL DISTRESS		
103	23	PRIMIGRAVIDA	13 WEEKS 3 DAYS	T1	29-12-2021		Delivered	FTND	38 WEEKS 1 DAY			
104	20	PRIMIGRAVIDA	12 WEEKS 3 DAYS	T1	01-01-2022	NO	Delivered	FT EMERGENCY LSCS	38 weeks 1 day	FETAL DISTRESS	No	
105	19	PRIMIGRAVIDA	12 WEEKS 1 DAY	T1	01-01-2022	NO	Delivered	PTD	36 WEEKS		NO	
106	24	PRIMIGRAVIDA	13 WEEKS	T1	03-01-2022	NO	Delivered	FTVD	38 WEEKS 3 DAYS		YES	GESTATIONAL HYPERTENSION
107	26	PRIMIGRAVIDA	11 WEEKS 5 DAYS	T1	04-01-2022	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
108	22	PRIMIGRAVIDA	13 WEEKS 1 DAY	T1	04-01-2022	NO	Delivered	PT EMERGENCY LSCS	34 WEEKS 2 DAYS	Preeclampsia with Fetal	YES	Preeclampsia
109	22	PRIMIGRAVIDA	14 WEEKS 5 DAYS	T1	10-01-2022	NO	Delivered	FTND	38 weeks 1 day		No	
110	20	PRIMIGRAVIDA	11 WEEKS 3 DAYS	T1	11-01-2022	NO	Delivered	FTVD	39 WEEKS 5 DAYS		NO	
111	32	G3P1L1A1	12 WEEKS 2 DAYS	T1	12-01-2022	NO	Delivered	FTND	38 weeks 1 day		No	
112	21	PRIMIGRAVIDA	12 WEEKS 5 DAYS	T1	13-01-2022	NO	Delivered	FTVD	39 WEEKS 5 DAYS		No	
113	25	G2A1	14 WEEKS 5 DAYS	T1	13-01-2022	NO	Delivered	FT Emergecy LSCS	40 weeks 1 day	Anamnios	No	
114	21	PRIMIGRAVIDA	11 WEEKS 6 DAYS	T1	16-01-2022	NO	Delivered	FTVD	37 WEEKS 5 DAYS		NO	
115	30	G2P1L1	12 WEEKS 1 DAY	T1	17-01-2022	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
116	21	PRIMIGRAVIDA	15 WEEKS 4 DAYS	T1	17-01-2022	NO	Delivered	FTND	39 WEEKS 1 DAY		NO	
117	31	G2P1L1	13 WEEKS 4 DAYS	T1	18-01-2022	NO	Delivered	FT EMERGENCY LSCS	40 weeks 1 day	FETAL DISTRESS	NO	
118	22	PRIMIGRAVIDA	13 WEEKS 5 DAYS	T1	19-01-2022	NO	Delivered	FTVD	38 WEEKS 1 DAY		NO	
119	26	PRIMIGRAVIDA	14 WEEKS 2 DAYS	T1	20-01-2022	NO	Delivered	FT EMERGENCY LSCS	37 weeks 4 days		No	
120	26	G2A1	14 WEEKS 6 DAYS	T1	21-01-2022	NO	Delivered	FTVD	38 WEEKS 2 DAYS		NO	
121	20	PRIMIGRAVIDA	14 WEEKS 5 DAYS	T1	21-01-2022	NO	Delivered	FT Elective LSCS	38 weeks 1 day	Previous LSCS	No	
122	24	PRIMIGRAVIDA	16 WEEKS	T1	22-01-2022	NO	Delivered	FTND	37 WEEKS		NO	
123	21	PRIMIGRAVIDA	11 WEEKS 5 DAYS	T1	25-01-2022	NO	Delivered	FTND	37 WEEKS 5 DAYS		NO	
124	25	G2P1L1	12 WEEKS	T1	26-01-2022	NO	DELIVERED	FTVD	38 weeks 1 day		NO	
125	19	PRIMIGRAVIDA	13 WEEKS 1 DAY	T1	30-01-2022	NO	Delivered	FTND	39 WEEKS		NO	
126	28	G2A1	14 WEEKA 1 DAY	T1	05-02-2022	NO	Delivered	PT EMERGENCY LSCS	35 WEEKS 1 DAY	VIOUS LSCS IN LABO	NO	
127	29	G3A2	14 WEEKS35 DAYS	T1	06-02-2022	NO	Delivered	FTND	37 weeks 4 days		NO	
128	19	PRIMIGRAVIDA	11 WEEKS 6 DAYS	T1	08-02-2022	NO	Delivered	PTD	36 WEEKS 4 DAYS		NO	
129	25	PRIMIGRAVIDA	16 WEEKS 5 DAYS	T1	08-02-2022	NO	Delivered	FT Emergency LSCS	40 WEEKS 1 DAY	VIOUS LSCS IN LABO	No	