A DISSERTATION ON

FETOMATERNAL OUTCOME OF PATIENTS WITH FEVER DURING TERM PREGNANCY

Submitted to

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M.S.DEGREE

OBSTETRICS AND GYNAECOLOGY

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MAY2022

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LIST OF ABBREVIATIONS

ARF- Acute Renal Failure

APN- Acute Pyelo Nephritis

BMI- Body mass index

CMV- Cytomegalo Virus

CRS- Congenital Rubella Syndrome

CDC- Centre for Disease Control and prevention

CRP- C-Reactive Protein

DIC- Disseminated Intravacular coagulation

DHF- Dengue Hemorrhagic Fever

DSS- Dengue Shock Syndrome

FIRS- Fetal Inflammatory Response Syndrome

GBS- Group B Streptococci

HSV- Herpes Simplex Virus

IVH- Intra Ventricular Hemorrhage

IUGR- Intra uterine growth retardation

IDSA- Infectious Disease society of America

IL- Interleukins

IF- Interferons

IUFD- Intra Uterine Fetal Death

LPS- Lipopolysaccharides

LBW- Low Birth Weight

PROM- Premature Rupture Of Membranes

PPH- Post partum Hemorrhage

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INTRODUCTION

Fever in pregnancy is a common clinical problem globally. The risk to the mother and fetus is significantly increased in pregnancy complicated by infection and fever.[1] Fever is one of the most frequent reasons for emergency consultation during pregnancy and may be associated with significant adverse outcomes, these being maternal (sepsis, organ damages) obstetrical (miscarriage, preterm birth, chorioamnionitis) or fetal (malformations, fetal demise). However, only one study including mainly second and third trimester pregnant women, evaluated causes of acute undifferentiated fever and 25 % of women had no identified cause.[2]

Fever in pregnant women is defined as a rectal temperature greater than or equal to 38.5˚C at rest and in a normal environment.[3] However, hyperthermia (temperature between 37.5 and 38.3˚C) is a physiological phenomenon resulting from the accumulation of heat in a context of maternal agitation, dehydration, environmental heat, it has no fetal or maternal repercussions. The distinction between hyperthermia and fever is not always easy, any thermal rise during labour must give rise to fears of infection and imposes the necessary preventive and curative measures.[4]

Fevers in the mother must be treated as any other serious illness. The effects on pregnancy depend on the extent of temperature elevation, its duration, and the stage of fetal development when it occurs. Mild exposures during the pre-implantation period and more severe exposures during embryonic and fetal development often result in miscarriage, premature labor, growth restriction, and stillbirth. Fever also causes a wide range of fetal structural and functional defects, with the central nervous system (CNS) being most at risk.[5,6,7]

Moreover, nearly 12 % (IC95 8.6–16.8) of patients with fever during pregnancy required hospitalization in intensive care unit and it has been shown that bacteremia is complicated by fetal loss in 10 % of cases.[8,9] There is no recommendation about fever in pregnant women, but usual care for undifferentiated fever is to introduce probabilistic antibiotic against listeria monocytogenes, responsible for overuse of antibiotics. Improving knowledge about etiology and management of fever in pregnant women could modify antibiotics prescription, which can have consequences on public health.[10] The importance of correctly orienting diagnosis and care is underscored by the current COVID-19 epidemic.

Because of maternal pyrexia, various inflammatory mediators as evidenced by umbilical cord blood cytokines is documented in the absence of neonatal sepsis.[11] The underlying Maternal cytokine polymorphism is strongly associated with both intra partum fever & cerebral palsy at term.[12,13] Some infectious diseases are more severe in pregnancy (e.g. Plasmodium falciparum, Listeria monocytogenes, hepatitis E virus (HEV), herpes simplex virus and influenza).

Increased brain temperature increases oxygen consumption and also lowering the threshold for hypoxic injury. Hypoxic brain injury is increased by hyperthermia in term neonates.[14,15]

Thus we wanted to determine the fetal-maternal impact of fever on full-term pregnancy with the following aim and objectives.

AIM AND OBJECTIVES

Aim:

- To find the maternal complications & fetal outcome of Patients with fever during term pregnancy.

Objectives:-

- To evaluate the association between fever during the term with the major maternal and fetal complications.
- To Compare the outcome with Normal Ante Natal mothers.

REVIEW OF LITERATURE

Background:-

Most of the efforts to reduce maternal mortality in India have focused on haemorrhage, hypertension and sepsis. There is little focus on the impact of infectious diseases on maternal, foetal and neonatal outcomes, which is demonstrated by the lack of well-conducted prospective studies in the literature. Owing to the anatomical and physiological changes, certain infections such as urinary tract infections (UTI) are more common during pregnancy.[16] Infections like Plasmodium falciparum malaria, hepatitis E and H1N1 influenza are more severe in pregnancy due to changes in the immune system.[17]

However, the contribution of these infections towards maternal mortality and near-miss morbidity is not clear. Therefore, they should be incorporated in the national maternal mortality data in greater detail. Only few studies have been conducted on the impact of fever on fetomaternal outcomes.[18] It is postulated that with the development of maternal death surveillance and response (MDSR) guidelines established by Government of India in 2017, more information will be available on the morbidity and mortality burden of these diseases.[19]

The incidence of fever varies with time period. During six month of one study, there was dengue outbreak in trichy. Hence this time period was selected to know the overall outcome of the fever in pregnancy. The fever during pregnancy is the omnious sign. Early detection and prompt management of fever prevents maternal mortality and morbidity. Any maternal hyperthermia (>38.9°C) potentially affect the fetus.[20]

Consequences of fever depends on the extent and duration of temperature elevation, timing of the exposure in pregnancy, and possibly on maternal nutritional status, comorbidities, medications, genetic background, and several other factors. The exposure of maternal temperature has been reported to lead to cell disruptions, vascular disruption and placental infarction, which affect the risk of structural and functional defect in the offspring.[21]

Contemporary obstetrics has witnessed improved maternal and fetal outcomes, owing to several advances. Early detection and prompt management of fever, has been a leading contributor to these developments. Any source of maternal hyperthermia that results in significant core temperature increase (>38.9°C), could potentially affect the fetus. Consequences of hyperthermia depend on the extent and duration of temperature elevation, timing of the exposure in pregnancy, and possibly on maternal nutritional status, comorbidities, medications, genetic background, and several other factors. Maternal fever is implicated as human teratogen. Even short

exposure of maternal temperature has been reported to lead to cell disruptions, vascular disruption and placental infarction, which affect the risk of structural and functional defect in the offspring.[22,23]

Fever:-

According to studies of healthy individuals 18-40 years of age, the mean oral temperature is 36.8C±0.4ºC (98.2ºC ± 0.7ºC) with low levels at 6 A.M and higher levels at 4-6 P.M. An A.M temperature of >37.2ºC or a P.M temperature of >37.7 ºC would define a fever. The normal daily temperature variation is typically 0.5ºC.

Fever is one of the oldest clinical indicators of disease in the mammalian host as well as one of the most common reasons for medical consultations worldwide.[24, 25] Fever often occurs in response to infection, inflammation and trauma. However, this view of fever is merely an oversimplification as a growing body of evidence now suggests that fever represents a complex adaptive response of the host to various immune challenges whether infectious or non-infectious. Although elevated body temperature is an indispensable component of the febrile response, it is not synonymous with fever. It is generally agreed that fever is a regulated rise in body temperature above normal daily fluctuations occurring in conjunction with an elevated thermoregulatory set point.[26, 27]

The complexity of the febrile response may be attributable to its multi-systemic effects orchestrated by endocrine, neurological, immunological and behavioural mechanisms. Apart from a regulated rise in body temperature, fever is also accompanied by various sickness behaviours, changes in metabolic and physiological characteristics of body systems and alterations in immune responses. The febrile response, therefore, remains a significant contributor to the pathogenesis, clinical presentation and outcome of many illnesses and diseases. Consequently, understanding fever and febrile response is vital in the diagnosis, treatment and follow-up of various ailments and diseases.[28]

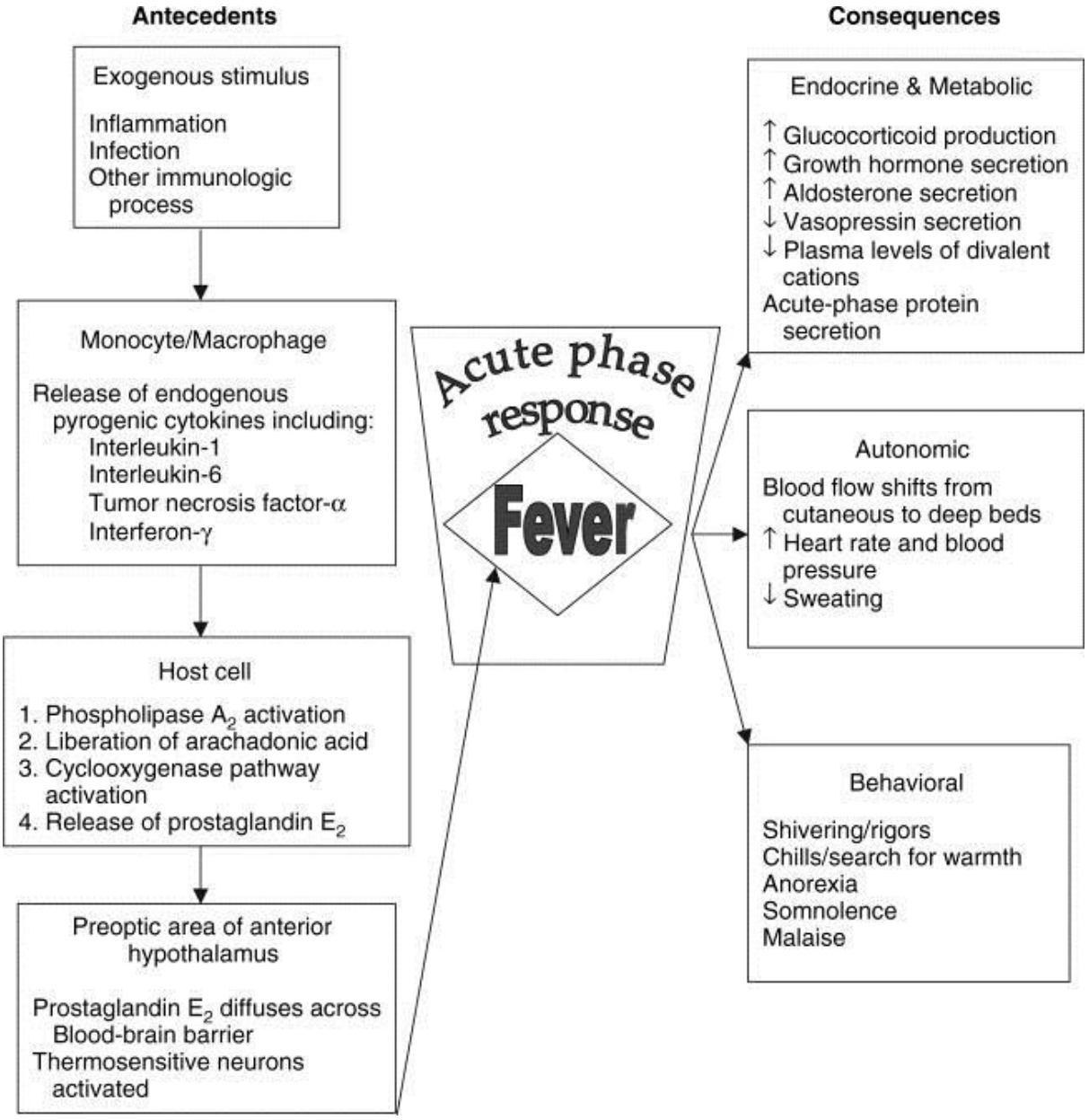
Definition and Terminology:-

Due to wide variability of body temperature in relation to several factors, defining febrile body temperatures remain a subject of controversy with varied definitions by different authors. However, most authors are in agreement with a study in which modern calibrated thermometers were used to measure 700 baseline-oral temperatures of 148 middle aged healthy adults of different races [10]. In this study, the mean oral temperature was 36.8±0.4 ◦C (98.2 ◦F) and body temperature exhibited a circadian rhythm with 99th percentile of the population having maximum morning (at 6.00 am) of 37.2 ◦C and maximum afternoon (at 4.00 pm) temperature of 37.7 ◦C. Thus, on the basis of this study data, fever in healthy middle aged adults may be defined as early morning oral temperature of >37.2 ◦C (>99 ◦F) or a temperature of >37.7 ◦C (>100 ◦F) at anytime during the day.

Based on guidelines for management of febrile illnesses provided by authorities such as World Health Organization (WHO)[29, 30] and the Society of Critical Care Medicine and the Infectious Disease Society of America (IDSA),[31] among others,[32,33] equivalent rectal temperature of ≥38 ◦C (100.4 ◦F) or axillary temperatures of ≥ 37.5 ◦C (99.5 ◦F) are indicative of fever in both adults and children. However, as compared to older children and adults, infants and young children experience higher and more prolonged fevers, more rapid temperature increases, and greater temperature fluctuations. In the geriatric group (>65 years), who are likely to have lower body temperatures, IDSA defines fever as single oral temperature >100 ◦F (>37.8 ◦C); or (2) repeated oral temperatures >99 ◦F (>37.2 ◦C) or rectal temperatures >99.5 ◦F (>37.5 ◦C); or (3) an increase in temperature of >2 ◦F (>1.1 ◦C) over the baseline temperature.[34]

Of the three major sites (i.e. rectal, oral and axillary) used for temperature assessment, rectal temperatures more closely estimate core temperatures than oral temperatures or axillary temperatures.[35] Although, axillary temperatures are convenient to undertake, they are the least accurate method of temperature measurement, especially in adults. Axillary thermometers take longer time to reach equilibrium and they are altered by various factors such as by ambient temperature, sweat, humidity and the density of hair in the axilla.

MECHANISM OF FEVER

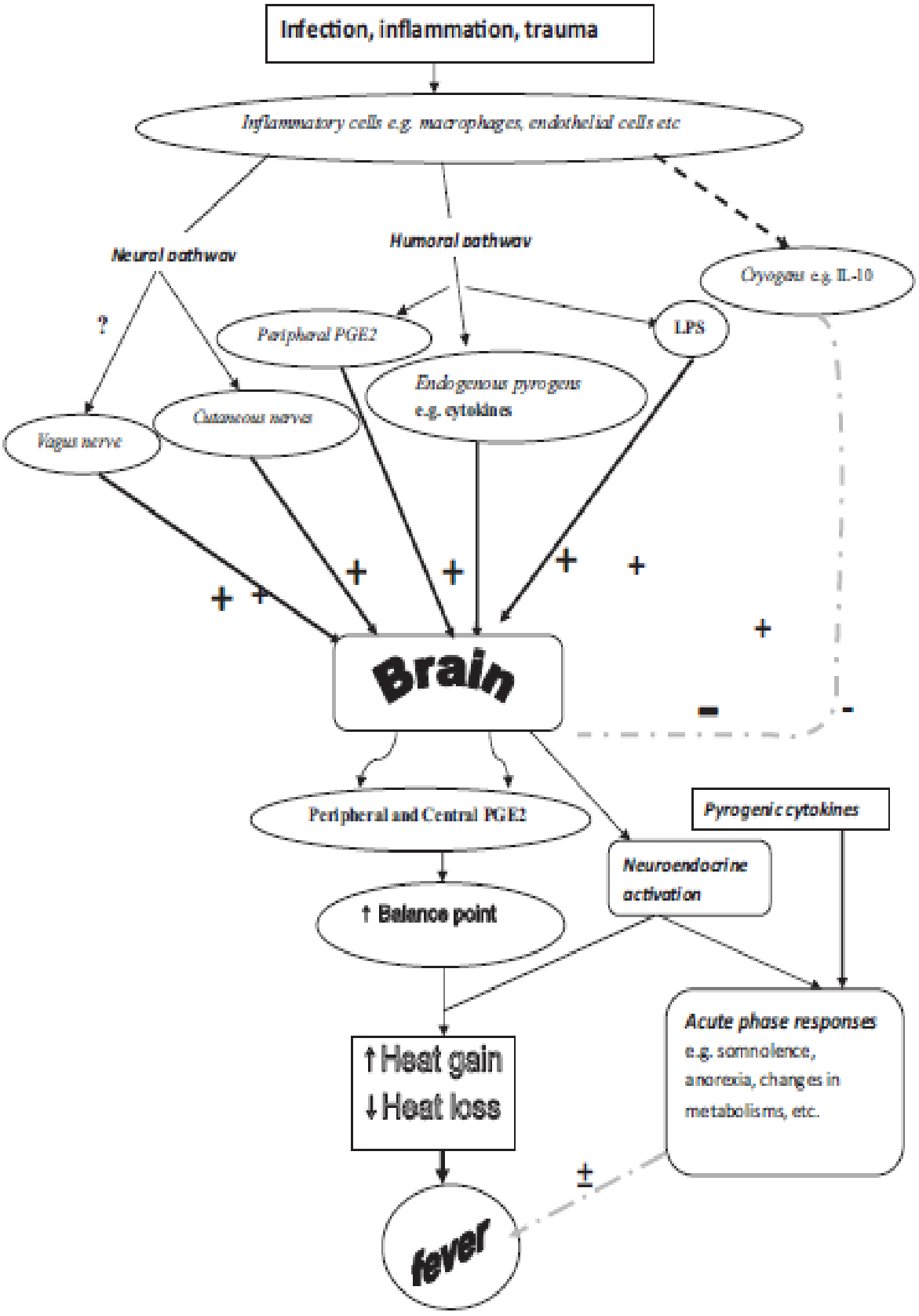


The role of pyrogens and cryogens:-

Pyrogens:

Pyrogens are classified into exogenous (produced outside the host) and endogenous (produced within the host) pyrogens based on their site of production. Exogenous pyrogens are, essentially, part or whole micro-organisms or products of microorganisms such as toxins. The gram negative cell wall component — lipopolysaccharide (LPS), remains the most widely studied exogenous pyrogen and most of the current data of the febrile response are based on studies using LPS as the pyrogenic agent. Other clinical significant endogenous pyrogens include muramyl dipeptidase — a constituent of cell walled micro-organisms, and enterotoxins of Staphylococcus aureus and group A and B Streptococcus collectively named superantigens.

Endogenous pyrogens are mainly pyrogenic cytokines including interleukins (IL) 6, IL-1, interferon gamma (INF-) and ciliary neurotropic factor (CNTF) and tumour necrosis factor, among others. However, TNF has both pyrogenic and antipyretic actions depending on experimental conditions.[36] Endogenous pyrogens are produced by immune cells such as neutrophils, macrophages and lymphocytes as well as by endothelial cells, astrocytes and glial cells in response to exposure to exogenous pyrogens. Certain endogenous substances such as antigen-antibody complexes, inflammatory bile acids, complements and various lymphocyte derived molecules may however servess pyrogens without induction by exogenous pyrogens.


Infection, inflammation, trauma
Inflammatory cells e.g. macrophages, endothelial cells etc
Neural pathway
Humoral pathway
Oxygen e.g. IL-10
?
Peripheral PGE2
LPS
Endogenous pyrogens
e.g. cytokines
Vagus nerve
Cutaneous nerves
+
+
+
+
+
+
+
-
-
Brain
Peripheral and Central PGE2
Pyrogenic cytokines
Neuroendocrine activation
↑ Balance point
↑Heat gain
↓Heat loss
Acute phase responses
e.g. somnolence, anorexia, changes in metabolisms, etc.
±
fever

Classification, types and patterns of fever:-

Fevers can be arbitrary classified into acute, subacute and chronic fevers based on duration. Acute fevers (<7 days in duration) are characteristics of infectious diseases such as malaria and viral-related upper respiratory tract infection while sub-acute fevers (usually not more than 2 weeks in duration) may be seen in cases of typhoid fever and intraabdominal abscess, among others. Chronic or persistent fevers (>2 weeks duration) are typical of chronic bacterial infections such as tuberculosis, viral infections like HIV, cancers and connective tissue diseases. However, any cause of acute fever can become persistent or chronic if untreated.

Based on the height of body temperature, fever can also be classified into low grade, moderate grade, high grade and hyperpyrexia (Table 1).[37] The height of body temperature may have some diagnostic and prognostic implications. Some studies have attributed high grade fevers in infants to serious bacterial infections,[40] although others have also shown that children with high fevers are at equally high risk for serious bacterial infections and for viral illness.

The height of fever may occasionally correlate with severity of illness, as suggested in experimental shigellosis and dengue virus infection,[40,41] as well as in acute falciparum malaria where presence of hyperpyrexia denotes complicated disease with personnel. Fever associated with relative bradycardia (temperature pulse dissociation or Faget's sign) is a feature of untreated typhoid, leishmaniasis, brucellosis, Legionnaire's disease and psittacosis, Yellow Fever, among others.[42]

Table 1 Normal and febrile body temperature ranges (rectal temperatures).

Body temperature	°C	°F
Normal	37–38	98.6–100.4
Mild/low grade fever	38.1–39	100.5–102.2
Moderate grade fever	39.1–40	102.2–104.0
High grade fever	40.1–41.1	104.1–106.0
Hyperpyrexia[a]	>41.1	>106.0

Data from Refs. [31,32]. NB—hypothermia = rectal temperature <35 °C (<95 °F).

[a] Hyperpyrexia in severe malaria is defined as rectal temperature above 40 °C [33]. C, centigrade; F, Fahrenheit.

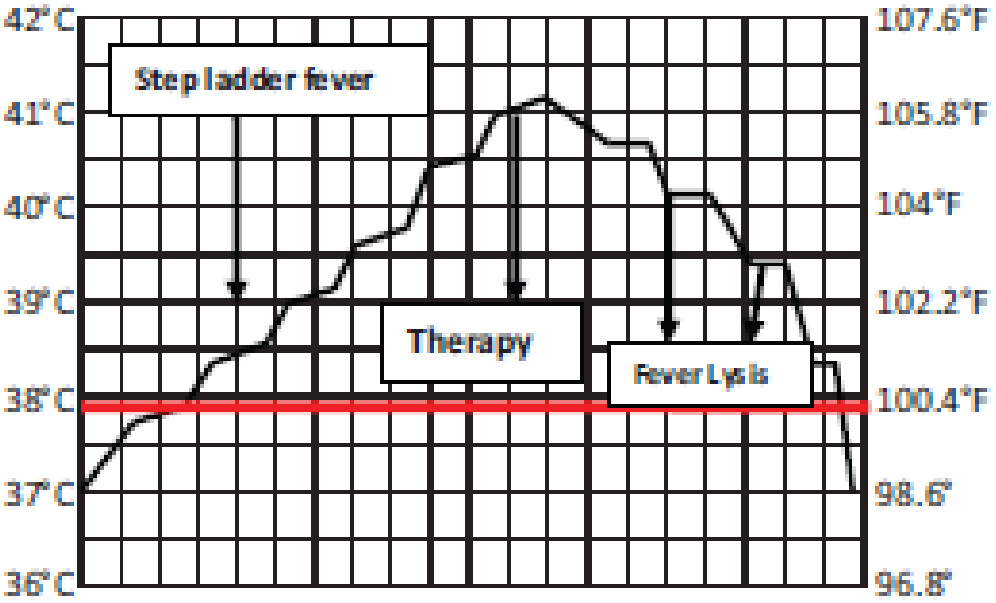


Figure 2 Continuous step ladder pattern of fever classical of typhoid fever. *Legend*: Continuous fevers do not fluctuate more than about 1 °C (1.5 °F) during 24 h, but at no time touches normal. Following effective therapy of typhoid fever, fever defervescence occurs gradually, by lysis.

Intermittent fever is defined as fever present only for several hours during the day. This pattern of fever can be seen in malaria, pyogenic infections, tuberculosis (TB), schistosomiasis, lymphomas, leptospira, borrelia, kala-azar, or septicemia. Sources of continuous, intermittent or transient bacteraemia may lead to continuous, intermittent or transient fevers respectively. In malaria, depending on the species of parasite, fever can occur with a periodicity of 24 h (quotidian due to plasmodium falciparum), 48 h (tertian — plasmodium ovale and vivax), or 72 h (quartan — Plasmodium malariae). The Pel-Epstein's fever is an intermittent low grade fever characterised by 3—10 days of fever with subsequent a febrile periods of 3—10 days. It is thought to be a typical but rare manifestation of Hodgkin's lymphoma.[42,43]

Remittent fever is defined as fever with daily fluctuations exceeding 2 ◦C but at no time touches normal.[43] Remittent fevers are often associated with infectious diseases such as infective endocarditis, rickettsiae infections, brucellosis, among others. Relapsing fevers refer to those that are recurring and separated by periods with low-grade fever or no fever.[44]. Periodic or relapsing fevers are seen in malaria, lymphoma, borrelia, cyclic neutropenia, and rat-bite fever[44]. Fever associated with night sweats has been described in infectious diseases such as TB, Nocardia, brucellosis, liver or lung abscess and sub-acute infective endocarditis, as well as in noninfectious diseases such as polyarteritis nodosa and cancers such as lymphomas.[45]

Pregnancy:-

Some infectious diseases are more common or more severe in pregnancy (e.g. Plasmodium falciparum, Listeria monocytogenes, hepatitis E virus (HEV), herpes simplex virus and influenza).[46,47,48] However, for most common causes of fever in the tropics there is little evidence as to whether pregnant women are at higher risk of disease, of severe disease and what the impacts are on mothers and their offspring, by pathogens and gestational age.[49,50]

Anatomical and physiological changes may predispose pregnant women to certain infections, such as those of the urinary tract. These occur in ~8% of pregnant women, causing pyelonephritis in 1–2%, most commonly during the second trimester, and may lead to preterm birth and low birth weight.[51,52] Listeriosis is reported to be 20 times more common during pregnancy than in the non-pregnant population, likely because of immunosuppression during pregnancy[53] with chorioamnionitis, placental abscesses, abortion, prematurity, fetal distress, vertical transmission and neonatal death reported.[54] Adverse birth outcomes such as maternal death, premature delivery, and low birth weight have been well documented in pregnant women with P. falciparum and P. vivax infection.[55, 56] Dengue, leptospirosis, typhoid fever and rickettsioses are common causes of fever in tropical areas[57] but whether the risk and severity of these diseases are higher in pregnancy is unclear.[58]

Obstetricians often face problems in treating fever in pregnancy due to its atypical presentations. Maternal immune function is usually decreased in normal pregnancy and many of the potent antibiotics should be used with caution in pregnant women due to the risk of teratogenicity. Therefore, some febrile diseases may take a more severe course in pregnancy leading to transplacental transmission of infectious agents and fetal risk.[59,60] Fevers in the mother must be treated as any other serious illness. The effects on pregnancy depend on the extent of temperature elevation, its duration, and the stage of fetal development when it occurs.

Mild exposures during the pre-implantation period and more severe exposures during embryonic and fetal development often result in miscarriage, premature labor, growth restriction, and stillbirth. Fever also causes a wide range of fetal structural and functional defects, with the central nervous system (CNS) being most at risk. While there is a greater incidence of neonatal morbidity and mortality with transmitted infections, not all maternal infections lead to transmission to the fetus, nor does transmission to the fetus lead to disease or sequelae.[61,62,63]

Pathogens commonly causing fever in pregnancy

Viruses:

1. Varicella-zoster virus

2. Coxsackievirus

3. Human parvovirus B19

4. Rubella

5. Cytomegalovirus

6. HIV

7. HSV

Bacteria:

1. Listeria

2. Syphilis

3. Borrelia

4. Group B streptococcus

5. Coliforms

Protozoa:

1. Toxoplasmosis

2. Malaria

The common infections during pregnancy

1. Urinary tract infection

2. Respiratory tract infection

3. Vaginal infections

4. Typhoid and paratyphoid fever

5. Malaria

6. Dengue

7. Tuberculosis

8.Chicken pox

Urinary Tract Infections in Pregnancy:-

Urinary tract infection (UTI) is a common clinical problem, which can involve the urethra, bladder, and kidney.[64] UTI affects all age groups, but women are more susceptible than men, due to short urethra, absence of prostatic secretion, pregnancy and easy contamination of the urinary tract with faecal flora.[65] Additionally, the physiological increase in plasma volume during pregnancy decreases urine concentration and up to 70% pregnant women develop glucosuria, which encourages bacterial growth in the urine.[66] Urogenital symptoms occur in almost all women during pregnancy. The frequency of overactive bladder symptoms starts from early pregnancy while urinary incontinence symptoms increase with gestational age although majority remain tolerable.[67] Lower urinary tract symptoms are almost universal in antenatal period and are transient. These symptoms may reflect pregnancy induced changes in urinary bladder and urethra or may be manifestation of cystitis and urethritis. Frequency, nocturia and stress incontinences are the most common complaints.

Increased incidence of urge incontinence has also been quoted.[68] Urinary incontinence in pregnancy is ascribed to detrusor instability which improves after delivery.[69,70] Infections, particularly in pregnancy and in the elderly, can be asymptomatic, but symptomatic bacteriuria is associated with an increased risk of intrauterine growth retardation and low birth weight.[71] Furthermore, untreated asymptomatic bacteriuria leads to the development of cystitis in approximately 30% of cases, and can lead to the development of pyelonephritis in about 50% cases.[72] In

addition acute pyelonephritis has been associated with anaemia. It is therefore essential to screen for urinary tract infection in pregnancy so that timely treatment could be offered.[73,74]

Sheikh et al demonstrated that, past history of urological problems was associated with an increased incidence of UTI in pregnancy.[75] The organisms that causes UTIs during pregnancy are the same as those found in non pregnant patients. Escherichia coli accounts for 80-90% of infections.[76]

Urinary tract infections (UTI), which are caused by the presence and growth of microorganisms in the urinary tract, are perhaps the single commonest bacterial infections of mankind[77] and in pregnancy, it may involve the lower urinary tract or the bladder.[78] UTI has been reported among 20% of the pregnant women and it is the most common cause of admission in obstetrical wards.[79]

Anatomically UTI can be classified into lower urinary tract infection involving the bladder and urethra and upper urinary tract infection involving the kidney, pelvis, and ureter. The majority of the UTI occur due to ascending infection.[80,81] Three common clinical manifestations of UTIs in pregnancy are: asymptomatic bacteriuria, acute cystitis and acute pyelonephritis.[82]

UTI is defined as the presence of at least 100,000 organisms per milliliter of urine in an asymptomatic patient, or as more than 100 organisms/mL of urine with accompanying pyuria (>5 WBCs/mL) in a symptomatic patient. Particularly in asymptomatic patients, a diagnosis of UTI should be supported by a positive culture for a uropathogen.[83] Untreated asymptomatic bacteriuria is a risk factor for acute cystitis (40%) and pyelonephritis (25-30%) in pregnancy. These cases account for 70% of all cases of symptomatic UTI among unscreened pregnant women. Symptomatic and asymptomatic bacteriuria have been reported among 17.9% and 13.0% pregnant women, respectively.[84]

Pregnancy increases the risk of UTI. At around 6th week of pregnancy, due to the physiological changes of pregnancy the ureters begin to dilate. This is also known as "hydronephrosis of pregnancy", which peaks at 22-26 weeks and continues to persist until delivery. Both progesterone and estrogens levels increase during pregnancy and these will lead to decreased ureteral and bladder tone. Increased plasma volume during pregnancy leads to decrease urine concentration and increased bladder volume. The combination of all these factors lead to urinary stasis and uretero-vesical reflux. Additionally, the apparent reduction in immunity of pregnant women appears to encourage the growth of both commensal and non-commensal microorganisms.[85] The physiological increase in plasma volume during pregnancy decreases urine concentration and up to 70% pregnant women develop glucosuria, which encourages bacterial growth in the urine,[86.]

Female gender itself is a risk factor because of short urethra, its proximity to vagina and anus and inability of women to empty their bladder completely. High incidence is seen in lower socioeconomic group.[87] Sexual activity and certain contraceptive methods are also said to increase the risk.[88] The anatomical relationship of female's urethra and the vagina makes it liable to trauma during sexual intercourse as well as bacteria been massaged up the urethra into the bladder during pregnancy/child birth.[89,90] Abnormalities of urinary tract or stones, diabetes mellitus, immunosupression and past history of UTI tend to increase the risk.[91,92]

Urinary tract infection during pregnancy contributes significantly to maternal and perinatal morbidity. Abortion, small birth size, maternal anemia, hypertension, preterm labour, phlebitis, thrombosis and chronic pyelonephritis are related to urinary tract infection during pregnancy.[93,94] E. coli remains the predominant organism implicated in urinary tract infection in pregnancy, though recent reports show changes in pattern of the infection. Recent studies in Nigeria show an increasing involvement of Klebsiella Spp. Staphylococcus aureus, Proteus spp., and Pseudomonas spp in urinary tract infection in pregnancy.[95]

Acute Pyelonephritis:-

The risk of acute pyelonephritis (APN) increases during pregnancy owing to anatomic and physiologic changes in the urinary tract.[96] Although the incidence of bacteriuria in pregnant women was estimated to be similar or slightly higher than that in nonpregnant women (2% or 10–13%) in previous studies,[97,98,99] a much higher incidence of symptomatic urinary tract infection (UTI) and progression to APN along with bacteriuria (up to 40%) has been reported.[100] APN is believed to be associated with urinary tract stasis due to hormonal changes during pregnancy, especially increased progesterone level, and is also partly caused by mechanical obstruction as pregnancy progresses. APN is the most frequent bacterial infection during pregnancy and one of the most common causes of antepartum hospitalization besides obstetric indications.[101,102,103]

Given that APN during pregnancy is associated with an increased risk of maternal and fetal morbidity, such as preterm birth, preeclampsia, stillbirth, anemia, sepsis, renal insufficiency, and acute respiratory distress syndrome,[104,105] while some resistant organisms are detected in routine urine culture,[106] the risk factors for APN should be closely monitored and managed during antenatal care. However, although some risk factors have been reported in previous studies, including multiparity, diabetes mellitus, urinary tract stones or malformation, and low socioeconomic status, it is not yet clear what increases the risk of APN during pregnancy.[107]

Although, asymptomatic bacteriuria is the most common form, acute pyelonephritis is the most prevalent medical complication of gestation, which occurs in 1% - 2% of all pregnancies and may result in significant fetal and maternal morbidity and mortality.[108] On the other hand, pyelonephritis is the leading cause of septic shock in pregnancy.[109] Normal perineal flora bacteria such as E. coli, Proteus and Klebsiella are the most important organisms leading to pyelonephritis during gestation. Infection usually ascends through bladder easily during gestation, since higher progesterone level causes additional urine stasis due to increased vesicoureteral reflux, decreased urethral peristaltism and bladder hypotonia.[111,112]

Asymptomatic UTI incidence does not increase in pregnant women in comparison to non-pregnants, but due to mentioned anatomical and physiological changes, the risk of clinical diseases increases in gestation and 30% - 40% of untreated cases develop symptomatic infection such as pyelonephritis.[113] However, bacteriuria eradication by antibacterial agents decreases symptomatic infection occurrence.

Regarding increased risk factors such as nulliparity, younger age, nephrolithiasis, anomalies, higher urinary pH and glucosuria, pyelonephritis is most prevalent during the second trimester.[114,115] Since pregnant women uterus develops pressure on the right urethra and causes some degrees of urinary stasis, pyelonephritis occurs mostly in the right kidney. Although, 25% of cases are two-sided, isolated left Kidney pyelonephritis can be associated with anomalies or anatomic urinary tract disturbances.

Abrupt chills and fever is common in disease onset and continues with persistent pain in one or both flunks. Nausea and vomiting are probable in patients and one-sided or bilateral costovertebral angle tenderness is present in 85% of patients. Prophylactic nitrofurantoin therapy until the end of gestation is important in pregnant women, since recurrence of pyelonephritis arises in 6% - 8% of patients. To diminish its complications, all patients with pyelonephritis should be admitted and rehydrated, empirical antibacterial regimen and vital signs monitoring.

Pneumonia:-

Concern that pneumonia occurring in a pregnant patient may be more frequent, exhibit atypical features, run a more severe course, or be more difficult to treat than in a non-pregnant patient is not unusual. Underlying these concerns are the recognised physiological and immunological changes that occur during pregnancy which may compromise the mother's ability to respond to an infection. Added to this are concerns for the health of the fetus.

Alterations in cellular immunity have been widely reported and are aimed primarily at protecting the fetus from the mother. These changes include decreased lymphocyte proliferative response, especially in the second and third trimesters, decreased natural killer cell activity, changes in T cell populations with a decrease in numbers of circulating helper T cells, reduced lymphocyte cytotoxic activity, and production by the trophoblast of substances that could block maternal recognition of fetal major histocompatibility antigens.[117,118]

In addition, hormones prevalent during pregnancy—including progesterone, human chorionic gonadotropin, alpha-fetoprotein and cortisol—may inhibit cell mediated immune function.[119] These changes could theoretically increase the risk from infection, particularly by viral and fungal pathogens.

Anatomically, the enlarging uterus causes elevation of the diaphragm by up to 4 cm and splaying of the thoracic cage. A 2.1 cm increase in the transverse diameter of the chest and a 5–7 cm increase in the circumference of the thoracic cage has been reported.[120] These changes may decrease the mother's ability to clear secretions. The decrease in functional residual capacity, increase in oxygen consumption, and increase in lung water that occur during pregnancy add to the vulnerability of the lung to injury from infection. Obstetric and anaesthetic interventions, including endotracheal intubation, pose further risks, not least from aspiration pneumonia.[121]

A true estimate of the incidence of pneumonia during pregnancy is difficult to obtain. The few studies of pneumonia during pregnancy are mostly retrospective and include few cases. Based on these studies, a marked variation in incidence over the years is evident. A very high incidence was reported in the years before 1965 ranging from 6.3 per 1000 deliveries to 8.5 per 1000 deliveries.[122,123] This had decreased in the 1970s and early 1980s to 0.44–0.78 per 1000 deliveries, presumably due to the introduction of antibiotics and improvements in obstetric

care.[124] More recently, an incidence of 1.2–2.7 per 1000 deliveries has been reported.[125] It has been proposed that this increase in incidence is a reflection of the higher proportion of pregnant women with chronic medical conditions. However, patients in these study cohorts are not directly comparable and the varying proportions with documented illicit drug use (14–52%) and HIV seropositivity (4–27%) may not simply be the result of time trends.[126]

Herpes Simplex virus:

Genital herpes simplex virus (HSV-2) is the most common sexually transmitted infection among the adult female population of the United States. The Centers for Disease Control and Prevention (CDC) estimates that about 1 out of 6 people overall (1 out of 5 women aged 14–49 and 1 out of 9 men aged 14–49) in the United States has genital HSV, with almost 800,000 new cases identified each year.[127]

HSV-1 (HSV-1) and 2 (HSV-2) are part of a large family of DNA viruses of which eight are known to be infectious in humans. HSV-1 and 2 are transmitted across epithelial mucosal cells as well as through skin interruptions and migrate to nerve tissues where they persist latent. HSV-1 predominates in orofacial lesions and typically is found in the trigeminal ganglia, while HSV-2 is most commonly found in the lumbosacral ganglia. Both HSV-1 and HSV-2 can cause genital lesions and shedding.

According to The National Health and Nutrition Examination Surveys (NHANES), there is an overall decrease in the seroprevalence of HSV-1 by 7% and of HSV-2 by 19%. NHANES indicates that the rates of HSV-2 are higher among women (23.1%) than men (11.2%) in the general population. Factors that affect a woman's risk of infection before pregnancy include ethnicity, poverty, cocaine abuse, earlier onset of sexual activity, number of lifetime sexual partners, sexual behavior, and the presence of bacterial vaginosis.[128]

The overall seroprevalence of HSV among pregnant women is 72%.[129] This represents any exposure to either HSV-1 or HSV-2 that resulted in infection and antibody formation. During pregnancy, HSV infection has been associated with spontaneous abortion, intrauterine growth restriction, preterm labor, and congenital and neonatal herpes infections. However, the clinical management revolves around decreasing vertical transmission to the fetus, thereby decreasing the risk of neonatal herpes infection.[130]

The presence of antibodies to both HSV-1 and HSV-2 at the onset of pregnancy (prior seroconversion) has the least risk of perinatal transmission. In contrast, primary or first genital HSV infection late in pregnancy carries a 30% to 50% risk of neonatal infection, while early pregnancy infection carries a risk of less than 1%, [131]. If primary HSV infection occurs during late pregnancy antibodies are not developed in time to suppress viral replication and shedding before labor. Transplacental or ascending transmembrane transmission of HSV from mother to fetus during pregnancy is uncommon; 80–90% of perinatal transmission occurs during

labor and delivery. However, neonatal infection with HSV can also occur in the setting of recurrent herpes. Symptom recurrence producing viral shedding at the onset of labor is associated with up to a 3% risk of neonatal herpes; both young age and recent infection are associated with increased viral shedding. Interestingly, asymptomatic viral shedding in recurrent disease at term has not been associated with neonatal disease.[132,133]

Neonatal herpes infection is classified into three categories: localized skin, eye, and mouth (SEM); central nervous system (CNS) with or without SEM; and disseminated disease (which carries a mortality rate in excess of 80%, if untreated.)[134]. Infected newborns can exhibit significant neurologic deficits, blindness, seizures, and learning disabilities. Suppressive antiviral therapy during the last month of pregnancy reduces the likelihood of asymptomatic viral shedding, clinical HSV recurrence, and caesarean delivery for recurrent lesions.[135] When lesions or prodromal symptoms are present at the onset of labor, caesarean section is recommended to minimize the risk of viral exposure to the infant, even if suppressive therapy has been used.[136]

Among women with asymptomatic HSV in labor, invasive procedures such as amniotomy, the use of fetal scalp electrodes and operative vaginal delivery, should be avoided. This decreases fetal exposure to vaginal secretions potentially containing the virus. Active management should be considered in these women when

membranes rupture before the onset of labor. When there is preterm premature rupture of the membranes (PPROM), the risks of prematurity must be weighed against the risks of HSV transmission. Each case will be dependent on the gestational age and clinical picture. There is no consensus on the best timing of delivery for women with PPROM and a history of HSV.[137]

Varicella Zoster Virus:

Varicella, also known as chickenpox, is the acute primary disease of varicella zoster virus. It is a common, highly contagious, self-limiting disease seen mainly during childhood. It is transmitted by respiratory droplets or close contact and causes a widespread maculopapular to vesicular rash that starts on the face and trunk and then moves to the extremities. The virus incubates for 15 days and is communicable 2 days before the onset of the rash until all the lesions have crusted or disappear. After an initial episode with varicella zoster leading to chickenpox the virus may persist latent in the dorsal root ganglia for years. Reactivation results in herpes zoster, which is more common in adults.[138]

The incidence of varicella in pregnancy is 0.7/1000. Because varicella is mainly a disease of childhood in the United States, most women are immune before they become pregnant. However, primary varicella infection during pregnancy is associated with significant maternal and fetal morbidity and mortality. While the childhood illness is self-limiting and mild, if varicella pneumonia develops during

pregnancy, it can run a more fulminant course. Approximately 10–20% of pregnant women who are infected with varicella will develop pneumonia, which carries a mortality rate of up to 40%.[139]

Fetal morbidity and mortality is related to the development of congenital varicella syndrome. This syndrome is characterized by limb hypoplasia, microcephaly, hydrocephaly, cataracts, intrauterine growth restriction, and mental retardation28. The risk of congenital varicella syndrome ranges from 0.4–2% with maternal varicella infection during the first 20 weeks of gestation. Development of this syndrome is thought to be a result of reactivation of the varicella virus in utero, as opposed to primary infection of the fetus.[140]

Herpes zoster in pregnancy is much less common than varicella, occurring in 1/10,000 pregnancies, or 0.1. The risk of the congenital varicella syndrome is negligible, because antibodies in the maternal blood prevent the virus from crossing the placenta and infecting the fetus. In 1994, Enders and colleagues showed no clinical evidence of infection in infants born to 366 women with herpes zoster in pregnancy. Neonatal infection may occur in 10% to 20% of neonates whose mothers became acutely infected from 5 days before delivery to 2 days after the delivery. It results from hematogenous dissemination of the virus across the placenta in the absence of maternal antibodies. Infants become symptomatic 5–10 days

postpartum. The clinical picture may vary from skin lesions to systemic illness, which has a mortality rate of about 30%.[141]

Cytomegalovirus:

Cytomegalovirus (CMV) is a ubiquitous virus with variable clinical manifestations. Seroprevalence increases with age, and differs based on geographic area and socioeconomic status. CMV infects 60% of women of childbearing age in developed countries and 90% in developing countries. The resulting seropositivity (maternal antibody status to CMV) is the most important factor in combatting congenital CMV infection. The remaining 40% of women in developed countries are susceptible to infection; and if this infection occurs during pregnancy, it can have detrimental effects on the pregnancy.[3]

Person-to-person transmission occurs by contact with infected nasopharyngeal secretions, urine, saliva, semen, cervical and vaginal secretions, breast milk, tissue, or blood. Primary maternal infection occurs in 1% to 4% of susceptible women and reactivation may occur in approximately 10% of seropositive women. Maternal infection with CMV during the antepartum period goes undetected the majority of the time, but can manifest as a mild febrile illness with nonspecific symptoms such as fatigue, myalgias, rhinitis, pharyingitis, and headache. Furthermore, pregnancy does not appear to affect the clinical severity of the infection.

Vertical transmission (from the mother to the fetus or newborn) occurs most commonly after maternal primary infection, usually by the following mechanisms: transplacental after the virus infects the placenta, intrapartum via ingestion or aspiration of cervicovaginal secretions during delivery, postpartum via breastfeeding, and ascending from the maternal genital tract antepartum

In terms of fetal effects of maternal infection, CMV is the most common congenital viral infection, with a birth prevalence of about 0.5% (range 0.2–2.5%).[142] CMV primarily affects the ventricle, the organ of Corti and the neurons of the eighth cranial nerve, which explains why it is the leading cause of congenital hearing loss. Furthermore, human neuronal cells are able to be infected in-vitro with CMV, which may explain the central nervous system effects during fetal development.[143]

Most newborns of women with primary CMV infection and almost all newborns of women with non-primary infection in pregnancy are initially asymptomatic. 10 to 15 percent of these initially asymptomatic newborns develop neurodevelopmental damage within the first three years of life.[144] Approximately 5 to 20 percent of newborns of mothers with primary CMV infection will have symptoms at birth. The mortality rate of these newborns is about 5 percent. Five to 15 percent of the asymptomatic newborns will develop sequelae later in life.[145]

Rubella:

The name rubella is derived from Latin, meaning “little red” because it was initially thought to be a variant of measles or scarlet fever. However, in the mid 1800s a German physician distinguished rubella as a distinct clinical entity, hence it being commonly known as German measles. The rubella virus, an enveloped RNA virus, is classified as a togavirus. It is transmitted through respiratory droplets and is primarily a mild disease in children. In adults, rubella is a self-limited disease characterized by rash. The rash characteristically begins on the face and spreads to the trunk and extremities. The incubation period is 12 to 23 days. The infectious period is from 7 days before to 7 days after rash onset. Importantly, rubella can be asymptomatic in 25% to 50% of the cases. When maternal infection occurs in the first trimester, fetal infection rates are up to 50%, dropping to <1% after 12 weeks. Peripartum maternal infection does not seem to increase the risk of CRS. Diagnosis of primary maternal infection should be made by serologic tests. Diagnosis of fetal infection includes detection of fetal serum IgM (but not until after 22–24 weeks of gestation) or viral culture of the amniotic fluid.[146]

Pregnancy outcomes as a result of maternal rubella infection include spontaneous abortion, fetal infection, stillbirths or fetal growth restriction and the congenital rubella syndrome (CRS). The CRS, represents the neonatal manifestations of antenatal infection with the rubella virus. The risk of CRS varies according to the gestational age at which maternal infection occurs. Therefore, counseling regarding

the fetal risk and management must be individualized. Two proposed mechanisms for rubella cytopathology includes virus-induced inhibition of cell division and direct cytopathic effects.[147]

Hepatitis:

Acute viral hepatitis is the most common cause of jaundice in pregnancy. The course of most viral infections is not affected by pregnancy.

Hepatitis A

Hepatitis A virus (HAV) infection is the second most common form of viral hepatitis in the United States. It is an RNA virus that is transmitted by the fecal oral route.[148] Infections occur early in life in areas where sanitation is poor and living conditions are crowded. The incidence of acute HAV infection in pregnancy is approximately 1:1000 women. Vertical transmission of HAV during the pregnancy or puerperium is rare.[149] Pregnancy should not impact a physician's management of HAV infection or vice-versa.

Hepatitis B

Hepatitis B virus (HBV) is the most common form of chronic hepatitis around the world. Chronic carriers can continue to transmit the disease for many years before becoming symptomatic. Infection occurs very often in early childhood when it is asymptomatic and then leads to the chronic carrier state. Chronic HBV infection

leads to increased risk for chronic hepatic insufficiency, cirrhosis and hepatocellular carcinoma. The age group most likely to be affected worldwide is the newborn population, particularly in areas with high prevalence of disease and lack of diagnosis of infected women whose infants are at risk of becoming chronic carriers. In the other hand, the primary causes of transmission in the young adult population where perinatal screening and adequate newborn prophylaxis exist are the exposure to contaminated blood products, body fluids, or sexual contact.[150]

A high maternal viral load appears to be the most important risk factor for perinatal transmission. Transplacental transmission and transmission due to obstetrical procedures are less frequent causes, while breastfeeding does not appear to pose a substantial risk. Delivery decisions should be made in the context of the usual obstetric indications.

Management options are available to decrease the risk of perinatal transmission. Universal screening of all pregnant women allows for identification of Hepatitis B surface antigen positive women. Women found to be positive should also be screened for HIV and other forms of hepatitis such as hepatitis A and hepatitis C. Monitoring of liver biochemical tests and viral load help guide management and extent of disease. All infants of mothers with HBV should receive passive-active immunization with hepatitis B IgG and the hepatitis B vaccine within 12 hours of delivery.

Hepatitis C

The hepatitis C virus (HCV) is a major cause of chronic hepatitis, cirrhosis and hepatocellular carcinoma around the world. HCV infection has a slow onset with symptoms in about 25% of patients. Approximately 40% of patients infected with HCV recover completely and the rest become chronic carriers. Twenty percent of the carriers develop cirrhosis and of those, up to 20% develop liver cancer.[151]

Approximately 60% of all new infections occur via intravenous (IV) drug use, 10–20% by sexual transmission, less than 6% by blood transfusion, and the remainder includes occupational and unknown exposures.[152] Only 25% of pregnant women report receiving blood products or IV drug use when HCV infection is diagnosed. Concurrent alcoholism, IV drug use (38%), and coexisting HIV infection (33%) are important associated risk factors.[153] The decline observed in the incidence and prevalence of HCV is attributed to needle exchange programs among intravenous drug users and improved blood donor screening. This has led to a relative increase in the importance of accidental needle sticks, and sexual and perinatal transmission in the incidence of HCV infections.

The World Health Organization (WHO) estimates that about 3% of the world's population has been infected with HCV and that there are more than 170 million chronic carriers who are at risk of developing liver cirrhosis and/or liver cancer. The highest prevalence is in Egypt (18–22%) and the lowest in Sweden (0.003%). As many as 2–4 million persons may be chronically infected in the United States, 5–10 million in Europe, and about 12 million in India. Of these, about 25% are symptomatic, but 60–80% may progress to chronic liver disease, and 20% of these develop cirrhosis.[154]

Women chronically infected with hepatitis C may have uneventful pregnancies; though vertical transmission is the greatest concern. In the United States and other industrialized nations, because of vaccination programs against hepatitis B, hepatitis C virus (HCV) has become the primary cause of chronic viral hepatitis in children, with vertical transmission becoming the leading source of infection.[155] The mechanism underlying vertical transmission is poorly understood. Overall the transmission rate appears to be less than 2 percent, when adjusted for certain clinical variables. For example, HIV co-infection results in a 19 percent transmission rate.[156] During pregnancy, pregnant women should be seen regularly by a gastroenterologist to monitor liver biochemical tests and viral load. During labor, prolonged rupture of membranes should be avoided, as well as invasive obstetric procedures. Caesarean delivery should be reserved for the usual obstetrical indications. Breastfeeding is not contraindicated.[157]

Influenza:-

Pregnant women are at high risk for severe complications of influenza during seasonal influenza periods and pandemics.[158] In addition, some studies suggest an increased risk for adverse outcomes among infants born to mothers infected with influenza during pregnancy.[159]

Pregnancy has been the highest risk factor for increased illness and death for both pandemic and seasonal influenza. Although appropriate non-pregnant control groups were generally not available, mortality rates among pregnant women in the pandemics of 1918 and 1957 appeared to be abnormally high.[160] Among 1,350 reported cases of influenza among pregnant women during the pandemic of 1918, the proportion of deaths was reported to be 27%. Similarly, among a small case series of 86 pregnant women hospitalized in Chicago for influenza in 1918, 45% died.[161] Among pregnancy-associated deaths in Minnesota during the 1957 pandemic, influenza was the leading cause of death, accounting for nearly 20% of deaths associated with pregnancy during the pandemic period; half of women of reproductive age who died were pregnant.

Pregnant women have also been shown to be at increased risk for influenza complications during seasonal influenza periods.[162] In a large study of >4,300 women of reproductive age during 19 influenza seasons, pregnant women were compared with postpartum women (a group considered to be most similar to

pregnant women demographically and with regard to their health) and were found to be significantly more likely to be hospitalized for a cardiopulmonary event during the influenza season. The risk for hospitalization increased as pregnancy progressed, with women at term nearly 5 times more likely to be hospitalized than postpartum women.[163] Similarly, during 3 influenza seasons in the late 1970s, rates of medical visits for acute respiratory disease were more than twice as high among pregnant women than non-pregnant women. At particularly high risk during the influenza season are pregnant women with underlying medical conditions for which influenza vaccination is recommended, such as asthma. On the basis of these data pregnant women are considered a population for which special considerations for prevention and treatment for influenza have been made.[164]

Dengue:

Dengue is one of the most rapidly spreading vector borne viral disease in the world. There is an increasing incidence of dengue in adult population in South Asian, South- East Asian and Latin American countries in recent years. The dengue virus comprises four distinct serotypes (DEN 1-4) but DEN-3 is frequently associated with severe disease.[165] Majority of dengue patients have a mild self-limiting illness and a few progresses to a severe disease. As the incidence of dengue in adults increases, the incidence in pregnant women also increases.[166]

Intravenous rehydration is the treatment of choice, which can reduce the mortality in dengue to less than 1% of severe cases.Dengue poses a significant threat to pregnant women. In present study, we have studied the pattern of dengue in pregnancy and the impact of dengue on the natural course of pregnancy. The maternal and fetal outcomes were also analyzed.

The clinical severity of disease has a wide spectrum, and according to the World Health Organization (WHO) dengue classification scheme, there are four grades of dengue, ranging from uncomplicated dengue fever (DF) to dengue hemorrhagic fever (DHF) and devastating dengue shock syndrome (DSS).

DHF is currently defined by the following four WHO criteria:

- Fever or recent history of fever lasting 2-7 days.
- Any hemorrhagic manifestation.
- Thrombocytopenia (platelet count of <100,000/mm3).
- Evidence of increased vascular permeability.

With the recent resurgences of the disease, an increasing the number of people including the pregnant women are affected. Dengue infection in pregnancy carries the risk of hemorrhage for both the mother and the new-born. Literature search reveals an increased incidence of pre-term deliveries, low birth weight babies, preeclampsia and caesarean sections. Vertical transmission has also been noted.

Gehlot H et al did a study on dengue fever in pregnancy and its maternal and fetal prognosis to assess the clinical profile, maternal and fetal outcome of dengue fever during pregnancy. They found that Thrombocytopenia (platelet count <1.5lakh/mm3) was found in 22 (88%) patients out of which 6 (24%) of them had platelet count below 20,000 cells/mm3 and 3(12%) patients required platelet transfusion. Other complications observed were spontaneous abortions (4%); preterm birth (16%), oligohydramnios (8%) and antepartum hemorrhage (4%). One patient was admitted to Intensive Care Unit. Fetal distress and meconium stained amniotic fluid was observed in 16% and 12% patients respectively. Adverse fetal outcome was observed in form of low birth weight, prematurity. 8% of the babies required NICU admission and 4% were Intra Uterine Fetal Death (IUFD). Maternal infection with the dengue virus during antenatal period represents a real risk of premature birth. Early onset or late onset in pregnancy appeared to have a bad prognosis. A high index of clinical suspicion is essential in any pregnant woman with fever during the epidemic. The treatment of dengue in pregnancy is mainly conservative as in non-pregnant adults. In case of high risk cases early referral to well-equipped health centres where technical, transfusion and intensive care facilities are available may prove lifesaving.[167]

Typhoid fever:

Typhoid fever is caused by the organism Salmonella enterica subspecies enterica serovar Typhi (Salmonella Typhi); a systemic infection transmitted predominantly through water or food contaminated by human feces.[168,169] Typhoid

fever presents clinically across a spectrum of severity with a range of symptoms and signs including fever, abdominal pain, nausea, and vomiting, that make differentiating it from other febrile and gastrointestinal illnesses challenging.[170] The 'gold standard' diagnostic method for typhoid fever is the culture of blood, bone marrow, or another normally sterile site. However, clinical microbiology services are not widely available in endemic areas and culture-based diagnosis has incom- plete sensitivity. [171] Additionally, delays in diagnosis and treatment occur as a result of barriers to care, such as difficulty accessing tertiary facilities because of delayed referral, distance, and the cost of healthcare.[172]

Timely and accurate diagnosis and treatment of typhoid fever in the community is needed to avert complications requiring hospitalization, and death. Typhoid complications include typhoid intestinal perforation (TIP), gastrointestinal hemorrhage, hepatitis, cholecystitis, myocarditis, shock, encephalopathy, pneumonia, and anemia. TIP and gastrointestinal hemorrhage are serious complications that are often fatal, even if managed surgically.[173]

Sulaiman K et al did a study on enteric fever and pregnancy and found that Cases of typhoid fever were seen throughout the year with no seasonal pattern in either group of women with culture confirmed disease. The average age in both groups was approximately 25 years. Overall, 71% of the 181 women with typhoid fever were infected with S. typhi. However, the proportion of pregnant or postpartum women with S. typhi (79%) was significantly higher than non-pregnant women

(64%). While most cases of typhoid fever in pregnancy occurred in the second and third trimesters (75%), only two cases occurred in the postpartum host.

Only two pregnant women with typhoid fever had any comorbid illness, one had a history of malaria in the last three months while the other had had acute viral hepatitis in the preceding six months. By comparison, 26 (27%) of the non pregnant women, a significantly higher number, presented with co-morbid illnesses. These included diabetes mellitus, hepatitis, asthma, pneumonia, malaria, thalassemia minor and tuberculosis. Prior use of antibiotics was observed in a total of 56% of all women with typhoid fever, and did not differ significantly between the pregnant and non-pregnant groups. The duration of fever prior to presentation was also similar in both groups. The symptoms of typhoid disease were assessed as upper gastrointestinal (nausea/vomiting), lower gastrointestinal (diarrhea/constipation), and cough. Pregnant women were significantly more likely to present with cough (36%), than non-pregnant women (21%), but were less likely to report upper gastrointestinal symptoms. pregnancy is an important risk factor for acquiring typhoid in adults and appears to modify disease expression, the disease itself may not affect pregnancy or its outcome in any significant way. However, these findings should be verified prospectively given the inherent biases associated with our retrospective study at a tertiary care institution where only the sicker pregnant women would be expected to seek care.[174]

Chorioamnionitis:

Chorioamnionitis or intraamniotic infection is an acute inflammation of the membranes and chorion of the placenta, typically due to ascending polymicrobial bacterial infection in the setting of membrane rupture. Chorioamnionitis can occur with intact membranes, and this appears to be especially common for the very small fastidious genital mycoplasmas such as Ureaplasma species and Mycoplasma hominis, found in the lower genital tract of over 70% of women.[175] Only rarely is hematogeneous spread implicated in chorioamnionitis, as occurs with Listeria monocytogenes.[176] When characteristic clinical signs are present, the condition is referred to as clinical chorioamnionitis or clinical intraamniotic infection.

Although there is significant overlap between clinical and histologic chorioamnionitis, the latter is a more common diagnosis based on pathologic findings on microscopic examination of the placenta that encompasses clinically unapparent (sub-clinical) chorioamnionitis as well as clinical chorioamnionitis. Funisitis, also a histopathologic diagnosis, is the extension of infection or inflammation to the umbilical cord. Overall the definition of chorioamnionitis varies according to key diagnostic criteria, which can be clinical (presence of typical clinical findings), microbiologic (culture of microbes from appropriately collected amniotic fluid or chorioamnion) or histopathologic (microscopic evidence of infection or inflammation on examination of the placenta or chorioamnionic specimens).

Chorioamnionitis leads to a 2 to 3-fold increased risk for cesarean delivery and 2 to 4-fold increase in endomyometritis, wound infection, pelvic abscess, bacteremia and postpartum hemorrhage.[177] The increase in postpartum hemorrhage appears to be due to dysfunctional uterine muscle contractions as a result of inflammation. Ten percent of women with chorioamnionitis have positive blood cultures (bacteremia) most commonly involving GBS and E. coli. Fortunately, however, septic shock, disseminated intravascular coagululation, adult respiratory distress syndrome and maternal death are only rarely encountered.[178]

Fetal complications—Fetal exposure to infection may lead to fetal death, neonatal sepsis and numerous other postnatal complications. The fetal response to infection – termed the Fetal Inflammatory Response Syndrome (FIRS) – may cause or aggravate some of these complications. FIRS is the fetal counterpart of the systemic inflammatory response syndrome (SIRS). Since clinical parameters analogous to those defining SIRS are difficult to ascertain in the fetus, FIRS was originally defined by elevation of cord blood IL-6 in the setting of preterm labor and PPROM[179] but can also occur in term gestations. The histopathologic hallmarks of FIRS are funisitis and chorionic vasculitis.[180] FIRS is now recognized to represent the fetal immune response to infection or injury mediated by the release of cytokines and chemokines such as interleukins, TNF-alpha, Creactive protein, and matrix metalloproteinases. FIRS has also been linked to preterm labor culminating in perinatal death and is associated, particularly among preterm neonates, with multi-organ injury, including chronic lung disease, periventricular, leucomalacia and

cerebral palsy, Although FIRS may occur in the setting of noninfectious inflammation, its magnitude tends to be significantly more robust with documented infection Although somewhat controversial, fetal exposure to genital mycoplasmas (U. urealyticum and M. hominis) has been associated with a fetal and neonatal systemic inflammatory response syndrome, pneumonia and BPD.[181,182]

Neonatal and long-term complications are the neonate exposed to intrauterine infection and inflammation may show adverse effects at or shortly after birth. Adverse outcomes may include perinatal death, asphyxia, early onset neonatal sepsis, septic shock, pneumonia, intraventricular hemorrhage (IVH), cerebral white matter damage, and longterm disability including cerebral palsy. In one study of term infants, neonatal pneumonia, sepsis and perinatal death did not occur in the absence of chorioamnionitis but occurred, respectively, in 4%, 8% and 2% of term deliveries associated with chorioamnionitis. In this study, respiratory distress occurred in 2% of term infants in absence of chorioamnionitis and 20% when chorioamnionitis was present. Preterm infants have an even higher rate of complications of chorioamnionitis than term infants, including perinatal death (25 vs. 6% preterm vs. term), neonatal sepsis (28 vs. 6 %), pneumonia (20 vs. 3%), grades 3 or 4 IVH (24 vs. 8 %) and respiratory distress (62 vs. 35%).[183] Overall, chorioamnionitis is associated with up to 40% of cases of early-onset neonatal sepsis. Chorioamnionitis is also well-established as a risk factor for long-term neurodevelopmental disability especially when it occurs prior to term. In term and near-term infants it is associated with a 4-fold increase in the frequency of cerebral palsy. [184,185]

MATERIALS AND METHODS

Study design:

This was a Hospital based Analytical cross sectional study

Study setting:

The present study was carried out in the Department of Obstetrics and Gynecology at Government Mohan kumaramangalam Medical College and Hospital, Salem.

Study period:

This study was conducted during the period of January 2020 to January 2021 for the period of 13 months.

Sample size:

For this study based on the feasibility, time constraints and due to informed consent. For this study 100 sample was recruited.

Study population:

Patients admitted in Antenatal ward & Labour ward with or without fever after the informed Oral and written consent in the local language with the following inclusion and exclusion criteria's.

Inclusion criteria–

- Pregnant mothers of 37wks completed gestation with fever for more than one day (Temperature more than 100 f orally).
- Patients giving informed consent.
- Sure of gestational age by dates or early 1st trimester USG.

Exclusion criteria–

- Patients not giving informed consent
- Gestational Age <37 weeks
- Covid positive mothers
- Any Chronic medical & surgical disorders

And the study participants were divided into two groups one with the exposure of fever and another group is Normal Ante natal mothers as 50-50 equally.

Study procedure:

In the group 1, Patients of 37weeks completed mothers with fever for more than one day and in the group 2 healthy AN mothers of 37weeks completed gestation were included. After getting consent from the patient ten ml of venous blood will be withdrawn for peripheral smear, blood culture and sensitivity, dengue Ig G and Ig M, total count, differential count, platelet count and haemoglobin. Urine sample will be taken for urine culture and sensitivity and urine routine.

The patients will be followed up during their stay in the hospital and till delivery. Fetal outcome will be studied by taking variables-low birth weight, intrauterine growth retardation, preterm delivery, neonatal sepsis and perinatal death. Maternal complications –post operative wound infection, post-partum haemorrhage, pneumonia, septicemia, jaundice, hypoglycemia and other complications of specific fever will be studied.

Data collection:

The Information was collected from the consenting patients using pre-validated questionnaire and interviews which was scheduled to take place during pregnancy while admitted in ward and by means of phone/mobile contacts after discharge. The details included questions concerning the events during the pregnancy including fever, age, period of gestation (the trimester of pregnancy during which fever diagnosis was recorded), occupation, socio-economic status, alcohol consumption, coffee intake, etc. Detailed questions was asked to all women regarding the incidence of fever during pregnancy, number of fever episodes, its duration, body temperature and gestational weeks in which fever was recorded.

Other Routine Investigations, fever investigations, General examination, Obstetric examination, Usg obstetrics & fetal Doppler study, Onset of labour, MODE of delivery, maternal morbidity and Mortality, perinatal follow up of newborn, APGAR, Morbidity and Mortality details were recorded.

Statistical Analysis:

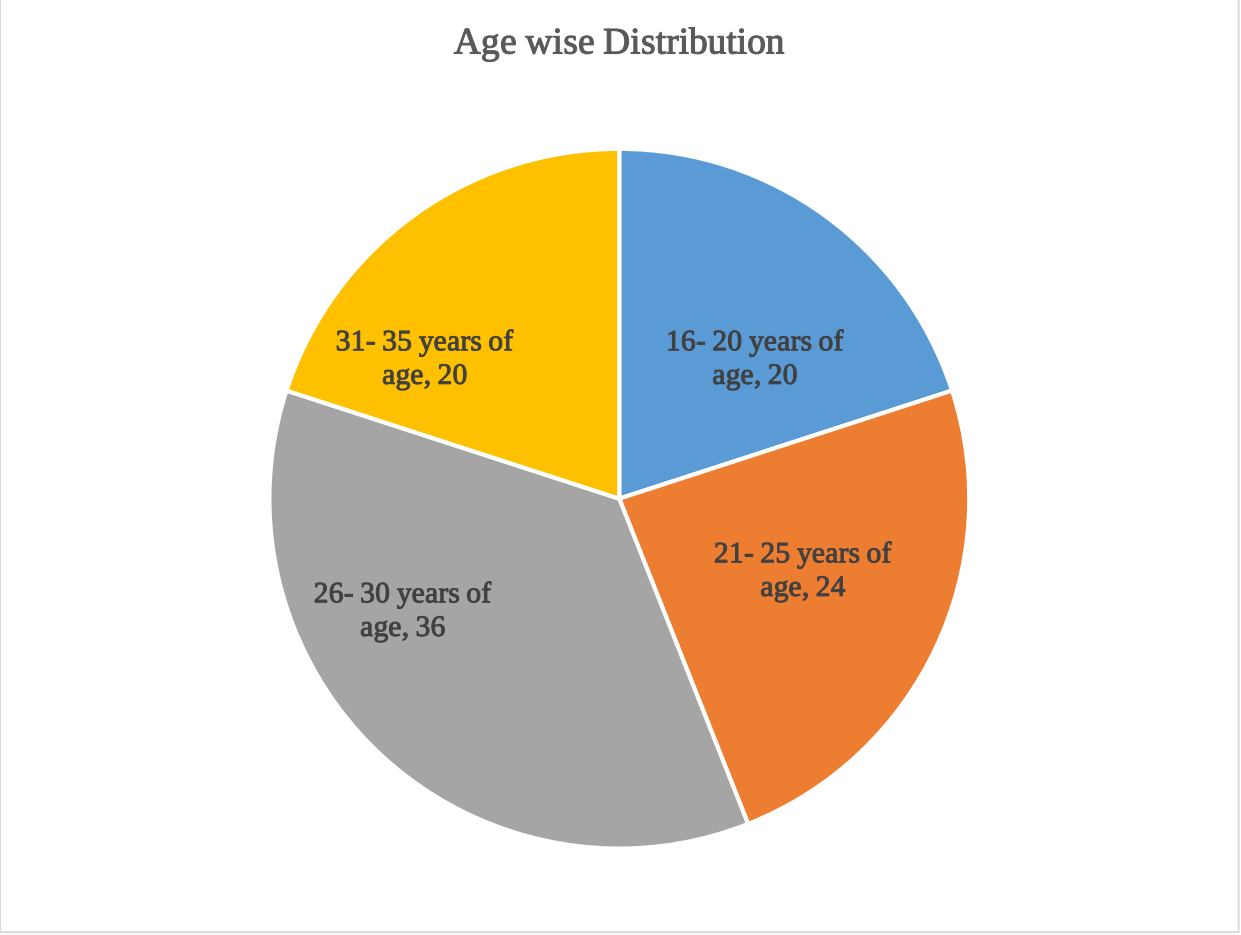
Data was entered in Ms excel and imported into SPSS 18 Software package. Descriptive statistics like mean, standard deviations and percentage proportions were used to describe baseline study participant parameters. Parametric tests were used to analyse the parametric data if it passed the tests of normality; if it failed then non-parametric tests were used for analysis. Chi-square test was used to analyse categorical data.

Ethics:

This study was conducted after obtaining the Intuitional Ethics and Research Committee approval.

RESULTS

Table 1: Age wise Distribution		
Age Group	Frequency	Percentage
16- 20 years of age	20	20%
21- 25 years of age	24	24%
26- 30 years of age	36	36%
31- 35 years of age	20	20%

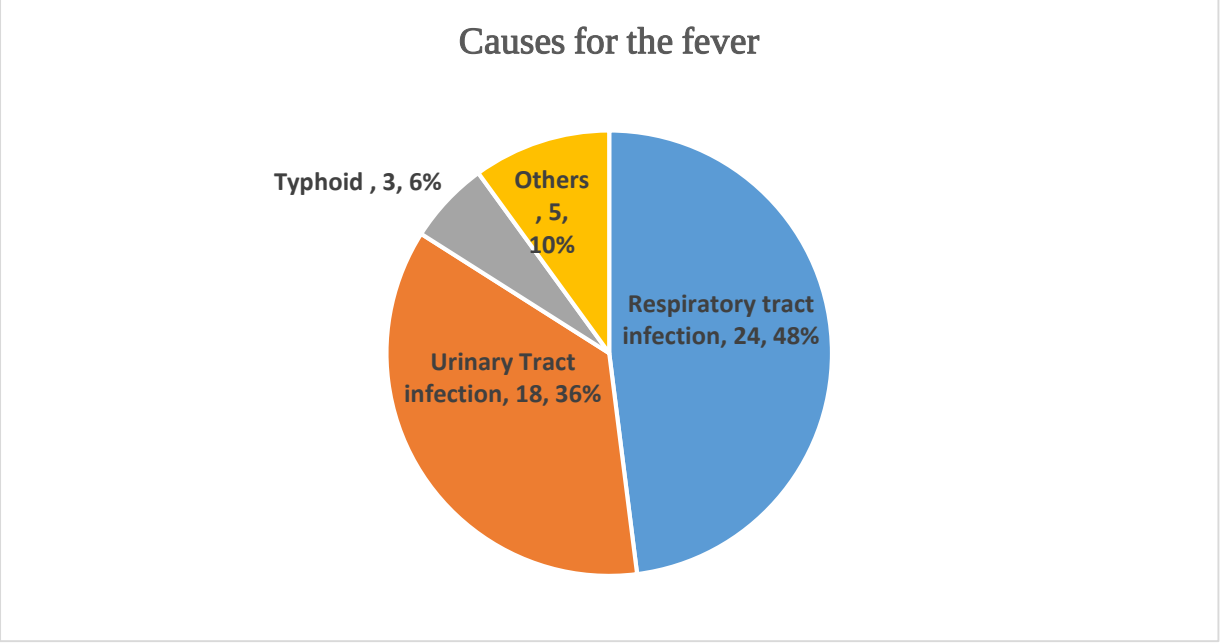


From the above table 1, the age wise distribution was shown out of 100 pregnant mothers in the study. About 20 were belongs to the age group of 16-20 years, 24 were belongs to the age group of 21-25 years, 36 were belongs to the age group of 26-30 years and 20 were belongs to the age group of 31-35 years. Majority of the cases represented from the 26-30 years age group.

Table 2 : Description of Fever Profile	
Fever cases	50
Mean duration of fever in days	2.22 ± 1.01
Mean Temperature	100.68± 0.74

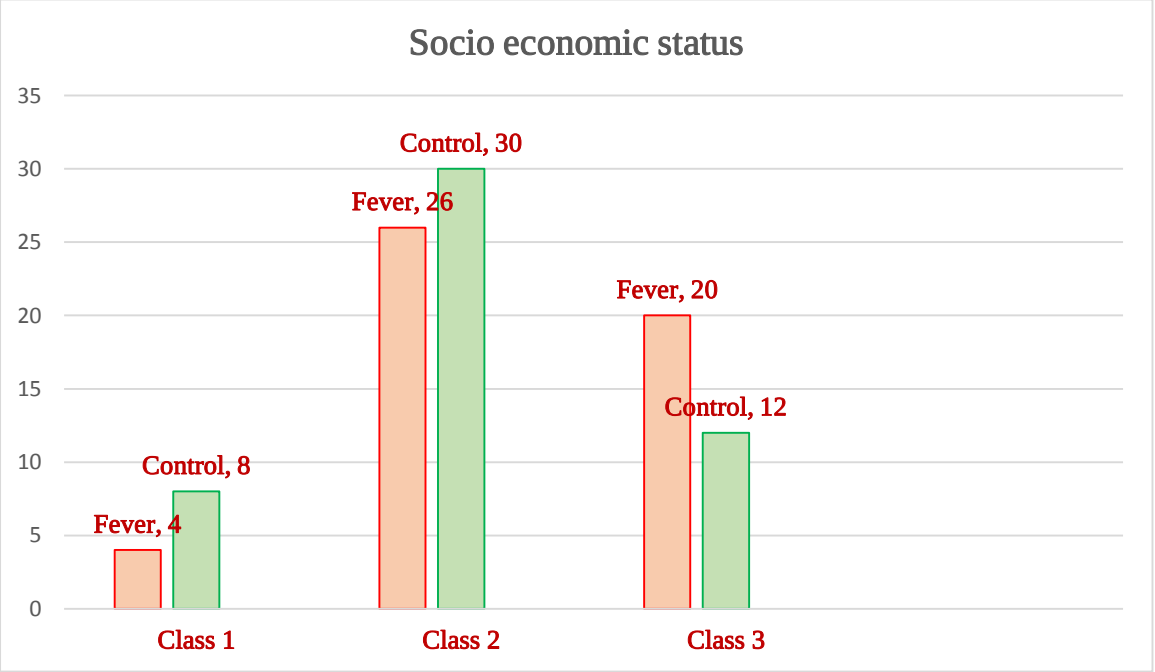
From the above table 2, out of 100 pregnant women. Equally 50 cases with fever and 50 cases without were involved in the study. Out of the 50 fever pregnant women. The mean duration of the fever in days was 2.22 ± 1.01. The mean temperature of the fever in Fahrenheit was 100.68± 0.74.

Table 3: Causes for the fever		
Causes	**Frequency**	**Percentages**
Respiratory tract infection	24	48%
Urinary Tract infection	18	36%
Typhoid	3	6%
Others	5	10%



From the above Table 3, Out of the 50 cases in our study the causes for the fever was, most of them had due to respiratory tract infections 24 (48%), followed by Urinary tract infection 18 (36%), Typhoid 3 (6%) and Others 5 (10%).

Table 4: Descriptive of Socio economic status			
Status	Fever (n=50)	Control (n=50)	Test of Significance
Class 1	4 (8%)	8 (16%)	Chi Square = 3.619 Degrees of Freedom =2 p-value = 0.1637
Class 2	26 (52%)	30 (60%)	
Class 3	20 (40%)	12 (24%)	



As seen in the above table 4, the description of the socio economic status was explained according to the modified BG Prasad Classification. About 4 (8%) and 8 (16%) were belongs to class 1, 26 (52%) from fever and 30 (60%) majority of the participants belongs to Class 2 and followed by 20 (40%) and 12 (24%) belongs to Class 3. There was no statistical association between socio economic statuses (0.1637).

Table 5: Frequencies of Parity			
Status	**Fever**	**Control**	**Test of Significance**
Primi	22 (44%)	30 (60%)	P value = 0.1160
Multi gravida	28 (56%)	20 (40%)	

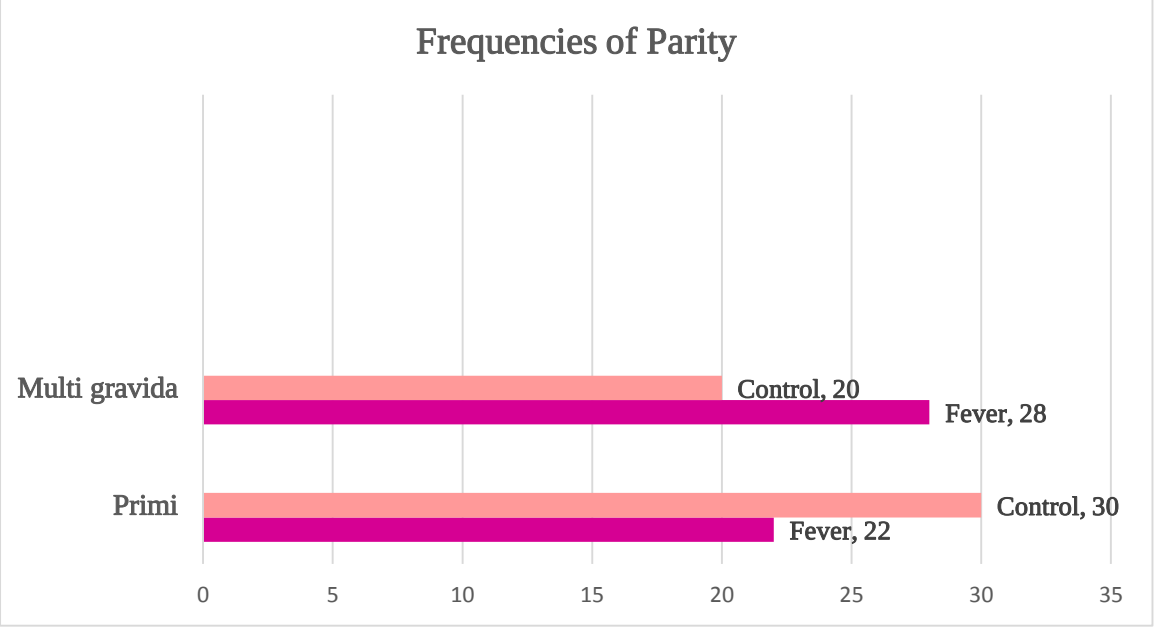


Table 5, explains the frequencies and percentages of the parity among the 50 pregnant women with fever and 50 pregnant women without fever. Among 50 fever pregnant women 22 (44%) were primi mothers and 28 (56%) were Multi gravida. Regarding 50 control, 30 (60%) were Primi mothers and 20 (40%) were Multi gravida. The association is statistically insignificant (p value 0.1160)

Table 6: Frequencies of Vital Measurements			
	Fever	Control	Chi Square test
Hypotension	12 (24%)	8 (16%)	P value = 0.4718
Tachycardia	28 (56%)	18 (36%)	

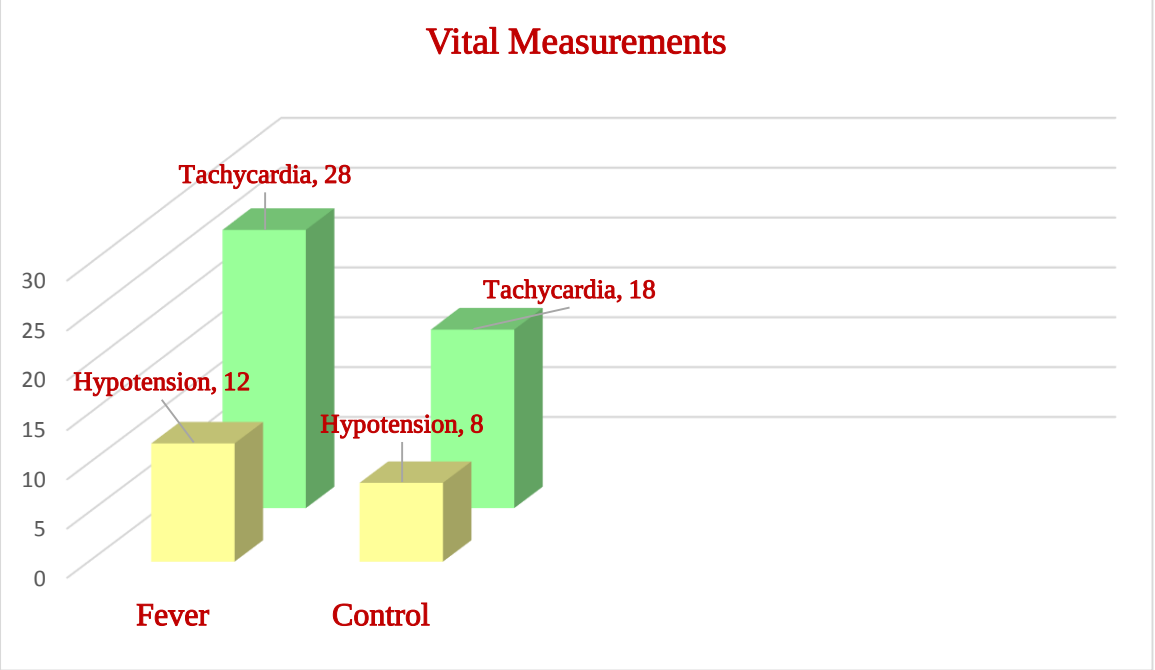


Table 6, explains the frequencies and percentages of the vital measurements among the 50 pregnant women 12 (24%) had Hypotension and 28 (56%) had Tachycardia. Regarding 50 control, 8 (16%) had hypotension and 18 (36%) had tachycardia. The association is statistically insignificant (p value 0.4718)

Table 7: Frequencies of Gestational Age			
Age	**Fever**	**Control**	**Chi Square test**
Term	46 (92%)	42 (84%)	P value = 0.1195
Post dated	4 (8%)	8 (16%)	

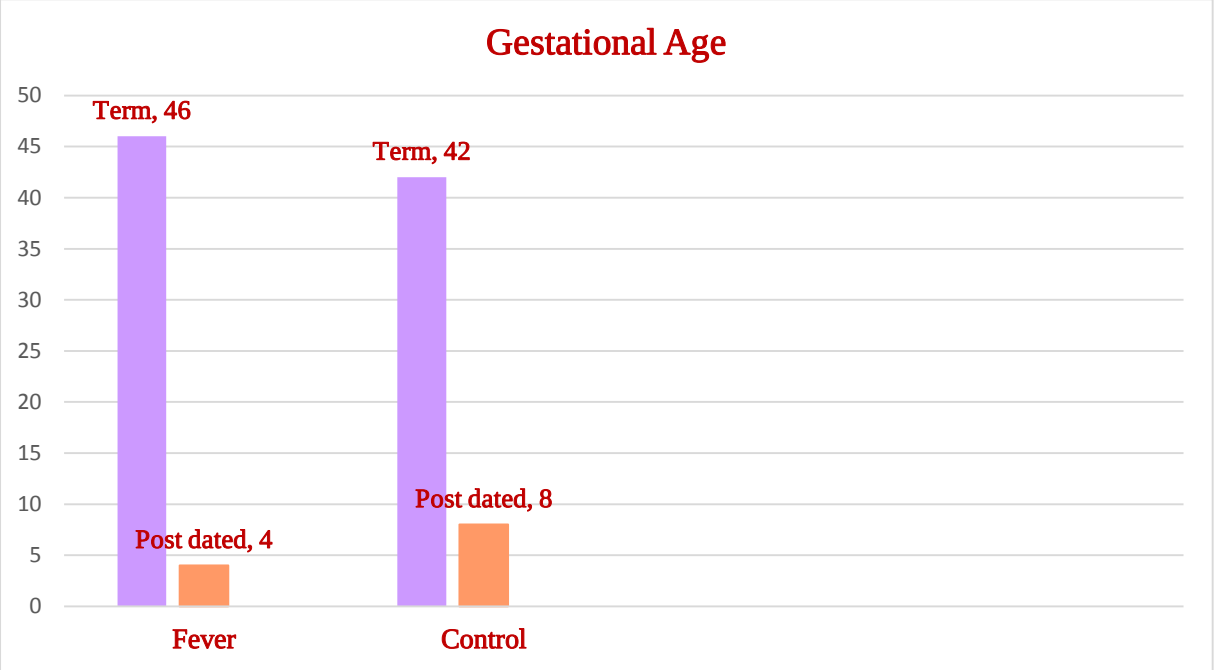


Table 7, explains the frequencies and percentages of the Gestational age among the 50 pregnant women with fever was 46 (92%) were in Term and 4 (8%) was Post-dated. Regarding 50 control, 42 (84%) were in Term and 8 (16%) were in Post-dated. The association was statistically insignificant (p value 0.1195)

Table 8: Relation of IUGR in pregnancy fever			
IUGR	**Fever**	**Control**	**Chi Square test**
Present	8 (16%)	3 (6%)	P value = 0.0627
Absent	42 (84%)	47 (94%)	

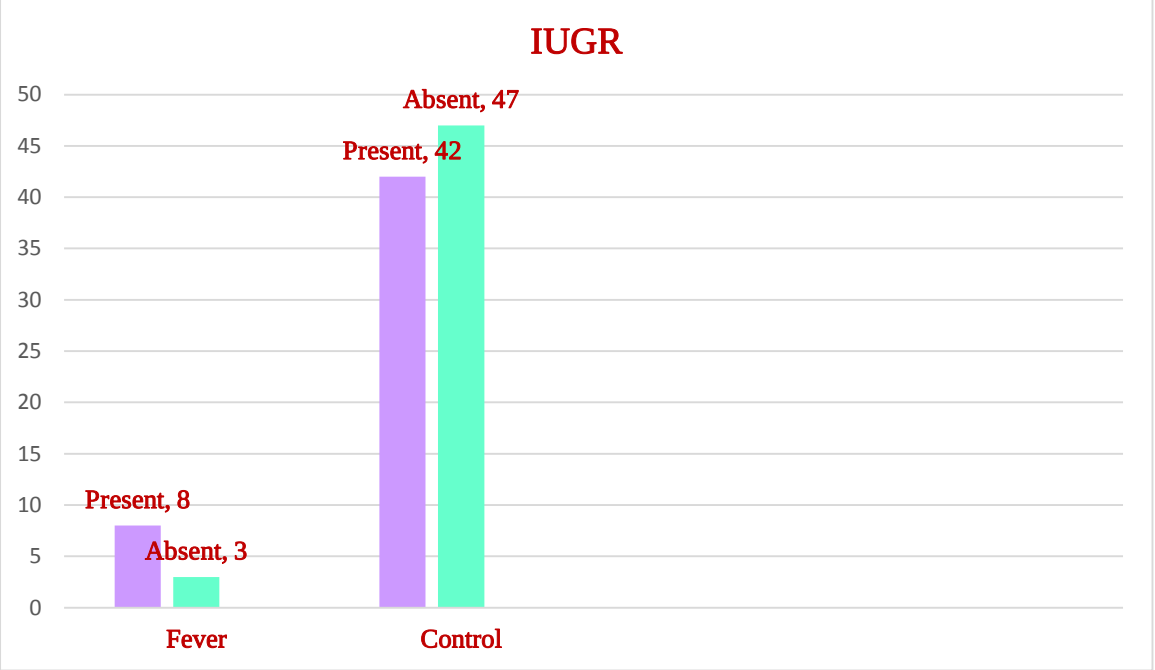


Table 8, explains the frequencies and percentages of the IUGR among the 50 pregnant women with fever was 8 (16%) had IUGR and in control only 3 (6%) had IUGR. Among the pregnant women with fever IUGR was slightly higher than the non-fever pregnant women. Though it's statistically insignificant (0.0627).

Table 9: Association between Labour onset with Fever and Control			
Labour Onset	**Fever**	**Control**	**Test of Significance**
Spontaneous	22 (44%)	26 (52%)	Degrees of Freedom = 2 p- Value = 0.4342
Induced	16 (32%)	12 (24%)	
Repeat LSCS	12 (24%)	12 (24%)	

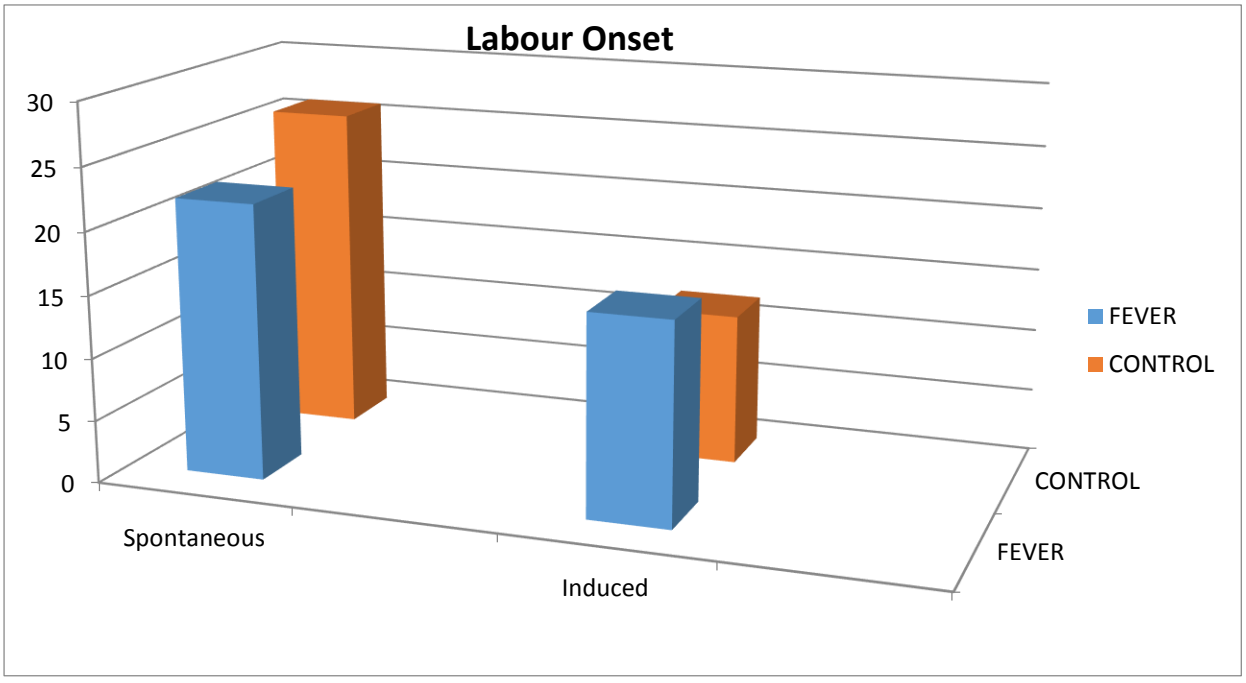


Table 9 explains study of 50 fever cases, 12 (24%) cases were Previous LSCS cases, 22 (44%) cases went into spontaneous labour, only 16 (32%) cases required intervention for the better maternofetal outcome. Out of 16 (8 primigravida, 8 multigravida) cases induced, 8(50%) cases were delivered vaginally and 8(50%) Cases underwent caeserean sections. In other group also, 12(24%) cases were Previous LSCS cases, 26 (52%) cases went into spontaneous labour,12 cases (24%) cases were induced. Out of 12 (10 primigravida, 2 multigravida) cases induced 10 cases delivered vaginally and 2 cases underwent LSCS. The association was statistically insignificant (p value0.4342).

Table 10: Association between Mode of Delivery with fever and control			
Mode	**Fever**	**Control**	**Test of Significance**
Labour Natural	14 (28%)	20 (40%)	Chi Square = 1.669 Degrees of Freedom =2 p-value = 0.4342
LSCS	33 (66%)	28 (56%)	
Forceps/Instrument assisted	3 (6%)	2 (4%)	

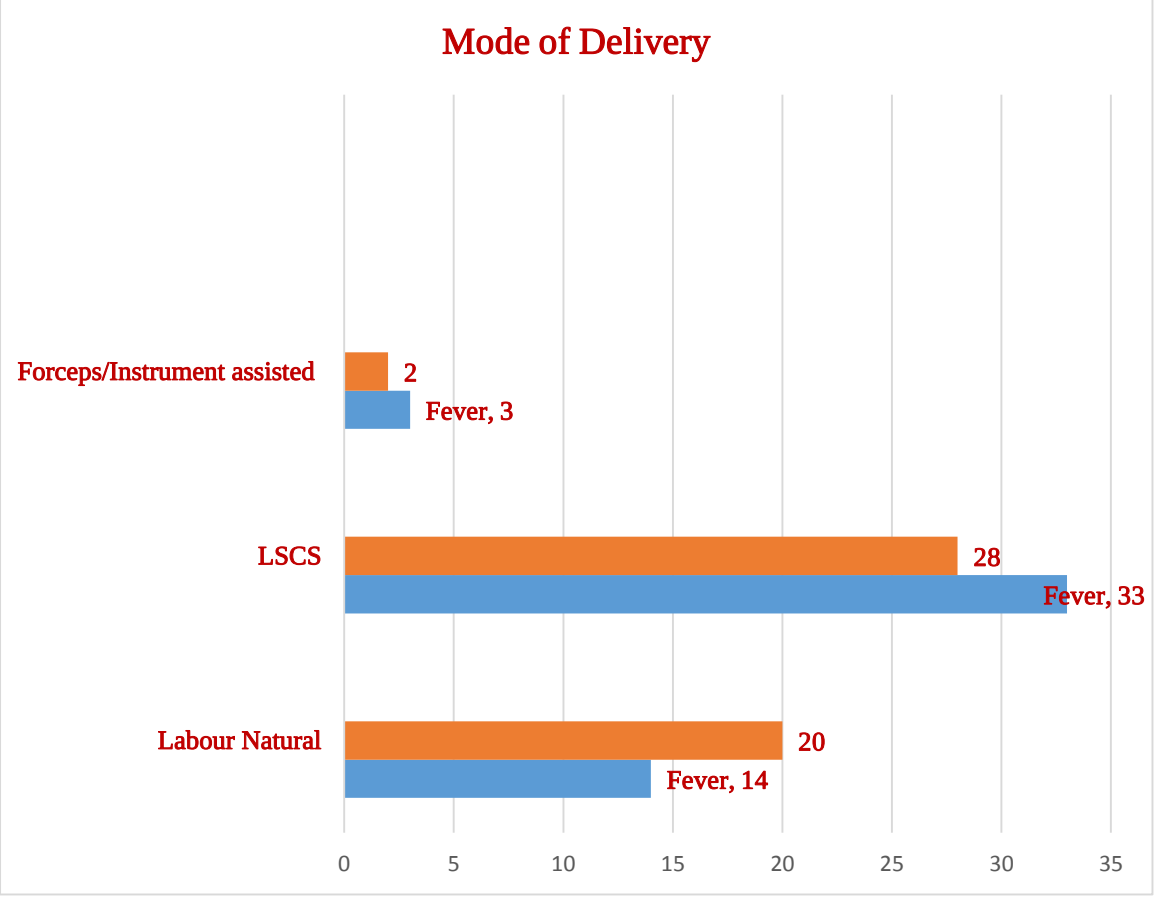


Table 10, explains the association between mode of delivery with pregnant mother with fever and without fever. Among the 50 pregnant women with fever, 14 (28%) had labour natural, 33 (66%) had LSCS and only 3 (6%) had Forceps/Instrument assisted. Regarding 50 control, 20 (40%) had labour natural, 28 (56%) had LSCS and only 2 (4%) had Forceps/Instrument assisted. The association was statistically insignificant (p value 0.4342)

Table 11: Association of Liquor colour and fever			
Liquor	**Fever**	**Control**	**Chi Square test**
Clear	25 (50%)	35 (70%)	P value= 0.0412
Meconium stained	25 (50%)	15 (30%)	

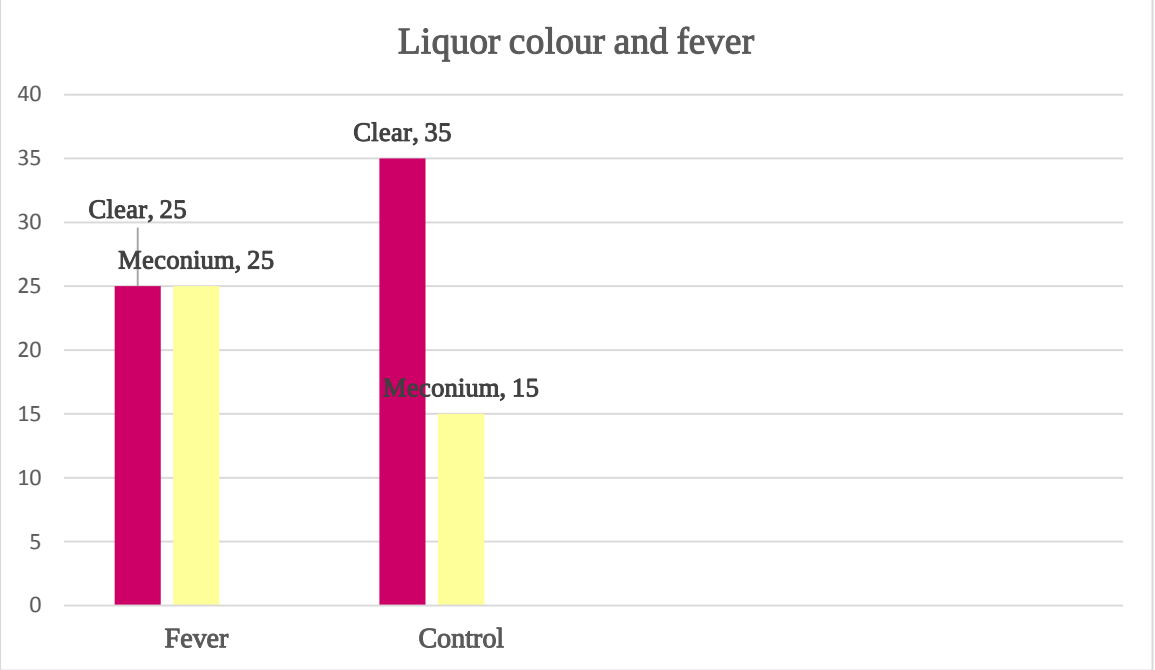


Table 11, explains the Association of liquor colour with pregnant mother with fever and without fever. Among the 50 pregnant women with fever had 25 (50%) clear Liquor and other half 25 (50%) had Meconium stained liquor. Regarding 50 control, 35 (70%) had clear liquor and 15 (30%) had meconium stained liquor. The association was found to be statistically significant (p value 0.0412)

Table 12: Correlation with Associated Comorbidities			
	Fever	Control	Test of Significance
Hypertension	16 (32%)	3 (6%)	Chi Square = 15.69
Anaemia	8 (16%)	5 (10%)	Degrees of Freedom =1
Pedal oedema	4 (8%)	1 (2%)	p-value = <0.0001
No Comorbidities	22 (44%)	41 (82%)	

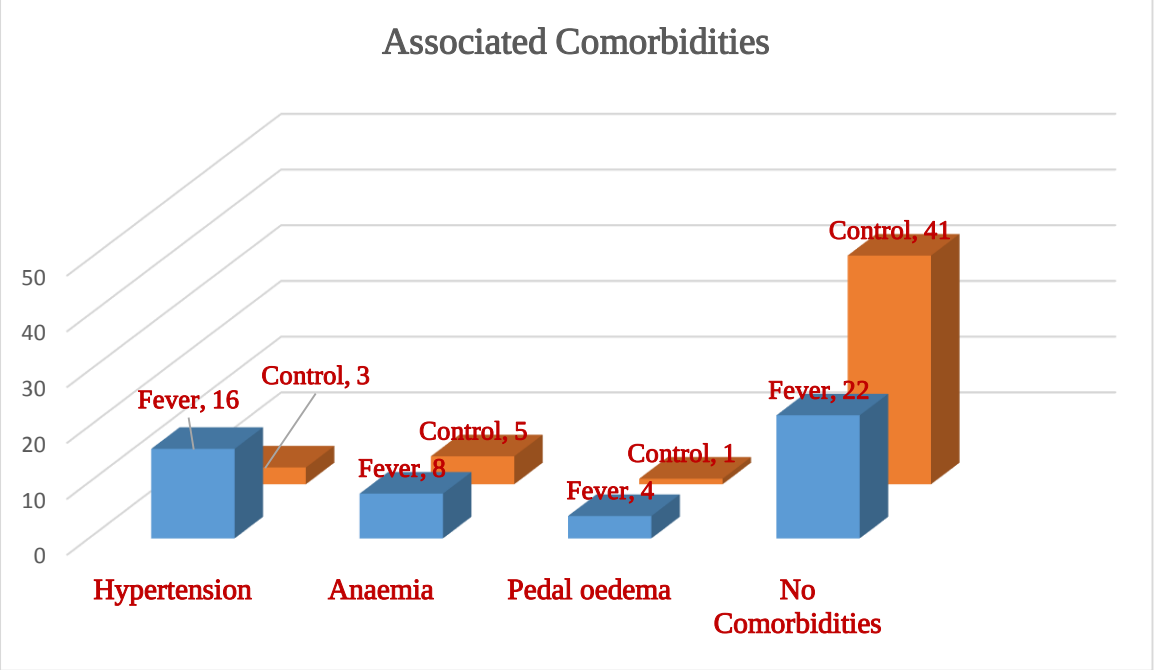


Table 12, explains the correlation between associated comorbidities with pregnant mother with fever and without fever. Among the 50 pregnant women with fever, 16 (32%) had Gestational Hypertension, 8 (16%) had Anaemia and only 4 (8%) had Pedal Oedema. Regarding 50 control, 3 (6%) had Gestational Hypertension, 5 (10%) had Anaemia and only 1 (2%) had Pedal oedema. The association was found to be statistically significant (p value < 0.0001)

Table 13: Correlation with Maternal Complications			
	Fever	Control	Chi Square test
No Complications	46 (92%)	49 (98%)	P value= 0.1688
Maternal Complications	4 (8%)	1 (2%)	

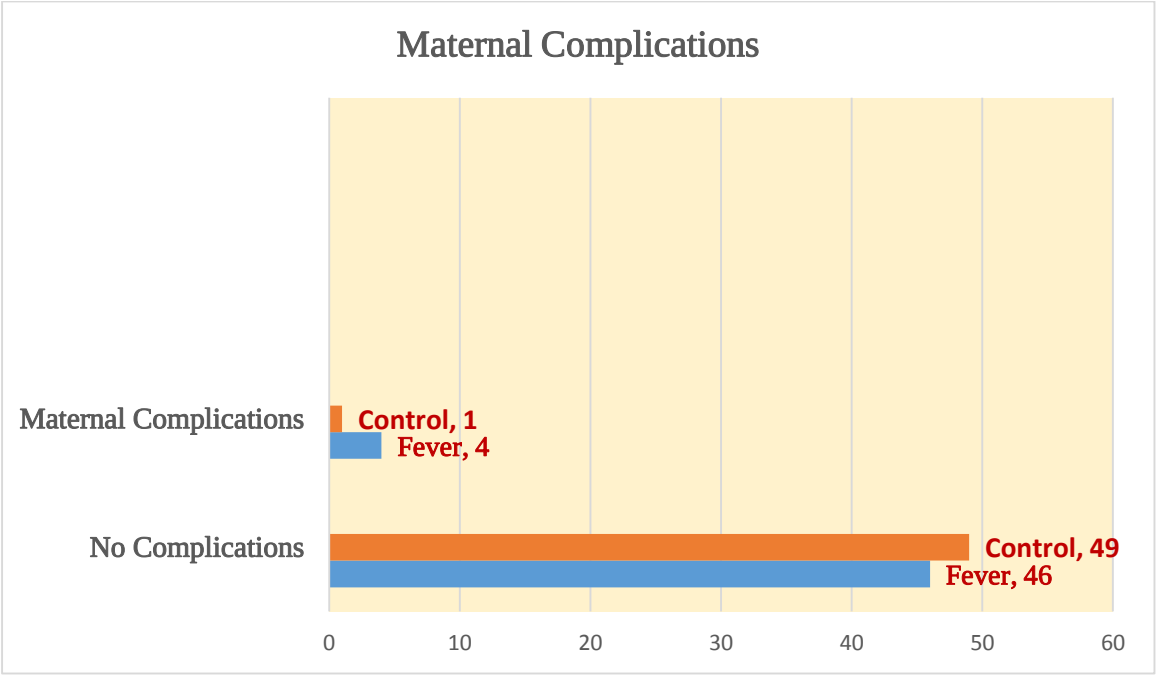


Table 13, explains the Correlation of Maternal Complications with pregnant mother with fever and without fever. Among the 50 pregnant women with fever had 4 (8%) had maternal complications such as Postpartum haemorrhage and 46 (92%) majority of them didn't have any complications. Regarding 50 control, 49 (98%) had no complications and only one case had the complication. The association was found statistically insignificant (p value 0.1688)

Table 14 : Relationship of Birth weight with fever in Pregnancy			
Weight	**Fever**	**Control**	**Chi Square test**
Normal	35 (70%)	38 (76%)	P value= 0.4992
Low birth weight	15 (30%)	12 (24%)	

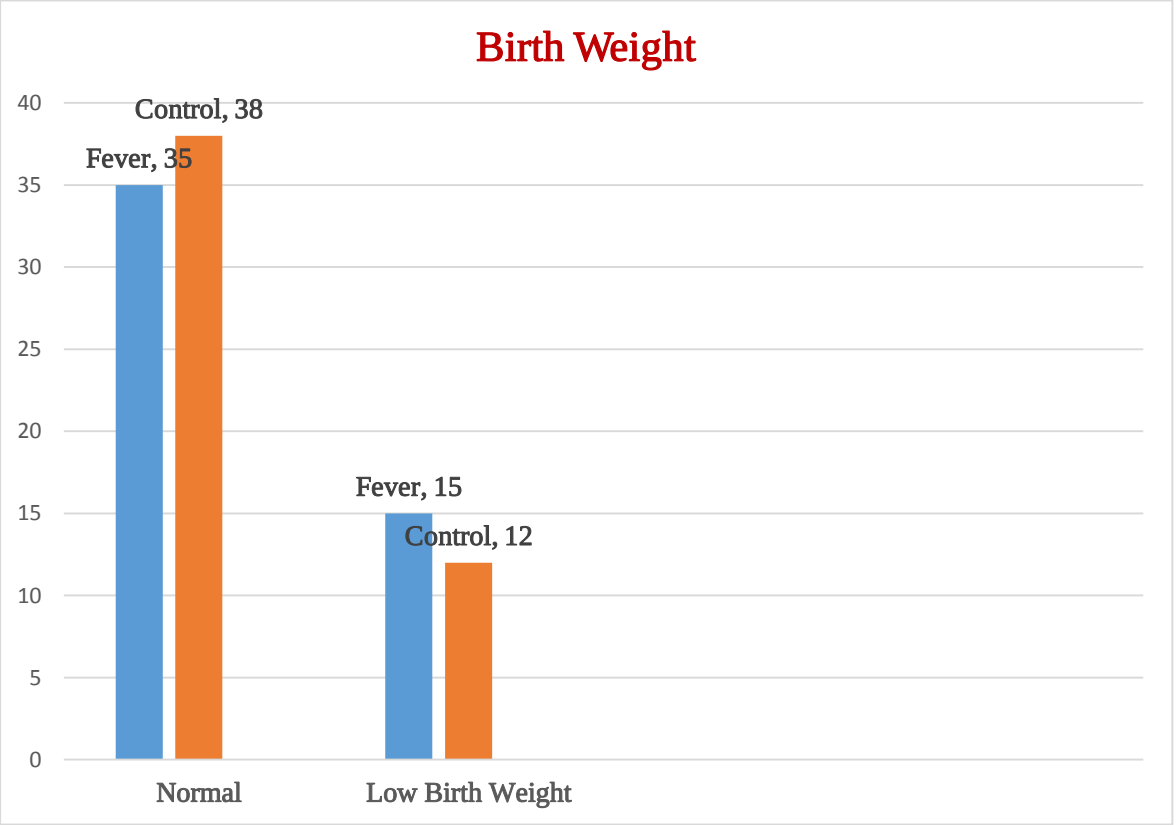


Table 14, explains the Relationship of Birth weight with pregnant mother with fever and without fever. Among the 50 pregnant women with fever had 35 (70%) had Normal birth weight babies and rest of the15 (30%) had Low birth weight babies less than 2.5kgs. Regarding 50 control, 38 (76%) had normal delivery and 12 (24%) had low birth weight babies. The association was found statistically insignificant (p value 0.4992)

Table 15: Relationship of APGAR Score with fever in Pregnancy			
APGAR Score (Mean)	Fever	Control	P Value
One minute	6.75 ±0.54	6.98± 0.46	0.0240
5 Minute	7.94 ±0.23	8.06 ±0.31	0.0302

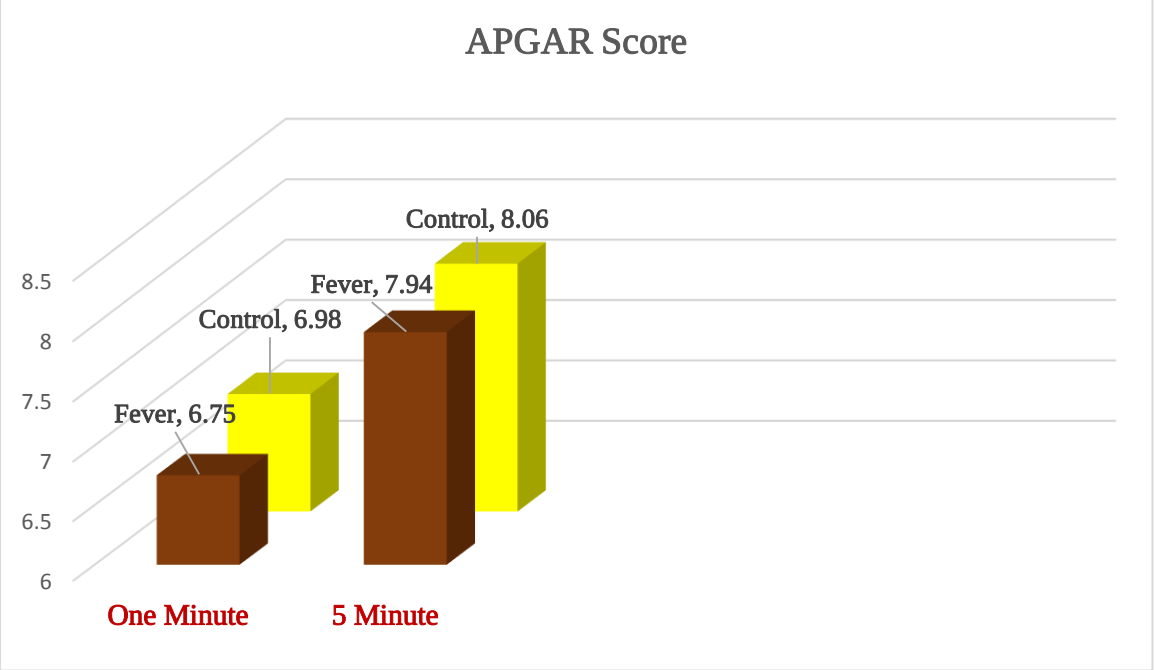


Table 15, explains the Relationship of APGAR Score with pregnant mother with fever and without fever. The mean APGAR score for One minute was 6.75 ±0.54 and for control 6.98± 0.46 it was found to be statistically significant (P value 0.0240). Regarding the APGAR Score for One minute was 7.94 ±0.23 and for controls 8.06 ±0.31. The association was found statistically significant (P value 0.0302).

Table 16: Correlation of Respiratory distress with fever in Pregnancy			
Distress	**Fever**	**Control**	**Chi Square test**
Present	4 (8%)	1 (2%)	P value= 0.1688
Absent	46 (92%)	49 (98%)	

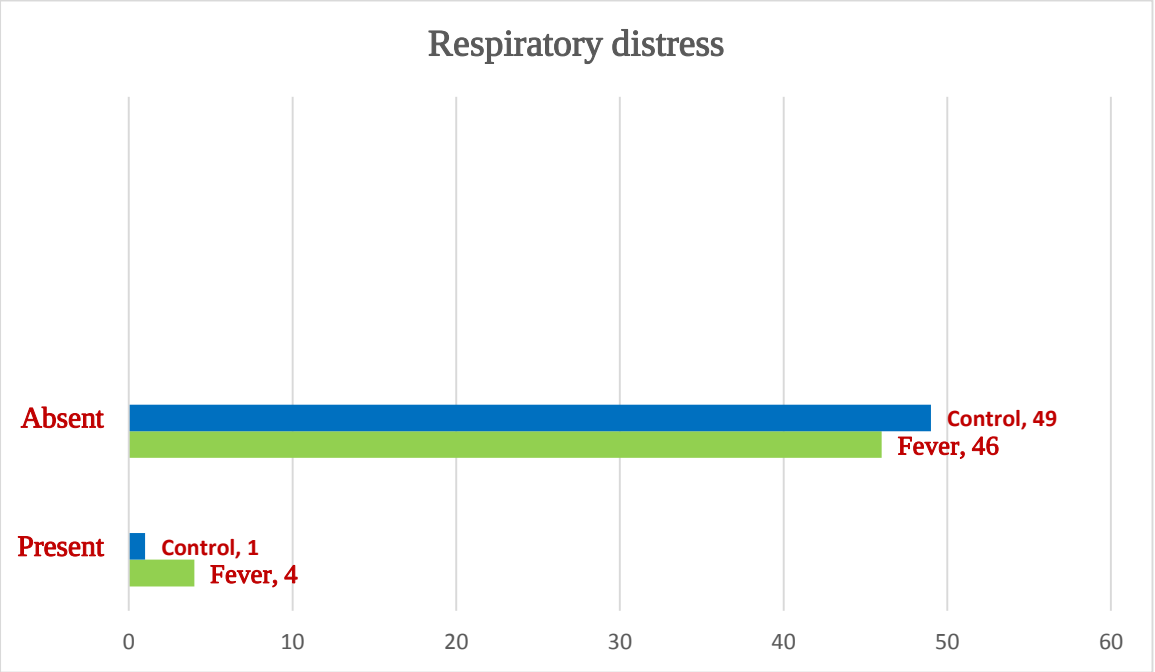


Table 16, explains the Correlation of Respiratory distress with pregnant mother with fever and without fever. Among the 50 pregnant women with fever had 4 (8%) had respiratory distress and only case from the control experienced respiratory distress. The association was found statistically insignificant (p value 0.1688).

Table 17: Association of NICU Admission with fever in Pregnancy			
NICU Admission	**Fever**	**Control**	**Chi Square test**
Required	44 (88%)	22 (44%)	p-value = <0.0001
Not Required	6 (12%)	28 (56%)	

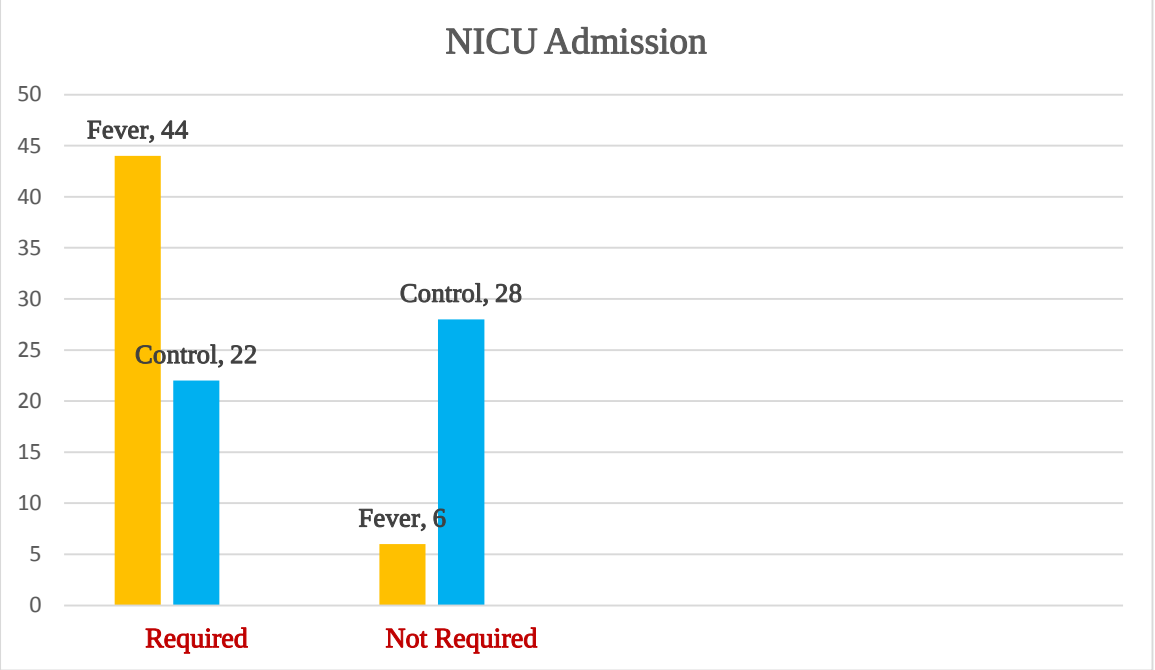


Table 17, explains the Association of NICU admission among the 50 pregnant women with fever was 44 (88%) required NICU admission and 6 (12%) doesn't required NICU admission. Regarding 50 control, 22 (44%) required NICU admission and 28 (56%) doesn't required NICU admission. The association was found to be statistically very significant (p value <0.0001).

DISCUSSION

Fever during pregnancy is a common clinical condition around the world, and when it is worsened by infection, it poses a considerable risk to both the mother and the foetus. Pyrexia during pregnancy might be difficult to manage because of its unusual symptoms. Because of the loss in maternal immune function, the strongest antibiotics must be administered with caution in pregnant women, due to the teratogenic risk.[186, 187]

In our study, 20% were belongs to the age group of 16-20 years, 24% were belongs to the age group of 21-25 years, 36% were belongs to the age group of 26-30 years and 20% were belongs to the age group of 31-35 years. Majority of the cases represented from the 26-30 years age group. Kaushik Banerjee et al also found the same most of the patients are in the age group of 20-30 years.

In our study, most of them had due to respiratory infections 24 (48%), followed by Urinary tract infection 18 (36%), Typhoid 3 (6%) and Others 5 (10%).

In the current study, fever pregnant women 44% were primi mothers and 56% were Multi gravida. Regarding control, 60% were Primi mothers and 40% were Multi gravida. This observation correlates with the study conducted by Chambers CD et al the fever group consisted 38% of primigravida.

In our study of 50 fever cases, 24% cases were Previous LSCScases, 44% cases went into spontaneous labour, only 32% cases required induction. Out of 32% cases induced, 50% cases were delivered vaginally and 50% Cases underwent

caeserean sections. So, Fever did not significantly affect onset of labour and the mode of delivery.

In our study, 92% were in Term and 4 (8%) was Post-dated. Among the pregnant women with fever IUGR was slightly higher (16%) than the non-fever pregnant women. Though it's statistically insignificant (0.0627).

In the present study 16% had IUGR and in control only 6% had IUGR. Among the pregnant women with fever IUGR was slightly higher than the non-fever pregnant women. Though it's statistically insignificant (0.0627). According to Lieberman et al[189], there is a substantial link between intrapartum fever and a low Apgar score. In our study the mean APGAR score for One minute was 6.75 $\pm$0.54 and for control 6.98$\pm$ 0.46 it was found to be statistically significant (P value 0.0240). Nath J et al found low APGAR score (<7) in 18.6% cases.[118] However, Biswas J and associates found a higher proportion of cases, i.e. 44.8% with similar low APGAR scores.

In our study 15% had Low birth weight among the pregnant women with fever. Low birth weight was the most common foetal result (56.8%), followed by pre-term birth, according to Biswas J et al. IUDs are used in 36 percent of cases, IUGR in 30% of cases, and stillbirth in 2.1 percent of cases (1.4 percent).[18] Nath J et al found almost identical results, with 52.6 percent low birth weight cases, 27.8 percent preterm deliveries, 20.3 percent IUGR, 5% stillbirths, and 2.8 percent IUDs. [188] Preterm labour was found to be 33 percent in a study conducted by Zeeman et al, and 28 percent in a study

conducted by Chambers CD et al. Cotch and colleagues discovered that women with chorioamnionitis had a higher rate of premature labour in their neonates (30 percent).

In this study, 50% clear Liquor and other half had Meconium stained liquor. The association was found to be statistically significant (p value 0.0412). Pregnant women with fever, 32% had Gestational Hypertension, 16% had Anaemia and only 8% had Pedal oedema. The association was found to be statistically significant (p value < 0.0001). Only 8% had maternal complications such as Postpartum haemorrhage. In this study, 8% of the newborn had respiratory distress and 88% required NICU admission.

CONCLUSION

Majority of the cases represented from the 26-30 years age group.The mean duration of the fever in days was 2.22 $\pm$ 1.01. The mean temperature of the fever in Fahrenheit was 100.68$\pm$ 0.74.

Most common cause of fever in pregnancy was respiratory tract infections. Urinary tract infection was the second common cause. Pregnant women with fever 24% had Hypotension and 56% had Tachycardia.

Pregnant women with fever had almost half the delivery with meconium stained significantly. Among 50 delivered fever mothers, 24% cases were Previous LSCS cases, 44% cases went into spontaneous labour, only 32% cases induced. Out of 32% cases induced, 50% cases were delivered vaginally and 50% Cases underwent caeserean sections. So, Fever did not significantly affect labour onset and the mode of delivery.

Significantly NICU admission was required among the fever cohorts.

This study was conducted in a full year to cover all seasons, since many febrile illness are seasonal, coverage of full year remove this bias and it reflects the true incidence of various causes of fever.

The current COVID-19 pandemic demonstrates performing a rapid diagnosis in pregnant women presenting with fever allow for appropriate care including Hospitalization, Out patient management and prescribing antibiotics.

Fever during pregnancy is a common occurrence that can lead to a wide range of issues for both the mother and the foetus and newborn. Because the most prevalent causes of fever are preventable, there has to be a greater emphasis on the importance of being aware of how to avoid such illnesses in order to avoid life-threatening foeto-maternal consequences.

Early diagnosis and therapy can be aided by simple laboratory testing. Standard infection control procedures in homes, communities, and healthcare settings, as well as increased health education and awareness, will go a long way toward reducing such negative foeto-maternal effects.

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PROFORMA

NAME:

AGE:

IP.NO:

D.O.A:

S.E.S:

EDUCATION:

ADDRESS:

INCOME:

OCCUPATION:

COMPLAINTS:

Duration of Fever

Type of Fever

Any H/O chills, rigors, vomiting

Any H/O Burning micturition

Any H/O Abdominal pain, loose stools

MENSTRUAL HISTORY:

MARITAL HISTORY

OBSTETRIC HISTORY:

PERSONAL HISTORY:

PAST MEDICAL HISTORY:

PAST SURGICAL HISTORY:

FAMILY HISTORY:

MODE OF DELIVERY:

INDICATION FOR LSCS:

COLOUR OF LIQUOR:

POST OPERATIVE COMPLICATIONS:

POST NATAL COMPLICATIONS;

BABY DETAILS:

GENERAL EXAMINATION:

Height:	Pallor:
Weight:	Icterus:
BMI:	Edema:

VITALS:

Temperature:	Pulse Rate:
Blood pressure:	Respiratory rate:

SYSTEMIC EXAMINATION:

CVS-

RS-

CNS-

P/A:

P/V:

CONSENT FORM

STUDY TITLE:

"FETOMATERNAL OUTCOME OF PATIENTS WITH FEVER DURING TERM PREGNANCY"

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY GMKMCH -SALEM

PARTICIPANT NAME:

AGE:

SEX:

I.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.
I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time :

Patient name;

Date :

Signature / Thumb Impression of Patient:

Place

Name and signature of the Investigator

ஆராய்ச்சி ஒப்புதல் படிவம்

பெயர்: தேதி:

வயது: உள்நோயாளி எண்:

பாலினம்: ஆய்வு சேர்க்கை எண்:

இந்த ஆய்வின் நோக்கம் மற்றும் விவரங்கள் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது .இவ்வாய்வில் இருந்து நான் எந்த நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எனக்கு எந்த பாதிப்பும் இல்லை என்பதையும் தெளிவாக புரிந்து கொண்டேன்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

இந்த ஆய்வில் எவ்வித நிர்பந்தமும் இன்றி எனது சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகின்றேன்.

நான் சுயநினைவுடனும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் சேர்த்துக்கொள்ள சம்மதிக்கின்றேன்.

ஆராய்சியாளர் ஒப்பம்

பங்கேற்பாளர் ஒப்பம்

(அ)

இடது பெருவிரல்ரேகை

MASTER CHART (CASES)

S.NO	NAME	AGE	PARITY	GESTATIONAL AGE	SOCIOECONOMIC STATUS	DURATION OF FEVER	TEMPERATURE	BP	PR	CAUSE	IUGR YES/NO	LABOUR ONSET	MODE OF DELIVERY	COLOUR OF LIQUOR	ASSOCIATED COMORBIDITY	PPH	SEPSIS	BIRTH WT	APGAR		RESPIRATORY DISTRESS	NICU ADMISSION	SEPSIS
1	Karthiga	32	G2P1L1	39wks+5days	3	2	100	110/70	116	RTI	YES	-	REPEAT LSCS	Clear	GHTN	-	-	2.2kg	7	8	NO	YES	-
2	Seetha	23	PRIMI	37wks	1	5	101	100/70	118	THYPHOID	-	Spontaneous	LSCS	MSAF	Severe PE	-	-	1.8kg	7	8	NO	YES	-
3	vanitha	23	G2P1L1	38wks+3days	2	2	100	100/60	118	UTI	-	Induced	LSCS	MSAF	-	-	-	2.5kg	7	8	NO	YES	-
4	Ishwarya	33	PRIMI	37wks+5days	2	1	101	120/70	98	OTHER	-	Spontaneous	LSCS	MSAF	-	-	-	2.4kg	5	7	YES	YES	-
5	Priya	32	G2P1L1	39wks+5days	3	2	100	110/70	116	RTI	YES	-	REPEAT LSCS	MSAF	GHTN	-	-	2.2kg	7	8	NO	YES	-
6	Pooja	23	G2P1L1	38wks+3days	2	2	100	100/60	118	RTI	-	Induced	LSCS	MSAF	-	-	-	2.5kg	7	8	NO	YES	-
7	Saraswati	19	G2A1	37wks+2days	3	2	100	90/60	112	UTI	-	Spontaneous	LSCS	Clear	GHTN	-	-	2.2kg	7	8	NO	YES	-
8	Sivaranjani	19	PRIMI	37wks+5days	2	3	101	90/60	118	RTI	-	Spontaneous	LN	Clear	-	-	-	2.7kg	7	8	NO	NO	-
9	Ranjani	27	PRIMI	38+2days	2	4	100	100/60	98	RTI	-	Spontaneous	LN	Clear	Anaemia	-	-	2.6kg	7	8	NO	YES	-
10	Kokila	26	PRIMI	38wks+6days	2	1	101	110/80	114	OTHER	-	Induced	LSCS	MSAF	-	-	-	2.8kg	6	8	NO	YES	-
11	Monisha	25	G3P2L2	39wks+2days	2	1	100	100/70	102	RTI	-	Spontaneous	LN	Clear	GHTN	YES	-	2.6kg	6	8	NO	YES	-
12	Geetha	23	PRIMI	37wks	1	2	101	100/70	118	THYPHOID	-	Spontaneous	LSCS	MSAF	Severe PE	-	-	1.8kg	7	8	NO	YES	-
13	Priya	19	PRIMI	37wks+5days	2	3	101	90/60	118	RTI	-	Spontaneous	LN	Clear	-	-	-	2.7kg	7	8	NO	YES	-
14	Priyadharshini	23	G3P1L1A1	37wks	3	1	102	110/80	116	UTI	-	-	REPEAT LSCS	MSAF	-	-	-	2.7kg	7	8	NO	YES	-
15	Jeeva	19	PRIMI	37wks+5days	2	3	101	90/60	118	RTI	-	Induced	LSCS	MSAF	-	-	-	2.7kg	7	8	NO	YES	-
16	Fathima	23	G2P1L1	38wks+3days	2	2	100	100/60	118	RTI	-	Spontaneous	LSCS	MSAF	-	-	-	2.5kg	7	8	NO	YES	-
17	Geethalaksmi	27	PRIMI	38wks	2	4	100	100/60	98	UTI	-	Induced	LSCS	MSAF	Anaemia	-	-	2.6kg	6	8	YES	YES	-
18	Anandhi	28	G2P1L1	38wks+2days	3	3	101	90/60	124	RTI	-	-	REPEAT LSCS	Clear	-	-	-	3.3kg	7	8	NO	YES	-
19	Sivanya	26	PRIMI	38wks+6days	2	1	101	110/80	114	UTI	-	Spontaneous	LSCS	MSAF	-	-	-	2.8kg	6	8	NO	YES	-
20	Rizwana Banu	19	G2A1	37wks+2days	3	2	100	90/60	112	OTHER	-	Spontaneous	LSCS	MSAF	GHTN	-	-	2.2kg	7	8	NO	YES	-
21	Hema	27	G2P1L0	40wks+2days	2	2	100	100/70	92	OTHER	-	Induced	VACCUM	MSAF	GHTN	-	-	2.7kg	7	8	NO	YES	-
22	Sathya	27	G2P1L0	40wks+2days	2	5	100	100/70	92	UTI	-	Induced	LN	Clear	GHTN	-	-	2.7kg	7	8	NO	NO	-
23	Latha	28	G2P1L1	38wks+2days	3	3	101	90/60	124	RTI	-	-	REPEAT LSCS	Clear	-	-	-	3.3kg	7	8	NO	YES	-
24	Karunya	27	PRIMI	38wks	2	4	100	100/60	98	RTI	-	Spontaneous	LN	Clear	Anaemia	-	-	2.6kg	7	8	NO	YES	-
25	Amin	23	G3P1L1A1	37wks	3	1	102	110/80	116	UTI	-	-	REPEAT LSCS	Clear	-	-	-	2.7kg	7	8	NO	YES	-
26	Jeeva	28	G2P1L1	38wks+2days	3	3	101	90/60	124	RTI	-	-	REPEAT LSCS	Clear	-	-	-	3.3kg	7	8	NO	YES	-
27	Kalaivani	32	G2P1L1	39wks+5days	3	2	100	110/70	116	OTHER	YES	-	REPEAT LSCS	Clear	GHTN	-	-	2.2kg	7	8	NO	YES	-
28	Rani	28	G2P1L1	38wks+2days	3	3	101	90/60	124	UTI	-	-	REPEAT LSCS	MSAF	-	-	-	3.3kg	7	8	NO	YES	-
29	Kalaiarasi	23	PRIMI	37wks	1	2	101	100/70	118	RTI	-	Spontaneous	LSCS	MSAF	Severe PE	-	-	1.8kg	7	8	NO	YES	-
30	Karthiga	27	G2P1L0	40wks+2days	2	2	100	100/70	92	THYPHOID	-	Spontaneous	LSCS	MSAF	GHTN	-	-	2.7kg	7	8	NO	YES	-
31	Keerthi	19	G2A1	37wks+2days	3	2	100	90/60	112	UTI	-	Spontaneous	LSCS	Clear	GHTN	-	-	2.2kg	7	8	NO	YES	-
32	kalyani	27	PRIMI	38wks	2	4	100	100/60	98	RTI	-	Spontaneous	FORCEPS	Clear	Anaemia	-	-	2.6kg	7	8	NO	YES	-
33	Ponni	33	PRIMI	37wks+5days	2	2	101	120/70	98	UTI	-	Spontaneous	LSCS	MSAF	-	-	-	2.4kg	5	7	YES	YES	-
34	Yasmin	19	G2A1	37wks+2days	3	2	100	90/60	112	RTI	-	Spontaneous	LSCS	Clear	GHTN	-	-	2.2kg	7	8	NO	YES	-
35	keerthana	23	G3P1L1A1	37wks	3	1	102	110/80	116	RTI	-	-	REPEAT LSCS	MSAF	-	-	-	2.7kg	7	8	NO	YES	-
36	Mythili	23	G2P1L1	38wks+3days	2	2	100	100/60	118	UTI	-	Induced	LSCS	MSAF	-	-	-	2.5kg	7	8	NO	YES	-
37	Kokila	26	PRIMI	38wks+6days	2	1	101	110/80	114	RTI	-	Induced	LSCS	MSAF	-	-	-	2.8kg	6	8	NO	YES	-
38	Gayathri	25	G3P2L2	39wks+2days	2	1	100	100/70	102	RTI	-	Spontaneous	LN	Clear	GHTN	-	-	2.6kg	6	8	NO	YES	-
39	Pavithra	24	PRIMI	39wks+6days	3	2	102	100/60	118	UTI	-	Induced	LN	Clear	Anaemia	YES	-	2.8kg	7	8	NO	YES	-
40	Radha	19	PRIMI	37wks+5days	2	3	101	90/60	118	UTI	-	Induced	VACCUM	MSAF		-	-	2.7kg	7	8	NO	YES	-
41	Seetha	33	PRIMI	37wks+5days	2	2	101	120/70	98	RTI	-	Spontaneous	LSCS	MSAF	-	-	-	2.4kg	5	7	YES	YES	-
42	Latha	23	G3P1L1A1	37wks	3	1	102	110/80	116	UTI	-	-	REPEAT LSCS	Clear	-	-	-	2.7kg	7	8	NO	YES	-
43	Priya	23	PRIMI	37wks	1	2	101	100/70	118	RTI	-	Spontaneous	LSCS	MSAF	Severe PE	YES	-	1.8kg	7	8	NO	YES	-
44	Panchali	24	PRIMI	39wks+6days	3	2	102	100/60	118	UTI	-	Spontaneous	LN	Clear	Anaemia	YES	-	2.8kg	7	8	NO	NO	-
45	suganthi	32	G2P1L1	39wks+5days	3	2	100	110/70	116	RTI	YES	-	REPEAT LSCS	MSAF	GHTN	-	-	2.2kg	7	8	NO	YES	-
46	Meena	24	PRIMI	39wks+6days	3	2	102	100/60	118	RTI	-	Spontaneous	LN	Clear	Anaemia	YES	-	2.8kg	7	8	NO	YES	-
47	Meera	25	G3P2L2	39wks+2days	2	1	100	100/70	102	UTI	-	Induced	LN	Clear	GHTN	-	-	2.6kg	7	8	NO	YES	-
48	Bakiyalakshmi	25	G3P2L2	39wks+2days	2	2	100	100/70	102	UTI	-	Induced	LN	Clear	GHTN	-	-	2.6kg	6	8	NO	YES	-
49	Anitha	24	PRIMI	39wks+6days	3	2	102	100/60	118	RTI	-	Induced	LN	Clear	Anaemia	YES	-	2.8kg	7	8	NO	YES	-
50	Jothi	27	G2P1L0	40wks+2days	2	2	100	100/70	92	UTI	-	Induced	LN	Clear	GHTN	-	-	2.7kg	7	8	NO	NO	-

MASTER CHART (CONTROL)

S.NO	NAME	AGE	PARITY	GESTATIONAL AGE	SOCIOECONOMIC STATUS	BP	PR	IUGR YES/NO	MODE OF ONSET	MODE OF DELIVERY	COLOUR OF LIQUOR	ASSOCIATED COMORBIDITY	PPH	SEPSIS	BIRTH WT	APGAR		RESPIRATORY DISTRESS	NICU ADMISSION	SEPSIS
1	Kavya	33	G3P2L2	37wks+3days	2	110/60	96	NO	-	REPEAT LSCS	Clear	GHTN	-	-	2.7kg	7	8	NO	YES	-
2	Jancy	20	G3P1L1A1	37wks+5days	2	120/70	118	NO	-	REPEAT LSCS	Clear	-	-	-	3.0kg	7	8	NO	NO	-
3	Pavithra	18	G2P1L1	37wks+3days	1	100/70	98	NO	-	REPEAT LSCS	Clear	-	-	-	2.8 kg	7	8	NO	NO	-
4	Kavilakshmi	27	G2P1L1	38wks+2days	2	120/70	108	NO	Induced	LN	Clear	-	-	-	2.9kg	6	8	NO	NO	-
5	Manjula	18	PRIMI	38wks	1	110/70	108	NO	Spontaneous	VACCUM	MSAF	Anaemia	-	-	2.3kg	7	8	NO	YES	-
6	Gokulapriya	32	PRIMI/BREECH	38+1day	2	120/70	114	NO	Spontaneous	LSCS	Clear	-	-	-	3kg	7	8	NO	NO	-
7	Vanitha	26	G2A1	38wks	2	100/70	94	NO	Spontaneous	LN	Clear	GHTN	YES	-	2.7kg	7	8	NO	NO	-
8	Jeevitha	18	PRIMI	38wks	1	110/70	108	NO	Spontaneous	LSCS	MSAF	Anaemia	-	-	2.3kg	7	8	NO	YES	-
9	Jothika	27	PRIMI	38wks	2	110/70	98	NO	Spontaneous	LN	Clear	-	-	-	2.2kg	6	9	NO	NO	-
10	Lakshmipathi	21	PRIMI	37wks+4days	3	110/70	96	NO	Spontaneous	LN	Clear	-	-	-	2.75kg	7	8	NO	YES	-
11	Veena	21	PRIMI	37wks+4days	3	110/70	96	NO	Induced	LN	Clear	-	-	-	2.75kg	7	8	NO	YES	-
12	Meena	31	G2P1L1	39wks+6days	2	110/70	96	NO	Spontaneous	LSCS	MSAF	-	-	-	3.2kg	7	8	NO	YES	-
13	Lakhmi	27	G3P2L2	38wks	2	110/70	98	YES	Induced	LN	Clear	-	-	-	2.2kg	6	9	NO	NO	-
14	Nandhini	21	PRIMI	37wks+4days	3	110/70	96	NO	Spontaneous	LSCS	Clear	-	-	-	2.75kg	7	8	NO	YES	-
15	Shalini	20	G3P1L1A1	37wks+5days	2	120/70	118	NO	-	REPEAT LSCS	Clear	-	-	-	3.0kg	7	8	NO	NO	-
16	Sathya	28	PRIMI	40wks+1days	3	90/70	118	NO	Induced	LN	Clear	-	-	-	3.25kg	7	8	NO	NO	-
17	Snega	26	G2A1	38wks	2	100/70	94	NO	Spontaneous	LN	Clear	GHTN	-	-	2.7kg	7	8	NO	NO	-
18	Anushya	18	PRIMI	38wks	1	110/70	108	NO	Spontaneous	LSCS	MSAF	Anaemia	-	-	2.3kg	7	8	NO	YES	-
19	Nirmala	28	PRIMI	40wks+2days	3	90/60	112	NO	Induced	LSCS	MSAF	-	-	-	2.4kg	5	6	YES	YES	-
20	Thangamani	27	PRIMI	38wks+2days	2	120/70	108	NO	Spontaneous	LN	Clear	-	-	-	2.9kg	6	8	NO	NO	-
21	Meenatchi	28	G2P1L1	40wks+1days	3	90/70	118	NO	Spontaneous	LN	Clear	-	-	-	3.25kg	7	8	NO	NO	-
22	Arivoli	20	G3P1L1A1	37wks+5days	2	120/70	118	NO	-	REPEAT LSCS	Clear	-	-	-	3.0kg	7	8	NO	NO	-
23	Alagi	18	G2P1L1	37wks+3days	1	100/70	98	NO	-	REPEAT LSCS	Clear	-	-	-	2.8 kg	7	8	NO	NO	-
24	Kalki	18	G2P1L1	37wks+3days	1	100/70	98	NO	-	REPEAT LSCS	Clear	-	-	-	2.8 kg	7	8	NO	NO	-
25	Ponnammal	18	PRIMI	38wks	1	110/70	108	NO	Spontaneous	LSCS	MSAF	Anaemia	-	-	2.3kg	7	8	NO	YES	-
26	Harini	33	G3P2L2	37wks+3days	2	110/60	96	NO	-	REPEAT LSCS	Clear	GHTN	-	-	2.7kg	7	8	NO	YES	-
27	Yasodha	28	PRIMI	40wks+2days	3	90/60	112	NO	Spontaneous	LSCS	MSAF	-	-	-	2.4kg	7	8	NO	YES	-
28	Janaki	33	G3P2L2	37wks+5days	2	120/70	98	NO	Spontaneous	LN	Clear	-	-	-	2.4kg	6	7	NO	YES	-
29	Umrin	31	G2P1L1	39wks+6days	2	110/70	96	NO	Spontaneous	LSCS	MSAF	-	-	-	3.2kg	7	8	NO	YES	-
30	Kavitha	21	PRIMI	37wks+4days	3	110/70	96	NO	Spontaneous	LN	Clear	-	-	-	2.75kg	7	8	NO	YES	-
31	Prema	32	PRIMI/BREECH	38+1day	2	120/70	114	NO	Spontaneous	LSCS	Clear	-	-	-	3kg	7	8	NO	NO	-
32	Mani	28	PRIMI	40wks+1days	3	90/70	118	NO	Induced	LN	Clear	-	-	-	3.25kg	7	8	NO	NO	-
33	Preethi	18	G2P1L1	37wks+3days	1	100/70	98	NO	-	REPEAT LSCS	Clear	-	-	-	2.8 kg	7	8	NO	NO	-
34	Ravina	31	G2P1L1	39wks+6days	2	110/70	96	NO	Spontaneous	LSCS	MSAF	-	-	-	3.2kg	7	8	NO	YES	-
35	Gayathri	20	G3P1L1A1	37wks+5days	2	120/70	118	NO	-	REPEAT LSCS	Clear	-	-	-	3.0kg	7	8	NO	NO	-
36	Faridha	26	G2A1	38wks	2	100/70	94	NO	Spontaneous	LN	Clear	GHTN	-	-	2.7kg	7	8	NO	NO	-
37	Hema	33	G3P2L2	37wks+3days	2	110/60	96	NO	-	REPEAT LSCS	Clear	GHTN	-	-	2.7kg	7	8	NO	YES	-
38	Amudha	27	PRIMI	38wks+2days	2	120/70	108	NO	Induced	LN	Clear	-	-	-	2.9kg	6	8	NO	NO	-
39	Obuli	27	PRIMI	38wks	2	110/70	98	YES	Spontaneous	LN	Clear	-	-	-	2KG	6	9	NO	NO	-
40	Priyadharshini	28	PRIMI	40wks+2days	3	90/60	112	NO	Induced	LSCS	MSAF	-	-	-	2.4kg	7	8	NO	YES	-
41	Kalaivani	26	PRIMI	38wks+6days	2	110/80	114	NO	Spontaneous	LSCS	MSAF	-	-	-	2.8kg	6	8	NO	YES	-
42	Keerthi	31	G2P1L1	39wks+6days	2	110/70	96	NO	Spontaneous	LSCS	MSAF	-	-	-	3.2kg	7	8	NO	YES	-
43	Tamilarasi	32	PRIMI/BREECH	38+1day	2	120/70	114	NO	Spontaneous	LSCS	MSAF	-	-	-	3kg	7	8	NO	NO	-
44	Imran	28	PRIMI	40wks+1days	3	90/70	118	NO	Induced	VACCUM	MSAF	-	-	-	3.25kg	7	8	NO	NO	-
45	Hemalatha	28	PRIMI	40wks+2days	3	90/60	112	NO	Induced	LSCS	MSAF	-	-	-	2.4kg	7	8	NO	YES	-
46	Immaculate mary	26	G2A1	38wks	2	100/70	94	NO	Induced	LN	Clear	GHTN	-	-	2.7kg	7	8	NO	NO	-
47	Mohanapriya	33	G3P2L2	37wks+3days	2	110/60	96	NO	-	REPEAT LSCS	Clear	GHTN	-	-	2.7kg	7	8	NO	YES	-
48	Kanaga	27	PRIMI	38wks	2	110/70	98	YES	Induced	LN	Clear	-	-	-	2.2kg	6	9	NO	NO	-
49	Preethi	32	PRIMI/BREECH	38+1day	2	120/70	114	NO	Spontaneous	LSCS	Clear	-	-	-	3kg	7	8	NO	NO	-
50	Jeeva	27	PRIMI	38wks+2days	2	120/70	108	NO	Induced	LN	Clear	-	-	-	2.9kg	6	8	NO	NO	-