

**“MATERNAL AND FETAL OUTCOME
OF PYREXIA IN PREGNANCY
BEYOND 28 WEEKS OF GESTATION”**

A prospective cohort study

Dissertation submitted

To

**THE TAMILNADU DR. M.G.R. MEDICAL
UNIVERSITY, CHENNAI**

*With partial fulfillment of the regulations
for the award of the degree of*

M.S (Obstetrics and Gynaecology)

Branch- II



**GOVERNMENT KILPAUK MEDICAL
COLLEGE CHENNAI**

April -2017

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled “ **MATERNAL AND FETAL OUTCOME OF PYREXIA IN PREGNANCY BEYOND 28 WEEKS OF GESTATION**” is the bonafide original work of **DR.SINDHUJA.R** under the guidance of **PROF DR. T.KRISHNAVENI MD., DGO.**, Department of Obstetrics and Gynaecology, KMCH, Chennai in partial fulfilment of the requirements for MS Obstetrics and Gynaecology branch II examination of the Tamilnadu Dr.MGR Medical university to be held in April 2017 . The period of Postgraduate study and training from June 2014 to April 2017.

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation title “ **Maternal and fetal outcome of pyrexia in pregnancy beyond 28 weeks of gestation**” is a bonafide, research work carried out by me, under the guidance of Prof.Dr.T.Krishnaveni MD DGO, Department of Obstetrics and Gynaecology, Kilpauk Medical College, Chennai-10.

This dissertation is submitted to, **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI**, in partial fulfillment of the degree of M.S. Obstetrics and Gynaecology examination to be held in **April 2017**.

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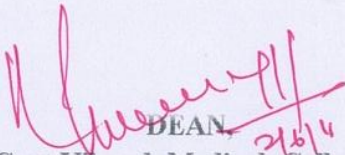
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The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval “**Maternal and fetal outcome of pyrexia pregnancy beyond 28 weeks of gestation**” - For Project Work submitted by **Dr.Sindhuja**, MS, Obstetrics & Gynaecology Govt. Kilpauk Medical College, Chennai – 10.

The Proposal is APPROVED.

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ACKNOWLEDGEMENT

I would like to thank God for the things he has bestowed upon me.

I would like to thank my parents for making me who I am today and for supporting me in every deed of mine.

I thank each and every person involved in making this manuscript from inception to publication.

I am most thankful to **Prof.DR.R.NARAYANA BABU M.D, DCH**, Dean, Kilpauk Medical College and Hospital for the opportunity to conduct this study in the Department of General Surgery, Government Kilpauk Medical College Hospital, Kilpauk Medical College, and Chennai-10.

My deepest gratitude to my guide and mentor **Prof.Dr.T.KRISHNAVENI MD DGO**, Professor, Department of Obstetrics and Gynaecology, Kilpauk Medical College, who has inspired me immeasurably during my training as a post graduate student.

I am very grateful to **Prof.Dr.T.K.SHAANTHY GUNASINGH, MD DGO**, Head of the department of Obstetrics and Gynaecology, Kilpauk Medical College, for her encouragement and unrestricted permission, to make use of Department of Obstetrics and Gynaecology.

I also acknowledge, invaluable advice and counseling received from, Dr. R.FATHIMAHASSAN MD DGO, Assistant professor and all assistant professors, Department of Obstetrics and Gynaecology, KMCH.

This study would have not been possible without the support of my fellow post graduates and interns who have been a source of help in need.

The most important part of any medical research is patients. I owe great deal of gratitude to each and every one of them.

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ABBREVIATIONS

LSCS	-	Lower segment cesarean section
PPH	-	Post partum haemorrhage
DIVC	-	Disseminated intra vascular coagulation
PROM	-	Pre labor rupture of membranes
PPROM	-	Preterm Pre labor rupture of membranes
LBW	-	Low birth weight
AFB	-	Acid fast bacilli
PUO	-	Pyrexia of unknown origin
AGE	-	Acute gastro enteritis
UTI	-	Urinary tract infection
RTI	-	Respiratory tract infection
Temp	-	Temperature
Hb	-	Haemoglobin
HIV	-	Human immunodeficiency virus
HSV	-	Herpes simplex virus
VZV	-	Varicella zoster virus
MRSA	-	Methicillin resistant Staphylococcus aureus.

CONTENTS

Sl.No	Title	Page No.
1.	INTRODUCTION	1
2.	REVIEW OF LITERATURE	4
3.	AIMS AND OBJECTIVES	50
4.	MATERIALS AND METHODS	52
5.	STATISTICAL ANALYSIS	54
6.	OBSERVATION AND RESULTS	55
7.	RESULTS	76
7.	DISCUSSION	81
8.	CONCLUSION	85
9.	BIBILIOGRAPHY	
10.	ANNEXURES	
	1.MASTER CHART	
	2.PROFORMA	
	3.CONSENT FORM	

INTRODUCTION

Pyrexia in pregnancy is a very common clinical entity worldwide. Fever during pregnancy causes significant maternal and fetal complications. Any acute or chronic infectious diseases may be aggravated during the period of pregnancy. Altered or compromised functions of immune system may predispose to several infections. Restrictions of antibiotics due to teratogenicity preclude the infection control. Anatomical and physiological changes occurs during pregnancy may predispose certain infections, for example, the urinary tract infections.

Some infections are affecting the mother and also may be transmitted to the fetus in utero. The effect of fever during pregnancy depends on the level of temperature rise, duration and the stage of fetal development. Some febrile diseases will lead to more severe and life threatening course in pregnancy and transplacental transmission leading to adverse fetal outcome. Pyrexia during pre-implantation, embryonic and fetal development period, may result in miscarriage, growth restriction, preterm labor and still birth.

Protein synthesis was interfered by hyperthermia via heat-shock proteins, S-phase cell death is induced and delay in mitotic activity M

phase. Vascular disruption and placental infarction also can happen. Ultimately it will lead to lethal malformations and fetal death. Furthermore, uterine contractility will be increased by pyrexia can lead to expulsion of the fetus at a non-viable stage of gestation. The hyperthermia induced fetomaternal outcome will differ according to the gestational time of exposure.

Fetus being an integral part of the fetomaternal unit and pregnancy involving numerous physiological changes and adaptations, pyrexia during the pregnancy affects both the mother and her fetus adversely. Normally during intrauterine life, the temperature of fetus is maintained by utero-placental circulation and heat-exchange at the amniotic fluid interface. Pyrexia effect on pregnancy depends on the extent of the rise in the temperature.

Because of maternal pyrexia, various inflammatory mediators as evidenced by umbilical cord blood cytokines is documented in the absence of neonatal sepsis (5).The underlying Maternal cytokine polymorphism is strongly associated with both intra partum fever & cerebral palsy at term.(6,7)

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 (8, 9).

The study was undertaken with the specific objective to assess the maternal and fetal complications due to pyrexia in pregnancy and also to find the different etiology of pyrexia in pregnancy.

REVIEW OF LITERATURE

Definition of fever

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

Body temperature elevation that exceeds the normal daily variation and occurs in conjunction with an increase in the hypothalamic set point is fever. Neurons in the vasomotor centre are activated and vasoconstriction will be commenced after the hypothalamic set point is raised. Shunting of blood away from the periphery to the internal organs essentially decreases heat loss from skin and the patient first notices vasoconstriction in the hands and feet.

The processes of heat conservation and heat production continue until the temperature of blood bathing the hypothalamic neurons matches the new thermostat setting. After that point is achieved the hypothalamus is maintaining the temperature at the febrile level by the same mechanism of heat balance that function in the afebrile state. Sweating and vasodilation will lead to loss of heat, continues until the blood temperature at the hypothalamic level matches the lower setting.

HYPERPYREXIA

Hyperpyrexia means a fever of $>41.5^{\circ}\text{C}$ ($>106.7^{\circ}\text{F}$) . This high fever can develop in severe infections and central nervous system (CNS) haemorrhage.

Although most patients with elevated body temperature have fever, there are circumstances in which elevated temperature represents not fever but hyperthermia (heat stroke). In hyperthermia, an uncontrolled increase in body temperature that exceeds the body's ability to lose heat. The setting of thermoregulatory center in hypothalamus is unchanged. In hyperthermia pyrogenic molecules are not involving in increasing body temperature. There are two mechanisms by which hyperthermia can result in dangerously high internal temperatures .Those are endogenous heat production and exogenous heat exposure. Despite physiologic and behavioral control of body temperature excessive heat production may cause hyperthermia.

Fever and hyperthermia should be distinguished, since hyperthermia can be rapidly fatal and characteristically does not respond to antipyretics. In an emergency situation, however making this distinction can be difficult. In systemic sepsis, temperatures can exceed 40.5°C (104.9°F).

The skin will be cold in fever due to peripheral vasoconstriction but in hyperthermia skin will be extremely hot due impairment of heat losing mechanisms. Adequate doses of aspirin or acetaminophen decrease the body temperature in fever, even in hyperthermia they decrease the temperature.

PYROGENS

Any substance that causes fever is called as pyrogens. They are broadly divided into exogenous and endogenous pyrogens. Exogenous pyrogens are mostly microbial products, microbial toxins, or that microorganism itself. Lipopolysaccharide (endotoxin) produced by all gram-negative bacteria is an example of exogenous pyrogen.

A group of antigens derived from gram positive bacteria are called as superantigens. They are enterotoxins of staphylococcus aureus and the groups A and B streptococcal toxins. Endotoxin is a highly pyrogenic molecule. Even a small dose 2–3 ng/kg administration in humans will lead to fever, leukocytosis, release of acute-phase proteins and generalized symptoms of malaise.

PYROGENIC CYTOKINES

Cytokines are proteins with small molecular weight (molecular mass, 10,000–20,000 Da). They mainly regulate the immune,

inflammatory, and hematopoietic processes in human body. For example, in several infections, the leukocytosis and absolute neutrophilia is due to the cytokines interleukin (IL)1 and IL-6. Some cytokines also cause fever. They are called as pyrogenic cytokines. These include IL-1, IL-6, tumor necrosis factor (TNF), ciliary neurotropic factor, a member of the IL-6 family, interferons (IFNs) mainly IFN- α .

Most of the bacterial and fungal products induce the synthesis and release of pyrogenic cytokines.

Non infectious conditions also cause fever in many circumstances. For example, inflammatory processes, trauma, tissue necrosis, and antigen-antibody complexes induce the production of IL-1, TNF.

The prostaglandin E2 (PGE2) levels are elevated in hypothalamic tissue and the third cerebral ventricle during fever. Near the circumventricular vascular organs (organum vasculosum of lamina terminalis)- (PGE2) levels are very high. Damage to these organs reduces the ability of pyrogens to produce fever. Thus interaction of exogenous pyrogens and pyrogenic cytokines with the endothelium of these capillaries is the first step in initiating fever.

Pyrogenic cytokines are produced by myeloid and endothelial cells. IL-1, IL-6, and TNF are released from these cells then enter the systemic circulation.

These cytokines elevate the level of PGE₂ in central nervous system near hypothalamus as well as in peripheral tissues. As PGE₂ level in hypothalamus account for rise in temperature, peripheral rise is the reason for constitutional symptoms of fever like myalgia.

Raising the hypothalamic set point for core temperature is the initial step of fever which will be initiated by PGE₂.

These PGE₂ acting via four different receptors in the body, among these receptors only the third (EP-3) is essential for fever: PGE₂ is not working exactly as a neurotransmitter but releasing of PGE₂ in hypothalamus will activate EP-3 on glial side and this stimulation results in the rapid release of cyclic adenosine 5'-monophosphate (cAMP), which is a neurotransmitter.

Ultimately these changes in glial cells will lead to activation of neuronal endings from the thermo regulatory centre. Either directly or indirectly increased cAMP level will lead to change in set point of hypothalamic set point. Hypothalamic endothelium is having separate receptors for microbial products also. These receptors are named as toll-

like receptors which are similar in many ways to IL-1 receptors. IL-1 receptors and Toll-like receptors are sharing the same signal transducing mechanism. Activation of either of these receptors causes fever.

PREGNANCY

The immune system of an individual may be innate or acquired. Innate immunity is determined by genetic and constitutional features of body, and is not related to immune system or antibodies. There are physiological, biochemical or anatomical differences between species regarding immune system.

Antigen stimulation will lead to production of IgM initially, followed by production of IgG in later stages. This pattern of response is not same in neonate; IgM is produced as a first response which is persisting for several weeks before the release of IgG. Detection of IgM in the umbilical cord blood indicates the in utero exposure of antigen mostly any congenital infections.

Passive immunity acquired by fetus from passage of IgG antibodies via placenta and IgA antibodies ingestion by colostrum. This passive immunity also can be acquired artificially by injection of immune products such as antitoxins, antisera, or immune globulin.

Proliferation of somatic cells is adapted to the normal body temperature range of the species. Increase in basal body temperature adversely affects the mitotic and meiotic proliferation of cells. In the same way embryonic death, abortion, growth retardation, and defects of development are the consequences of hyperthermic episodes during pregnancy.

In some studies it has been proposed that complications following a maternal fever is due to metabolic changes in the mother because of infection, and not due to the associated elevation of temperature.

The Collaborative Perinatal Study of the National Institute of the Neurological Diseases and Stroke found that children of mothers who presented with kidney-urinary tract infections (UTIs) and fever were approximately twice as likely to have neurological problems. And they require institutional care as children whose mothers had the only infection without any episodes of fever.

The association between maternal influenza and fever in the second trimester of pregnancy and the later onset of schizophrenia in the offspring showing that hyperthermia being the important initiating factors for this condition. And also other neurological conditions of uncertain etiology such as cerebral palsy and autism.

In some patients leukocytic invasion of the chorioamnion (chorioamnionitis) and umbilical cord (funisitis) showing the immune responses of mother and fetus respectively. There are numerous insults like hypoxic injury, trauma, meconium or allergens, for the immune response as mentioned above. The most common is the immune response to subclinical or clinical infection.

There are several ways in which maternal infection might lead to inflammation within the fetal tissue. Bacterial products cross the placenta and bind with cell-membrane receptors like CD-14 and toll-like receptors. They initiate a cascade of intracellular events in inflammatory cells. They further activate nuclear factor κ -B and production of pro-inflammatory cytokines. These pro-inflammatory cytokines are granulocyte colony-stimulating factor, tumor necrosis factor- α , interleukin-1 β , C-reactive protein and Interferon γ . They cause direct toxic effect on neurons mainly on oligodendrocyte precursor populations lead to gliosis with release of nitric oxide, dysfunction of mitochondria and microglial activation in the brain.

Not only maternal pyrexia due to infective cause and also non-infective cause like epidural anaesthesia increase the deleterious effects of hypoxia in fetal brain. It happens mainly due to increase in metabolic rate and demand for oxygen in brain.

Systemic hypotension, endothelial injury and leukocyte aggregation in fetus may contribute to local tissue ischemia, mainly in vulnerable areas. Most of these mechanisms ultimately result in cell death.

Mode of transmission of infectious agents include via umbilical blood flow or by direct amniotic fluid spread, cervical ascending transmission ,intra partum exposure to maternal secretions of vagina and blood, or by postpartum exposure to maternal respiratory secretions or breast milk.

Pregnancy is a state of immunosuppression. In pregnancy among CD4 -positive T cells Th1 type cells are decreased in numbers and Th2 type cells are increased. Therefore Th2 secreting cytokines levels are high and they are interleukins 4, 5, 10, and 13.

Th1-type cells producing cytokines are interferon gamma and interleukin 2-appears to be decreased in circulation, leading to a Th2 bias. Rapid clearance of some microbial organisms depend upon the Th1 type cells producing cytokines, so this function is impaired in pregnancy. Importantly, the Th2 humoral immune response remains intact.

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

Urinary tract infections (UTIs) are common during pregnancy. There are several factors which predispose this condition .They include increased volume of bladder, decreased tone of bladder along with decreased tone of urethra leading to stasis of urine and ureterovesical reflux. Urinary progesterins and estrogens are increased which may lead to a decreased resistance for invading bacteria in the lower urinary tract.

In various studies prevalence of UTI in pregnancy varies between 4%

and 7%. Among these patients only 1% -2% of pregnant women with UTI are symptomatic. Most of the women acquire bacteriuria before pregnancy.

At the first examination, the rates of bacteriuria in pregnant women are similar to those in non-pregnant women with similar risk factors. About 37% - 57% of bacteriuric women develop UTIs during pregnancy with an additional 1% risk of infections in pregnancy. Bacteriuria in pregnancy is associated with many adverse fetal outcome which include preterm delivery ,neonatal mortality and low birth weight of babies.

Urinary tract infections in pregnancy mainly divided under three entities;

- 1) Asymptomatic bacteriuria,
- 2) Acute cystitis
- 3) Acute pyelonephritis.

Along with other symptoms like dysuria, increased frequency, urinary tract infection is a common cause of fever in pregnant women .

The diagnosis of UTI is based two factors

- 1) Clinical findings and
- 2) More than 100000 colony forming U/mL in urinary specimen.

The most common organisms causing UTI are gastrointestinal organisms mainly E.coli. Re infection of the urinary tract is more common even with proper treatment because of the rectal reservoir. There is no difference between the pregnant and non pregnant women among infective organism of UTI

The organisms are

- a) E.coli(most common 80-90%)
- b) *Proteus mirabilis*
- c) *Klebsiella pneumonia* .
- d) *Enterococci*
- e) *Gardenella vaginalis*
- f) Gram-positive organisms such as group B

Streptococcus and *Staphylococcus saprophyticus* are less common.

Of all the risk factors for UTI in pregnancy mentioned above already, urethral catheterization may be foremost reason for nosocomial urinary tract infections.

Acute pyelonephritis

It is a serious systemic illness that can evolve into sepsis in pregnant woman mainly occurring during the later stages of pregnancy, mainly third trimester. Various studies showing that incidence is 2% for pyelonephritis in pregnant women. The diagnosis of pyelonephritis is mainly depends on the clinical symptoms and culture of the urine specimen.

Systemic symptoms are more common like fever, chills, nausea, vomiting, and flank pain. Symptoms of lower tract infection like increased frequency and dysuria may or may not be present.

There should be concern about two main issues in pyelonephritis

- 1) Presence of abnormalities of urinary tract
- 2) Associated risks of the mother and fetus, such as toxemia, hypertension, prematurity and perinatal mortality.

Early, aggressive management with appropriate antibiotics like second- or third-generation cephalosporins, an aminopenicillin, or an aminoglycoside is important in resolving the infection as well as decreasing the chance of complications from pyelonephritis. Hospitalization may or may not necessary depending upon the condition

of the patient. However, mainly hospitalization is indicated for patients who are presenting with signs of sepsis, who are unable to accept oral food and who are all in moderate to severe dehydration. The duration of parenteral antibiotic treatment in pyelonephritis should be extended until the patient become afebrile.

In the initial stage of treatment most of the patients respond to hydration and prompt antibiotic treatment within 24 to 48 hours. There are many cases with treatment failure even with aggressive treatment. The main reason is resistance of the infecting organism for the particular antibiotic can be overcome by culture and sensitivity of specimen. If the patient's fever and systemic illness remain after appropriate antibiotic therapy, we should suspect underlying anatomic abnormality in urinary tract. Urolithiasis may be the cause of persistent infection, which occurs in 1 in 1500 pregnant ladies or less commonly.

Congenital renal anomalies, perinephric abscess, ureterovesical reflux may also occur. Increases in urinary progesterins and estrogens will lead to inability of the lower urinary tract to resist the invading microorganism. Of all risk factors, urethral catheterization contributes the most to the incidence of nosocomial urinary tract infections.

Asymptomatic Bacteriuria

There are lot of general measures to improve this condition which include plenty of oral fluids to increase urine output ,oral treatment with potassium citrate solution to alkalinize urine and treatment with cranberry juice also improve symptoms which is unproven.

The definition of asymptomatic bacteriuria is two consecutive positive cultures of the same species. The general measures to treat UTI include antibiotics, drinking more fluid. The pregnant women should be screened for bacteriuria by urine culture at least once in early pregnancy, and it should be treated accordingly. Antibiotic sensitivity testing based treatment and usual course of treatment is mainly up to 5-7 days. After 1- 4 weeks of treatment follow up cultures should be obtained and at least once before delivery. Upto 30% of patients will present with persistence of bacteriuria if left untreated.

According to cochrane systematic review if asymptomatic bacteriuria is treated timely in a pregnant women ,it will decrease the risk of developing pyelonephritis in future and also reducing the risk of preterm delivery.

Conventional antibiotic treatment is always superior than single-dose therapy in asymptomatic bacteriuria. Commonly using drugs which are consider safe and effective in pregnancy:β-lactam antibiotics, including amoxicillin and cephalosporins, and nitrofurantoin.

PNEUMONIA

Pneumonia is the most frequent cause of non-obstetric infection in the pregnant patient. There is no difference in incidence of pneumonia in pregnant and non-pregnant adults. The incidence is 1.1 to 2.7 per 1,000 deliveries. Pneumonia is very infrequent in the setting of pregnancy. But pneumonia may lead to many serious complications of pregnancy.

The different implications for the pregnant woman depends on the etiology and type of pneumonia. In pregnant lady with predisposing factors such as history of smoking or respiratory disease the incidence of pneumonia is particularly high. As similar like other infectious disease in pregnancy, pneumonia can have an impact on the mother and fetus, contributing to morbidity and mortality when compared to the nonpregnant women.

There are many anatomic changes occur during pregnancy;

Enlargement of uterus causing the diaphragmatic elevation, and enlargement of the chest dimension particularly transverse diameter. Collectively, all these alterations lead to decrease the ability of the pregnant woman for clearing the respiratory secretions and exogenous agents. Because of these predisposing factors it is very important to identify tachypnea which may be a only finding in pneumonia or other

pathological condition. Risk factors for maternal pneumonia include HIV, sickle cell disease, cystic fibrosis, antepartum systemic corticosteroids, asthma and anaemia. Mother's age and parity are not the risk factors for increased incidence of pneumonia in pregnant women. Incidence of pneumonia is around 50%- 80% in third trimester in different studies. The symptoms of acute bacterial pneumonia are mainly cough, fever, dyspnea, chills and sputum production both in pregnant and non pregnant women.

Both pregnant and non pregnant women mortality due to pneumonia is almost same. But maternal complications are increased during pregnancy.

Respiratory failure and Mechanical ventilation are major complications documented in pregnancy. Regarding fetal outcome in maternal pneumonia, premature delivery and low birth weight was frequently documented. The main underlying pathological cascade in pneumonia is active host inflammatory mediators in response to infection in lungs exerts effects on uterus and growing fetus. It will result in preterm labour. The incidence range of 1.9% to 12% in neonatal mortality noticed in different series of studies, But anomaly in newborn is not associated with antepartum maternal pneumonia according to many

studies. Pneumonia also cause maternal complications like anemia, empyema, bacteremia, and death.

The individual pathogens causing pneumonia in the pregnant patient is almost similar to those reported in non pregnant hosts of similar age group. Prevalence of some infectious agents are more common in pregnancy .They cause a major hazard to the pregnant women compared to non pregnant women because of suppressed cell mediated immunity in pregnancy.

The most common pathogens in pregnancy associated pneumonia *Streptococcus pneumoniae*, *Haemophilus Influenza*, *Legionella pneumonia* Fungal pneumonia in pregnancy are rare.

But fungal infections like *Cryptococcus neoformans*, *Histoplasma capsulatum*, *Sporothrix shenkii*, *Blastomyces dermatitidis* can cause pneumonia that is usually mild and self-limited disease.

VARICELLA

It is caused by Varicella zoster virus. Varicella presents with a 1- to 2-day flu-like prodrome, which is followed by pruritic vesicular lesions that crust over in 3 to 7 days. Adults are presenting with severe symptoms, and a quarter of varicella deaths are within 5 percent of non immune patients (Centers for Disease Control and Prevention, 2007).

Mortality is predominately due to varicella pneumonia, which is thought to be more severe during adulthood and particularly in pregnancy. Between 5 and 20 percent of infected pregnant women developed pneumonitis (Centers for Disease Control and Prevention, 2007; Harger, 2002). Predisposing factor for VZV pneumonia include smoking and having more than 100 cutaneous lesions. Maternal mortality rates with pneumonia have decreased to 1 to 2 percent (Chandra et al, 1998). Symptoms of pneumonia usually appear 3 to 5 days into the course of illness. Symptoms include fever, tachypnea, dry cough, shortness of breath, and pleuritic pain. Nodular infiltrates are similar to other viral pneumonias .Although resolution of pneumonitis parallels that of skin lesions, fever and compromised pulmonary function may persist for weeks. If reactivation of primary varicella infection occurs after some years , it causes herpes zoster or shingles (Whitley et al, 2012). The clinical presentation will be unilateral dermatomal vesicular eruption with severe pain. Zoster is not frequent or severe in pregnancy. Enders et al and associates (1994) reviewed 366 cases during pregnancy and found little evidence that zoster causes congenital malformations. Zoster is contagious if blisters are broken, although less than primary varicella infection.

In women with chicken pox during the first half of pregnancy, the fetus may develop a special form manifestation called as congenital varicella syndrome. Some features include chorioretinitis, microphthalmia, cerebral cortical atrophy, growth restriction, hydronephrosis, limb hypoplasia, and cicatricial skin lesions. Enders and coworkers (1994) evaluated 1373 pregnant women with varicella infection. If maternal varicella infection occurs before 13 weeks of pregnancy, only 0.4 percent incidence of neonates get congenital varicella. The maximum risk is when the infection is occurring between 13 and 20 weeks. After 20 weeks' gestation, the researchers found no clinical evidence of congenital infection. Thus, congenital infections, particularly after 20 weeks, are uncommon. Subsequent sporadic reports have described central nervous system abnormalities and skin lesions in fetuses who developed congenital varicella in weeks 21 to 28 of gestation (Lamont et al, 2011; Marin et al, 2007). Bone defects and atrophy in lower limbs and scarring in fetus occurs when the mother gets infected during the first trimester.

RUBEOLA (MEASLES)

Measles is caused by a highly contagious RNA virus of the family Paramyxoviridae that only infects humans. Annual outbreaks occur in late winter and early spring. Transmission is primarily by droplets in the air,

and the secondary attack rate among contacts exceeds 90 percent (Moss, 2012). Infection is characterized by fever, coryza, conjunctivitis, and cough. Erythematous maculopapular rash develops on the face and neck and then it will spread to the back, trunk, and upper and lower limbs. Koplik spots are small white lesions with surrounding erythema found within the oral cavity. Diagnosis is most commonly performed by serology, although RT-PCR tests are available. Treatment is supportive. Pregnant women without evidence of measles immunity should be administered intravenous immune globulin (IVIG), 400 mg/kg within six days exposure of measles (McLean et al, 2013). Active vaccination is contraindicated during pregnancy. However, in postpartum period vaccination can be done, and breast feeding is not contraindicated (Ohji et al, 2009). There is no proven teratogenicity with the virus exposure (Siegel et al, 1973). However, incidence of abortion , preterm delivery, and low-birth weight are noted with maternal measles (American Academy of Pediatrics, 2006; Siegel, 1966). Neonatal infection is more common if pregnant women develop measles shortly before birth especially preterm neonates.

PARVOVIRUS

The prevalence of asymptomatic infection is almost 20-30 percent. In the last few days of the viremic phase patient may present with fever,

headache, flu like symptoms. after several days, slapped-cheek appearance will be present which is a bright red rash with erythroderma affects the face. Milder rashes and symmetrical polyarthralgia are present in adults for many weeks. There are adverse effect of parvovirus infection on pregnancy (Valeur-Jensen et al, 1999). During recovery, there is IgM antibody production for 7 to 10 days post infection, and this persists for 3 to 4 months. Several days after IgM is produced, IgG antibody is detectable and persists for life with natural immunity.

CYTOMEGALO VIRUS

Pregnancy does not increase the risk or severity of maternal CMV infection. Asymptomatic infection is most common, but 10 to 15 percent of infected adults will present with mononucleosis-like syndrome which consists of low grade fever, pharyngitis, significant lymph adenopathy, and polyarthrititis involving small and large joints. Immunocompromised women may develop myocarditis, pneumonitis, hepatitis, retinitis, gastroenteritis, or meningoencephalitis. In one study most women with primary infection of CMV are showing increased serum aminotransferases level. Reactivation disease usually is asymptomatic, although viral shedding is common. Upto 40 percent of patients infected with CMV are transmitting the infection to the fetus and causing severe morbidity. In contrast, recurrent maternal infection infects the fetus in

only 0.15 to 1 percent of cases. A review of nine studies of CMV vertical transmission rates reported first-trimester transmission in 36 percent, second-trimester in 40 percent, and third-trimester in 65 percent (Picone et al, 2013). Acquired immunity during pregnancy because of previous CMV infection results in a 70-percent risk reduction of congenital CMV infection in subsequent pregnancies (Fowler et al, 2003). However, maternal immunity does not prevent recurrences, and maternal antibodies do not prevent fetal infection. Sometimes different viral strain can cause fetal infection even in sero positive women (Ross et al, 2011). When a newborn has apparent sequelae of in utero-acquired CMV infection, it is referred to as symptomatic CMV infection. Congenital infection is a syndrome that includes growth restriction, microcephaly, intracranial calcifications, chorioretinitis, mental and motor retardation, sensorineural deficits, hepatosplenomegaly, jaundice, hemolytic anemia, and thrombocytopenic purpura . The pathogenesis of these outcomes has been reviewed by Cheeran et al and colleagues (2009). Among the 40,000 CMV infected neonates born every year, only five to ten percent showing this syndrome (Fowler et al, 1992). Thus, most infected infants are asymptomatic at birth, but some develop late-onset sequelae. It include hearing loss, neurological deficits, chorioretinitis, psychomotor retardation, and learning disabilities.

BACTERIAL INFECTIONS

Group A Streptococcus

Infections caused by *Streptococcus pyogenes* are important in pregnant women. It is the main bacterial infection that causes acute pharyngitis and is usually associated with several systemic and cutaneous infections. *S. pyogenes* produces numerous toxins and enzymes responsible for the local and systemic toxicity. Pyrogenic exotoxin-producing strains are usually associated with severe disease (Mason et al, 2012; Wessels et al, 2012). In most cases, streptococcal pharyngitis, scarlet fever, and erysipelas are not life threatening. Treatment, usually with penicillin, is similar in pregnant and nonpregnant women (Shulman et al, 2012). In the United States, *Streptococcus pyogenes* infrequently causes puerperal infection. The incidence of these infections is increasing (Deutscher et al, 2011; Hamilton et al, 2013; Mason et al, 2012; Wessels et al, 2012). The early 1990s saw the rise of streptococcal toxic shock syndrome, manifested by hypotension, fever, and evidence of multiorgan failure with associated bacteremia. The case-fatality rate approximates 30 percent, and morbidity and mortality rates are improved with early recognition. Treatment includes clindamycin or penicillin therapy and often surgical debridement (Hamilton et al, 2013). No vaccine for group A streptococcus is commercially available. Group B *Streptococcus*

Streptococcus agalactiae is a group B organism that can be found to colonize the gastrointestinal and genitourinary tract in 20 to 30 percent of pregnant women. Throughout pregnancy, group B *Streptococcus* (GBS) is isolated in a transient, intermittent, or chronic fashion. The extent of clinical manifestation with maternal and fetal GBS ranges from only colonization without symptoms to septicemia. *Streptococcus agalactiae* is showing many adverse pregnancy outcomes which include including preterm labor, premature rupture of membranes, clinical and subclinical chorioamnionitis, and fetal infections.

Maternal bacteriuria, pyelonephritis, osteomyelitis, mastitis, and puerperal infections are other manifestation in pregnant women by GBS.

MRSA AND PREGNANCY

Anovaginal colonization with *Staphylococcus aureus* is identified in 10 to 25 percent of obstetrical patients (Andrews et al, 2008; Creech et al, 2010; Top, 2010). MRSA has been isolated in 0.5 to 3.5 percent of these women. Skin and soft tissue infections are the most frequent presentation of MRSA in pregnancy. Mastitis has been reported in MRSA complicating pregnancy (Laibl et al, 2005; Lee et al, 2010; Reddy et al, 2007). Perineal abscesses, wound infections at sites such as abdominal and episiotomy incisions, and chorioamnionitis are also associated with

MRSA (Lareau et al, 2010; Pimentel et al, 2009; Rotas et al, 2007; Thurman et al, 2008). An increase in CA-MRSA infections has been reported in neonatal intensive care units and newborn nurseries. In these settings, infection is frequently associated with maternal and health-care worker MRSA skin infections and infected breast milk.

VULVAR ABSCESS

Labia majora infections, which begin as cellulitis, have the potential for significant expansion and abscess formation. Risk factors include diabetes, obesity, perineal shaving, and immunosuppression. For early cellulitis, sitz baths and oral antibiotics are reasonable treatment. If present, a small abscess can be incised and drained, wound cultures obtained, abscess cavity packed, and surrounding cellulitis treated with oral antibiotics. These infections are typically polymicrobial, and suitable broad-spectrum antimicrobials are given along with coverage for MRSA (Thurman et al, 2008). For severe infections, especially in immunosuppressed or pregnant patients, hospitalization and intravenous antimicrobial therapy are often warranted due to increased risks for necrotizing fasciitis. Large abscesses are best drained in the operating room with adequate analgesia or anesthesia. Bartholin gland cysts are unilateral, sterile, and there is no need of any active treatment during pregnancy. If a cyst is sufficiently large to obstruct delivery, then needle

aspiration is an appropriate temporary measure. With gland duct infection, a localized unilateral vulvar bulge, tenderness, and erythema are present. Treatment is given with broad-spectrum antimicrobials, and drainage is established. In addition to obtaining wound cultures, testing is done for *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. For small abscess, incision and placement of a catheter may be suitable. For larger abscesses with extensive cellulitis, incision and drainage followed by antibiotic treatment is needed. In these cases, incision and drainage is followed by marsupialization of incised edges. Occasionally, abscesses of the periurethral glands develop. The largest of these, the skene gland, may require drainage and broad-spectrum antimicrobial treatment if there is suppuration.

LISTERIOSIS

Listeria monocytogenes is mostly an uncommon but probably underdiagnosed cause of neonatal sepsis. This facultative intracellular gram-positive bacillus can be isolated from the feces of one to five percent of adults. Nearly all cases of listeriosis are thought to be food-borne (Silk et al, 2013). Outbreaks have been caused by raw vegetables, coleslaw, apple cider, melons, milk, fresh Mexican-style cheese, smoked fish, and processed foods, such as pâté, hummus, wieners, and sliced deli meats (Cartwright et al, 2013; Centers for Disease Control and

Prevention, 2013i; Janakiraman et al, 2008; Varma et al, 2007; Voetsch et al, 2007). The old age, pregnant and immunocompromised patients are main groups vulnerable for listeria infections. The incidence of such infections appears to be increasing in several countries worldwide (Allenberger et al, 2010; Cartwright et al, 2013; Goulet et al, 2012). In 1651 cases reported in 2009 to 2011, the Centers for Disease Control and Prevention found that fourteen percent patient were pregnant women (Silk, 2013). And in a recent review, pregnant women had a significantly higher rate for listeriosis compared with nonpregnant reproductive-aged women (Pouillot et al, 2012). It is unclear why pregnant women still account for a significant number of these reported cases. Because of suppressed cell-mediated immunity in pregnant women, they are more prone for listeria infections , (Baud, 2011; Jamieson, 2006b; Wing, 2002). Another is that placental trophoblasts are susceptible to invasion by *Listeria monocytogenes* (Le Monnier et al, 2007).

SALMONELLA

Salmonella species are major causes of food-borne illness (Peques et al, 2012). Six serotypes account for most cases in the United States, including Salmonella subtypes typhimurium and enteritidis. Non-typhoid Salmonella gastroenteritis is transmitted through food contamination. Symptoms includes non bloody diarrhea, abdominal pain, fever, chills,

nausea, and vomiting begin 6 to 48 hours after exposure. Diagnosis is made by stool studies. Rare case reports have linked Salmonella bacteremia with abortion.

SHIGELLOSIS BACILLARY

Shigella induced dysentery is not uncommon and also highly contagious cause of inflammatory diarrhea which is exudative in adults. In day-care centers shigellosis is common among children and is transmitted via the fecal-oral route. The extent of clinical manifestation from mild diarrhea to severe dysentery, bloody stools, abdominal cramping, tenesmus, fever, and systemic toxicity.

MALARIA

Malaria is a mosquito borne infectious disease caused by the parasite protozoans belonging to Plasmodium species. 4 types of species affects the humans includes Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale and Plasmodium malariae. The disease is transmitted by an infected female Anopheles mosquito. The symptoms include fever, fatigue, vomiting, headache, seizures, coma and death .It is diagnosed by the microscopic examination of blood using blood films or with antigen based diagnostic tests.

Malaria in pregnancy affects both the mother and fetus adversely. The physiological changes of pregnancy and pathological changes due to malaria have an adverse effect on the course of each other. Local production of chemokines leads to monocyte accumulation in placenta. These pigment contained placental monocytes are associated with maternal anaemia and fetal low birth weight. It also leads to preterm labour. Antenatal women with plasmodium falciparum infections are more prone to get high levels of parasitemia, hypoglycaemia ,preterm labour, fetal distress, spontaneous abortions, still birth, acute pulmonary edema and cerebral malaria. Therefore all patients with fever should be screened for malarial parasites.

DENGUE FEVER

Dengue fever is caused by dengue virus, a mosquito borne tropical disease. It is transmitted by Aedes aegypti mosquito. The symptoms were high fever ,vomiting, headache, muscle pain, joint pain and a characteristic skin rash. In some patients the disease had a severe course and develops in to dengue haemorrhagic fever and dengue shock syndrome resulting in bleeding, low platelet count, plasma leakage and low blood pressure.

Dengue fever has 3 stages- febrile phase, critical phase and recovery phase. Dengue fever during pregnancy had adverse maternal and fetal complications. The most common perinatal outcome was low birth weight and preterm labour. The immune response to dengue infection promotes preterm labour by inducing placental inflammation and trophoblast apoptosis, producing inflammatory cytokines and chemokines. The risk of vertical transmission is well established among women with dengue infection during the perinatal period. Termination of pregnancy was not indicated in dengue infection. During parturition severe bleeding may occur during delivery or surgical procedures in patients with thrombocytopenia or plasma leak. Blood and blood products should be transfused immediately to save the life of the woman. Neonates born to women who had dengue infection at term or just before or during delivery should be closely monitored for the risk of vertical transmission.

TUBERCULOSIS

It is an infectious disease caused by *Mycobacterium tuberculosis*. It mainly affects the lungs but also affects the different parts of the body. The symptoms include chronic cough with blood stained sputum, fever, night sweats and weight loss. The incidence of tuberculosis among the pregnant women would be high as in the general population. The study by Schaefer et al reported a case rate of 18-29/100,000 in pregnancy and

a recent UK study reported an incidence of 4.2 per 100,000 pregnant women. Tuberculosis is one of the leading cause of death among the pregnant women aged 15-45 years.

The obstetric and neonatal complications includes spontaneous abortions, suboptimal weight gain, small for date uterus, preterm labour, low birth weight and increased neonatal mortality. Congenital tuberculosis is a very rare complication of in utero infection. It is due to haematogenous spread through the umbilical vein to the fetal liver or by ingestion and aspiration of infected amniotic fluid. The primary focus develops in the liver with the involvement of peri portal lymph nodes. Secondly the bacilli infects the lungs. The diagnosis of neonatal tuberculosis includes demonstration of primary hepatic complex or caseating granuloma on percutaneous liver biopsy, tuberculous infection of the placenta or maternal genital tuberculosis.

Treatment is achieved through Directly Observed Short Course (DOTS). The combination therapy includes isoniazide and rifampicin compulsorily supported by ethambutol and pyrazinamide for at least 6 months. RNTCP recommends breast-feeding of neonates regardless of the mothers TB status.

INFLUENZA

Pregnant women with influenza are more likely to develop severe illness and to die than the general population, based on data from seasonal influenza and from the influenza pandemics of 1918 to 1919, 1957 to 1958, and 2009 to 2010. The increased severity of influenza in pregnant women is thought to be related to normal physiologic changes that occur during pregnancy. For example, heart rate and oxygen consumption increase, lung capacity decreases, and there is a shift away from cell-mediated immunity.

Because of the increased severity of influenza in pregnancy, inactivated influenza vaccine is recommended for pregnant women, regardless of trimester of pregnancy. In addition, pregnant women with suspected or confirmed influenza should receive prompt empiric treatment with appropriate antiviral drugs.

Clinical manifestations of influenza in pregnant women are similar to those in the general population, and include fever, cough, rhinorrhea, sore throat, headache, shortness of breath, and myalgia. Increased rates of spontaneous miscarriage, low birthweight, preterm delivery and fetal death have been observed and reported, mainly during influenza pandemics.

Antigen detection-based rapid influenza diagnostic tests (RIDTs) are simple to perform and interpret, but have limited sensitivity and specificity in detecting influenza virus. Although viral culture remains the gold standard for diagnosis. Polymerase chain reactions are also used for diagnosis. Antiviral therapy for influenza virus infection should be initiated within 48 hours of symptom onset.

TYPHOID FEVER

It is a bacterial infection caused by *Salmonella typhi*. The symptoms includes gradual onset of fever for several days, weakness, abdominal pain, constipation, headache, vomiting. Some people may develop skin rash with rose coloured spots. Signs include hepatomegaly and splenomegaly. Incidence of typhoid was said to be more common in Indian subcontinent. It is a highly contagious disease. It is transmitted by food and water contaminated by the bacteria. Without treatment the symptoms get worse and they develop complications like bowel perforation and internal haemorrhage. A study has shown that complications such as gastrointestinal bleeding were more common in women who were not pregnant.

One study found 25 cases of typhoid infection in 15,940 births. Out of these 25 cases, there was one first trimester miscarriage, six pregnancy

losses (in second and third trimesters), six premature births and 12 normal births.

Another study compared 181 pregnant women who had typhoid, with randomly selected age-matched pregnant women without typhoid. This study found that there was no increased risk of premature birth or pregnancy complications because of typhoid. The study found no apparent effect of typhoid fever on pregnancy outcome, such as: gestational age at delivery, pregnancy complications, modes of delivery, birth weight, birth Apgar scores. The disease is diagnosed by blood culture and widal test. It is treated with antibiotics. There are two types of vaccines –live and injectable vaccine. Live vaccines are not recommended during pregnancy. The injectable vaccines is an inactivated vaccine which is considered safe during pregnancy.

VAGINAL INFECTIONS

Trichomonas vaginalis a sexually transmitted vaginal infection was associated with preterm delivery and low birth weight. The symptoms include itching, profuse watery vaginal discharge, vaginal irritation and odour. Bacterial vaginosis is not a sexually transmitted infection but it is more common in sexually active women. Many studies have shown an association between bacterial vaginosis and preterm birth, premature

rupture of membranes and low birth weight. Yeast infections like candidiasis are also more common during pregnancy. Yeast infections also causes premature rupture of membranes and low birth weight.

CHORIOAMNIONITIS

It is defined as inflammation or infection of the placenta and fetal membranes. It may be clinical, subclinical or histologic diagnosis. Clinical infections occurs in 1-2% of term and 5-10% of preterm deliveries. More than 5 vaginal examinations and internal fetal monitoring are independent risk factors.

It is commonly caused by poly microbial ascending vaginal infections. Most common organisms are Ureaplasma, Mycoplasma, gram negative bacilli such as Bacteroides, Coliforms, anaerobes.

It is clinically diagnosed by Maternal fever and one or two of the following symptoms: maternal tachycardia (>100 bpm), fetal tachycardia (>160 bpm), uterine tenderness, foul smelling amniotic fluid, or maternal leukocytosis ($>15,000$ with left shift). Fetal tachycardia is often the most sensitive additional clinical symptom indicating acute chorioamnionitis. Prompt diagnosis and administration of intrapartum antibiotics are important in reducing maternal and neonatal morbidity. Chorioamnionitis may lead to decreased uterine contractility and increased cesarean section

due to failure of progress of labor. Post partum haemorrhage are more common after delivery. Cesarean section increases risk of postpartum complications such as wound infection (10% incidence) and endomyometritis.

Neonatal Complications include increased risk of neonatal sepsis, pneumonia, respiratory distress and mortality. Increased risk of low Apgar scores, hypotension, neonatal seizures, need for resuscitation at delivery, interventricular hemorrhage, periventricular leukomalacia and cerebral palsy. Intrapartum antibiotics reduce risk of neonatal infection.

In April 2015, **Jhuma Biswas et al, Kaushik Banerjee et al, Poushali Sanya et al, Mousumi Datta et al, Subhendu Choudhury et al, Shyamal Dasgupta et al, Sayantane Sen Gupta et al, Amar Nath Dey et al** studied the **feto maternal outcome of pyrexia in pregnancy** at Kolkata. They studied 183 antenatal women with pyrexia. Maternal complications and fetal outcome like low birth weight, preterm labor, five minutes APGAR score less than 7, perinatal death was recorded. The maternal complications among the study group was compared according to their gestational age, age and parity. Results of this study –About 35% of the patients had malaria. It was the leading cause of fever and it had adverse maternal and fetal outcome. There was no statistical association between gestational age, parity, age and the maternal complications. The

etiology of fever in their study includes malaria (35%), respiratory tract infection, tuberculosis, viral hepatitis and urinary tract infections. The major maternal complications like diarrhea, hypoglycemia, pleural effusion, convulsion, pneumonia, severe anemia and acute renal failure. They reported that jaundice was the most common complication among the pregnant females. The other etiology of fever includes typhoid (7.1%), viral hepatitis(10.4%) and chicken pox (6%). Fetal outcome- About 58.8% of the babies had low birth weight. The mean birth weight was taken as 2.44 kg. The other fetal outcome were preterm labour (36%), intra uterine growth retardation (30%), APGAR score less than 7 minutes at birth(44%),and perinatal death(2.1%).According to the etiology of fever ,the adverse fetal outcome for that etiology was also studied.They reported that malaria causes more adverse maternal and fetal complications.Urinary tract infections causes more fetal complications than maternal complications. (10).

In November 2010 **Vilada Chansamouthet al, Syvilay Thammasacket al,Rattanaphone Phetsouvanh et al, ValyKeoluangkot et al, CatrinE.Moore et al** studied the etiologies and impact of fever in pregnant in patients in Vientiane,Laos. 250pregnantwomen were recruited in this study between February2006 and November 2010.The median gestational age on admission was24(4–

43) weeks. The median tympanic admission temperature was 38.5°Of these 250 women 149 (60%) had a laboratory diagnosis. Of these 149 patients 132 patients had a single disease and 17 patients had mixed infections. They reported that dengue fever was the most common cause of fever among their patients in that area (53%).The other causes of fever includes leptospirosis (0.4%), japanese encephalitis (0.4%),appendicitis (0.8%), pyelonephritis,scrub typhus, murine typhus, typhoid ,staphylococcus aureus septicaemia and plasmodium falciparum malaria.About 78% of women with dengue,rickettsia and typhoid had spontaneous abortions,preterm labour,still birth,low birth weight and maternal death as their complications. (50).

In 2013 Prabhat Agarwal et al, Sowmya Srivastava et al, Urvashi varma et al, Rekha rani et al studied the **Pregnancy outcome in women with dengue infection in northern India**. The study was conducted in SN medical college, Agra from July 2011 to December 2012.Retrospective analysis of pregnant women with dengue infection was conducted in 25 patients. Dengue infection was observed in 28% of primigravida and 72% of multigravida. Of these 25 patients 24% had dengue fever,56% had dengue haemorrhagic fever and 20% had dengue shock syndrome. 80% of patients was positive for IgM dengue serology,68% was positive for NS antigen and in 14% both tests were

positive. There was no mortality in patients infected with dengue in early pregnancy. Preterm delivery was observed in 68% of patients. 32% had abruption placenta, 32% had post partum haemorrhage, 52% had oligohydromnios. 52% of women had low birth weight. 5 babies died during their early neonatal period. There were 3 cases of maternal mortality. They concluded that dengue fever in pregnancy leads to adverse maternal and fetal outcome. Antenatal patients infected with dengue virus requires close observation for maternal and fetal complications. (22)

In 2014 Jayati Nath et al, Snehal Mahajan et al conducted A Clinical Study on Pyrexia in Pregnancy with Special Emphasis on Fetomaternal Outcome in the department of Obstetrics & Gynecology, Teerthanker medical college and research centre Moradabad, Uttar Pradesh. They studied 185 antenatal patients with pyrexia. The etiology of fever includes malaria (26%), dengue (21%), urinary tract infection (20%), tuberculosis (4%), respiratory tract infections (12.4%), chicken pox (2.8%), typhoid (7.0%) and PUO (Pyrexia of unknown origin) (3.2%). The various maternal complications reported in this study includes diarrhoea, acute gastro enteritis, hypoglycemia, bronchopneumonia, pleural effusion, acute renal failure, jaundice and convulsions. Adverse fetal outcomes encountered were low birth weight (52.6%), preterm

labor (27.8%), intra uterine growth retardation (IUGR) (20.3%), low APGAR score (< 7) at 5 minutes after birth (18.6%) perinatal mortality (5.0%), intrauterine fetal death (IUFD) (2.8%). The most common adverse effect was low birth weight. About 65% of deliveries had more than one complication. Urinary tract infections had more adverse fetal outcome. (12)

In 2010, **T.V. Chitra et al and Seetha Panicker et al studied the maternal and fetal outcome of dengue fever in pregnancy.** It was conducted at P.S.G institute of medical sciences and research, Coimbatore. They studied 14 pregnant women infected with dengue virus. The gestational age at which the infection occurs appears to be significant. The infection during first and third trimester had a bad prognosis. Maternal complications like dengue haemorrhagic fever, disseminated intravascular coagulation, pregnancy induced hypertension had occurred in 6 patients. In neonates there was no evidence of dengue infection. Two patients had severe thrombocytopenia at term. They were treated with blood and blood products before and after delivery.(14)

In April 2011 Vaishali Jain et al, Vinita Daset et al, Anjoo Agarwal et al & Amita Pandey et al studied asymptomatic bacteriuria & obstetric outcome following treatment in early versus late pregnancy in north Indian women. A Tertiary care teaching

hospital of North India conducted prospective cohort study. Antenatal women upto 20 week (n=371) and between 32 to 34 week gestation (n=274) having no urinary complaints were included. Asymptomatic bacteriuria was found in 17 % antenatal women till 20 weeks and in 16 % between 32 to 34 weeks gestation. Increased incidence of pre eclampsia, preterm premature rupture of membrane (PPROM), preterm labour (PTL), intrauterine growth restriction (IUGR), low birth weight (LBW) was seen in late detected women (32-34 week) as compared to ASB negative women. There was no significant difference between early detected women and asymptomatic bacteriuria negative women. They concluded that early detection and treatment of asymptomatic bacteriuria during pregnancy prevents complications like low birth weight, intrauterine growth retardation, preterm labour and PPRM. (15)

In 2013 **Verma Indu et al, Avasthi Kumkum et al, Berry Vandana et al** studied **Urogenital Infections as a Risk Factor for Preterm Labour**. Urogenital infections and their association with preterm labour was observed in 104 antenatal women. Urogenital infection was observed in 19 antenatal women in the study group and they are compared with 9 women in the control group. P value is 0.027 which is statistically significant. They studied that there is significant association between urogenital infections and preterm labour. Urogenital

infections contribute significantly to the preventable causes of preterm labor.(21)

In 2006 Naseem Saba et al, Anwar Sultana et al and Ihsanullah Mahsud et al studied outcome and complications of malaria in pregnancy. They studied 129 patients infected with malarial parasites. The infection was more common in multigravida than primigravida. Plasmodium falciparum infection was more common than vivax infection. 62% of patients had moderate anaemia (haemoglobin 8-10g/dl). 38 % of patients had severe anaemia (haemoglobin <8 g/dl). 2% of patients had cerebral malaria. Pregnancy outcome- 30% had puerperal pyrexia, 18% had spontaneous abortion, 9% babies died in neonatal period and 6% had pre-term labor. They concluded that malaria adversely affects the pregnancy outcome and it increases the risk of spontaneous abortion, stillbirths, preterm labor and low birth weight. (17)

In march 1997 Andrews Herbst et al studied maternal fever in relation to fetal tachycardia, cord artery acidaemia and neonatal infection. 248 infants whose mothers developed fever during labour were compared with 248 control infants. Women were matched for parity and duration of labour. They studied that maternal fever during term labour was associated with perinatal infection but not with acidaemia at birth.

In August 2011 **Francesca Bonvicini et al, Chiar Pucetti et al, Nunzio et al, Salfia et al** studied **gestational and fetal outcome in (parvo virus) B19 infection**. B 19 infection during pregnancy is a potential threat to the fetus. The virus has the ability to infect the fetal tissues and fetal erythroid precursor cells. B19 infection causes transitory fetal anemia, non immune fetal hydrops, miscarriage and intrauterine fetal demise. They studied 72 pregnant patients infected with parvo virus infection. Following maternal infection the fetus and neonates were clinically evaluated to monitor the pregnancy outcome. B19 IgM was positive in 94.1% persons and B19 DNA was positive in 96.3%.Maximum sensitivity was obtained with both B19 IgM and B19 DNA.10.2 % of pregnancies had fetal death.Vertical transmission was observed in 39% of pregnancy. If the maternal infection had occurred with in 20 weeks of gestation there was highest chances for congenital infections and adverse fetal outcome.11.9% of the fetus had B19 fetal hydrops.They concluded that prompt diagnosis of B19 infection with both IgM B19 and B19 DNA should be encouraged to prevent the maternal and fetal complications.(18).

In 2014 **Julie Werenberg Driier et al, Anne Marie Nybo Andersen et al, Gabriele Berg et al** studied **fever in pregnancy and health impacts in the offspring, a systematic review and meta**

analysis. They concluded that there is increased risks for the offspring when the mother exposed to fever during pregnancy. The strongest evidence was available for neural tube defects, congenital heart defects and oral clefts in which meta-analyses suggested between a 1.5- and nearly 3-fold increased risk with fever exposure in the first trimester. Antipyretics may have a protective effect when used during febrile episodes.(11)

AIMS AND OBJECTIVES OF MY STUDY

1. To find the maternal complications and fetal outcome of pyrexia in pregnancy beyond 28 weeks of gestation
2. To find the etiology and prevalence of pyrexia in pregnancy beyond 28 weeks of gestation

TYPE OF STUDY

Prospective Cohort study

PERIOD OF STUDY

MARCH 2016-AUGUST 2016

PLACE OF STUDY

Antenatal OPD,antenatal Ward, Labour ward

Dept of Obstetrics & Gynaecology

Govt Kilpauk Medical College &Hospital, Chennai

INCLUSION CRITERIA

CASES

- 1) Pregnant women with fever for more than 2 days (temperature $>38^{\circ}\text{C}$ orally)

2) Gestational age 28-40wks(sure of gestational age by LMP or USG in 1st and early 2nd trimester)

CONTROL

❖ Healthy pregnant women of gestational age of 28-40wks

EXCLUSION CRITERIA

- 1) Pyrexia due to blood transfusion
- 2) Connective tissue disorder
- 3) Renal disorder
- 4) Severe Anemia
- 5) Cardiovascular disease

SAMPLE SIZE

Cohort (exposed to fever) - 90

Control (not exposed to fever) -90

MATERIALS AND METHODS

All the patients attending the antenatal opd and admitted in antenatal and labour ward with fever who satisfy the eligibility criteria will be included till the sample size is reached and compared to equal number of healthy pregnant women without fever beyond 28 weeks of gestation.

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.

Detailed history, general examination, obstetrical examination will be performed.

General physician opinion will be obtained for the admitted cases and patients are treated according to their advice.

The patients will be followed up during their stay in the hospital and till delivery.

Phone number of the patients will be obtained and they asked to visit antenatal OPD once in two weeks till delivery.

At the time of delivery birth weight of the baby, gestational age at birth, APGAR score will be studied.

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.

BENEFITS OF THE STUDY

- 1) To find out the various maternal and fetal complications of pyrexia in pregnancy.
- 2) To find out the etiology and prevalence of pyrexia in pregnancy.
- 3) To prevent the maternal and fetal morbidity and mortality occurring due to fever and to treat the infections at the appropriate time to prevent the complications.

STATISTICAL ANALYSIS

The prevalence of low birth weight ,preterm delivery, intrauterine growth retardation, perinatal death and neonatal sepsis between the two groups will be calculated.

Relative risk will be calculated. The prevalence of various causes of fever will be enlisted. Various complications of maternal fever will be enlisted.

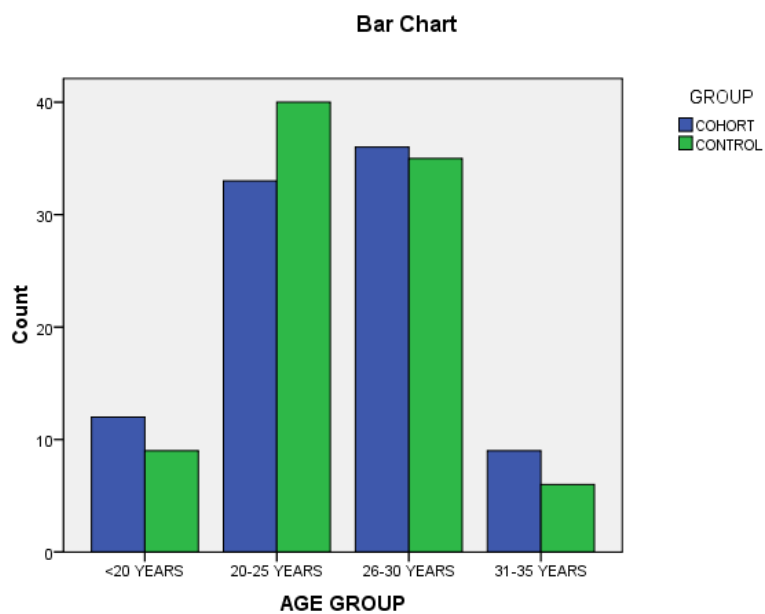
Data collection was done. The maternal and fetal complications of 180 pregnant women were evaluated and entered into the Microsoft excel sheet. The values were used for analysis.

The Chi-squared test was used for analysis .The software tools used for the purpose were downloaded from the internet. The values obtained were confirmed using another similar software to check the validity. The mean of the groups, the standard deviation, P-value and relative risk were calculated. The results were again converted into pie charts, bar diagrams for the sake of easy under-standing, and are presented as follows.

OBSERVATION AND RESULTS

AGE GROUP

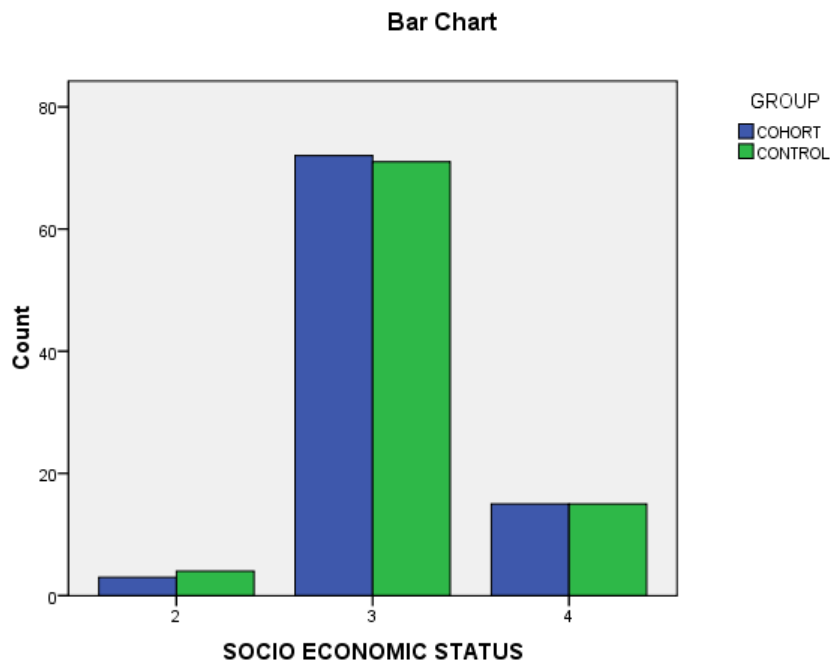
			GROUP		
			COHORT	CONTROL	Total
AGE GROUP	<20 YEARS	Count	12	9	21
		% within GROUP	13.3%	10.0%	11.7%
	20-25 YEARS	Count	33	40	73
		% within GROUP	36.7%	44.4%	40.6%
	26-30 YEARS	Count	36	35	71
		% within GROUP	40.0%	38.9%	39.4%
	31-35 YEARS	Count	9	6	15
		% within GROUP	10.0%	6.7%	8.3%
Total		Count	90	90	180
		% within GROUP	100.0%	100.0%	100.0%



Chi square value is 1.714 and p value is 0.634 which is statistically not significant. Most of the cases and controls belong to 20-25 and 26-30 years. Maternal age does not play a part in maternal complications and adverse fetal outcome.

SOCIO ECONOMIC STATUS

		GROUP		
		COHORT	CONTROL	Total
SOCIO ECONOMIC STATUS 2	Count	3	4	7
	% within GROUP	3.3%	4.4%	3.9%
3	Count	72	71	143
	% within GROUP	80.0%	78.9%	79.4%
4	Count	15	15	30
	% within GROUP	16.7%	16.7%	16.7%
Total	Count	90	90	180
	% within GROUP	100.0%	100.0%	100.0%



1-Upper socio economic class

2-Upper middle socio economic class

3-Lower middle socio economic class

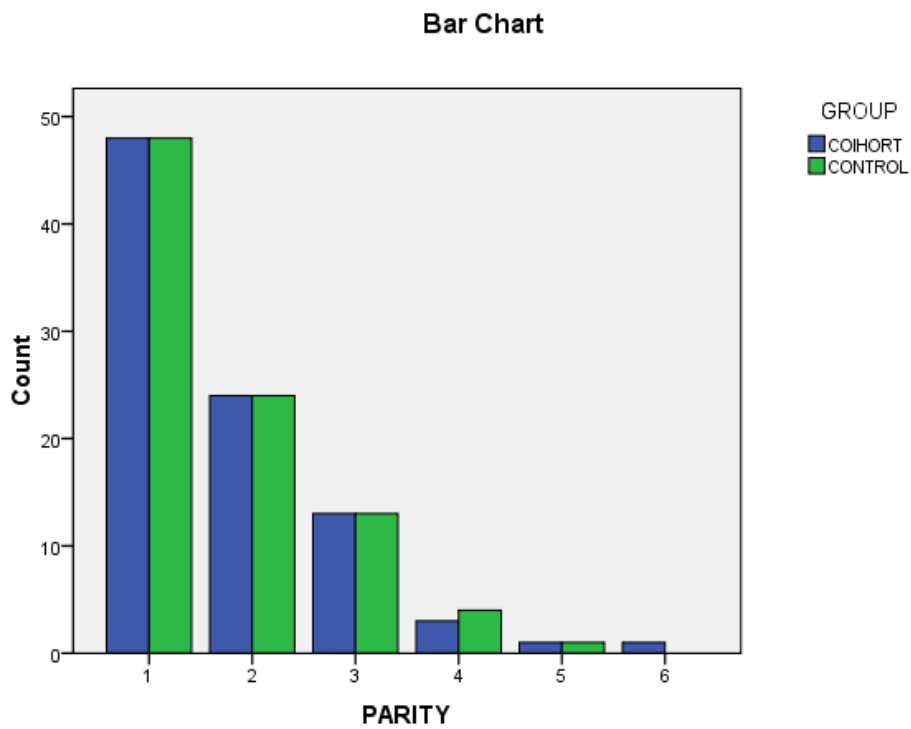
4-Lower socio economic class

Chi square value is 0.050 and p value is 0.928 which is statistically not significant. Maternal complications and fetal complications are not due to socio economic status.

GRAVIDA AND PARITY

TABLE

			GROUP		Total
			COHORT	CONTROL	
PARITY 1	Count	48	48	96	
	% within GROUP	53.3%	53.3%	53.3%	
2	Count	24	24	48	
	% within GROUP	26.7%	26.7%	26.7%	
3	Count	13	13	26	
	% within GROUP	14.4%	14.4%	14.4%	
4	Count	3	4	7	
	% within GROUP	3.3%	4.4%	3.9%	
5	Count	1	1	2	
	% within GROUP	1.1%	1.1%	1.1%	
6	Count	1	0	1	
	% within GROUP	1.1%	.0%	.6%	
Total	Count	90	90	180	
	% within GROUP	100.0%	100.0%	100.0%	



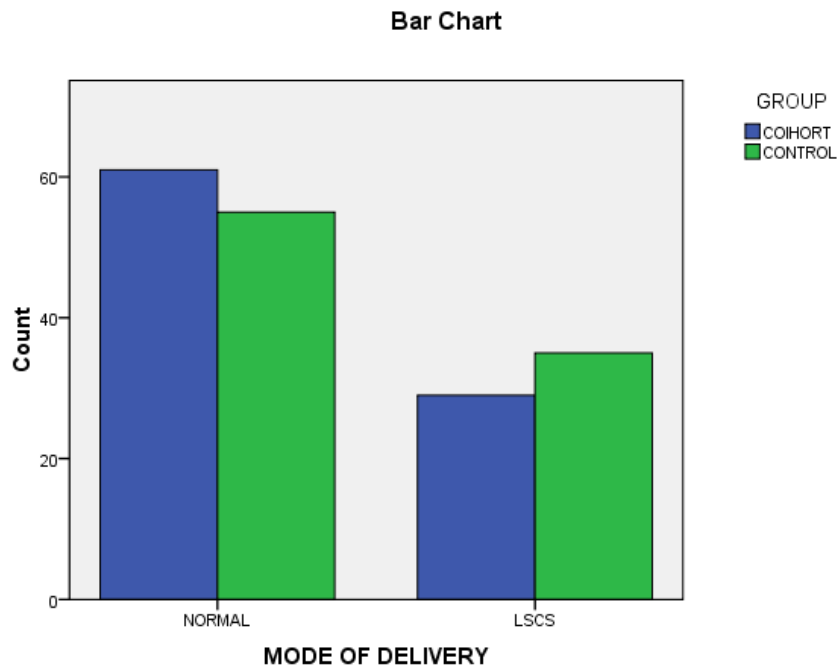
- 1- Gravida 1
- 2- Gravida 2
- 3- Gravida 3
- 4- Gravida 4
- 5- Gravida 5
- 6- Gravida 6

Chi square value is 1.143 and p value is 0.950 which is statistically not significant. Maternal and fetal complications due to fever are not related to gravida and parity.

	N	Mean	Std. Deviation	Std. Error Mean
TOTAL COUNT	90	11060.67	3242.968	341.839
PLATELET COUNT	90	2.617778	.7746805	.0816585
HAEMOGLOBIN IN GRAMS	90	9.924444	.8555583	.0901838

MODE OF DELIVERY

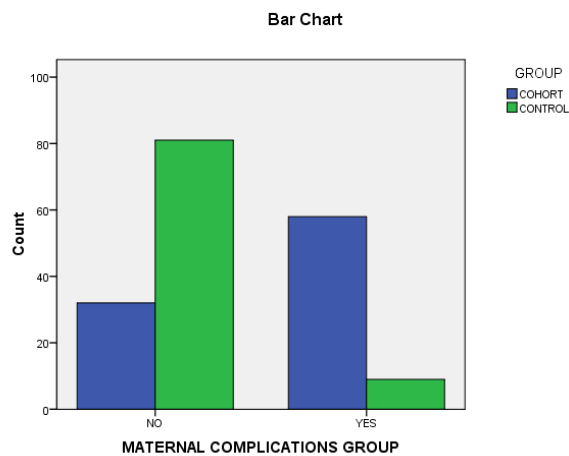
			GROUP		
			COHORT	CONTROL	Total
MODE OF DELIVERY	NORMAL	Count	61	55	116
		% within GROUP	67.8%	61.1%	64.4%
	LSCS	Count	29	35	64
		% within GROUP	32.2%	38.9%	35.6%
Total		Count	90	90	180
		% within GROUP	100.0%	100.0%	100.0%



In cohort 67.8 % and in control group 61.1% had normal vaginal delivery. 32.2% of cohort and 38.9 % of the control group had emergency LSCS. Chi square value is 0.873 and p value is 0.350. In cohort group fever during labor had more emergency LSCS due to meconium stained amniotic fluid, fetal distress and non reactive cardiotocography.

MATERNAL COMPLICATIONS

			GROUP		
			COHORT	CONTROL	Total
MATERNAL COMPLICATIONS GROUP	NO	Count	32	81	113
		% within GROUP	35.6%	90.0%	62.8%
	YES	Count	58	9	67
		% within GROUP	64.4%	10.0%	37.2%
Total	Count	90	90	180	
	% within GROUP	100.0%	100.0%	100.0%	

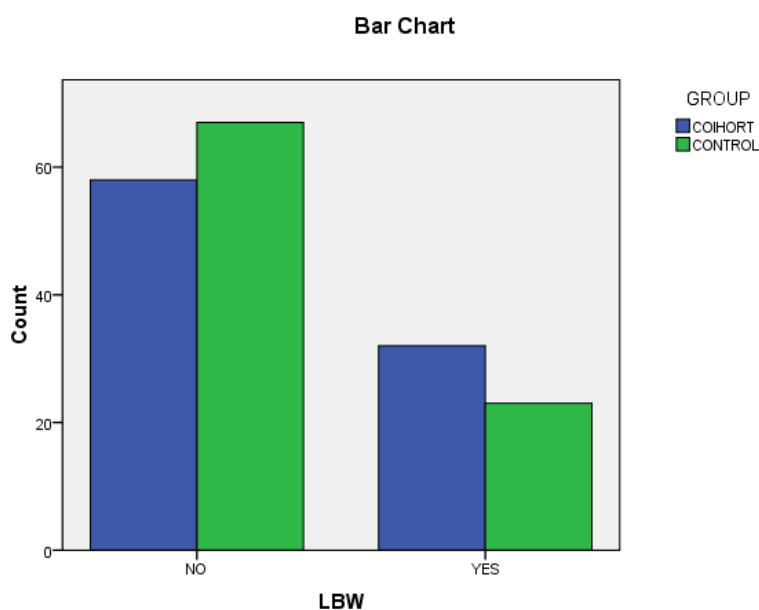


Maternal complications-chi square value is 57.084 and the p value is 0.000 which is statistically more significant. Maternal complications due to fever are more in cohort when compared to control group.

LOW BIRTH WEIGHT

			GROUP		Total
			COIHORT	CONTROL	
LBW	NO	Count	58	67	125
		% within GROUP	64.4%	74.4%	69.4%
	YES	Count	32	23	55
		% within GROUP	35.6%	25.6%	30.6%
	Total	Count	90	90	180
		% within GROUP	100.0%	100.0%	100.0%

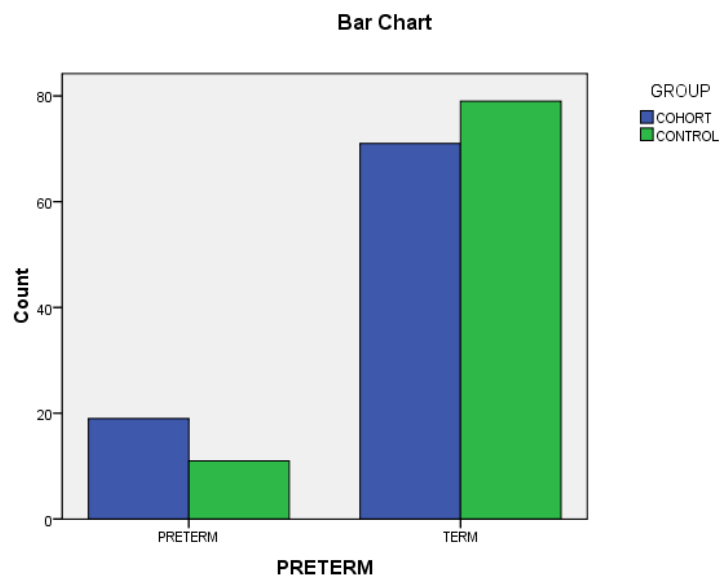
Chi square value is 2.121 and p value is 0.145 which is statistically not significant. The birth weight of the baby is due to gestational age at birth.



PRETERM LABOUR

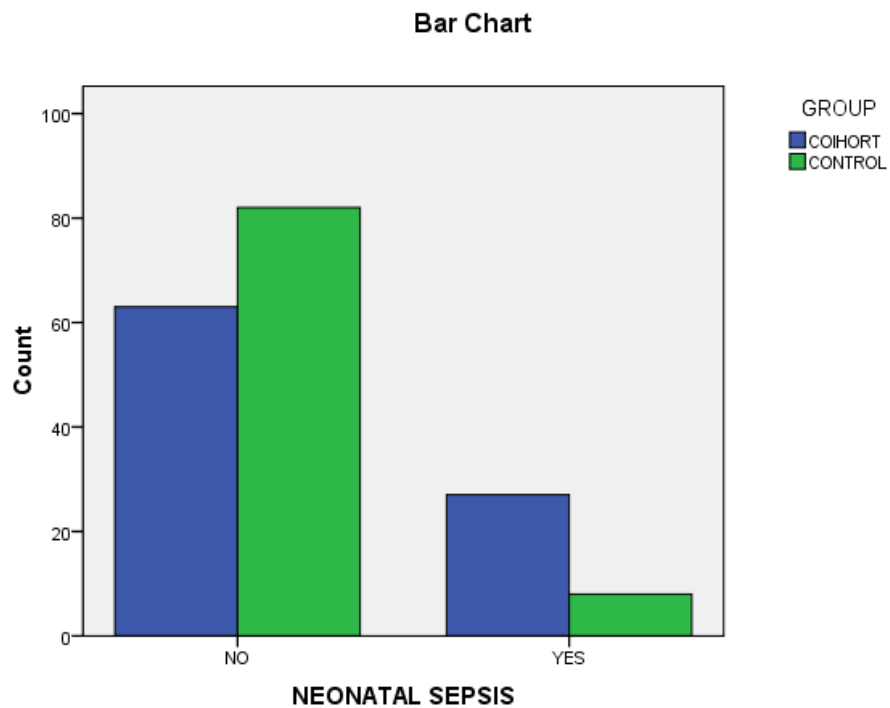
21.1% in the study group and 12.2% in the control group had preterm labour. The chi square value is 2.560 and the p value is 0.110. Relative risk is 1.7273 and 95% confidence interval is 3.4186. Preterm labour is more in the study group compared to the control group.

			GROUP		Total
			COHORT	CONTROL	
PRETERM	PRETERM	Count	19	11	30
		% within GROUP	21.1%	12.2%	16.7%
TERM	TERM	Count	71	79	150
		% within GROUP	78.9%	87.8%	83.3%
Total	Total	Count	90	90	180
		% within GROUP	100.0%	100.0%	100.0%



NEONATAL SEPSIS

			GROUP		
			COHORT	CONTROL	Total
NEONATAL SEPSIS	NO	Count	63	82	145
		% within GROUP	70.0%	91.1%	80.6%
	YES	Count	27	8	35
		% within GROUP	30.0%	8.9%	19.4%
Total	Count	90	90	180	
	% within GROUP	100.0%	100.0%	100.0%	

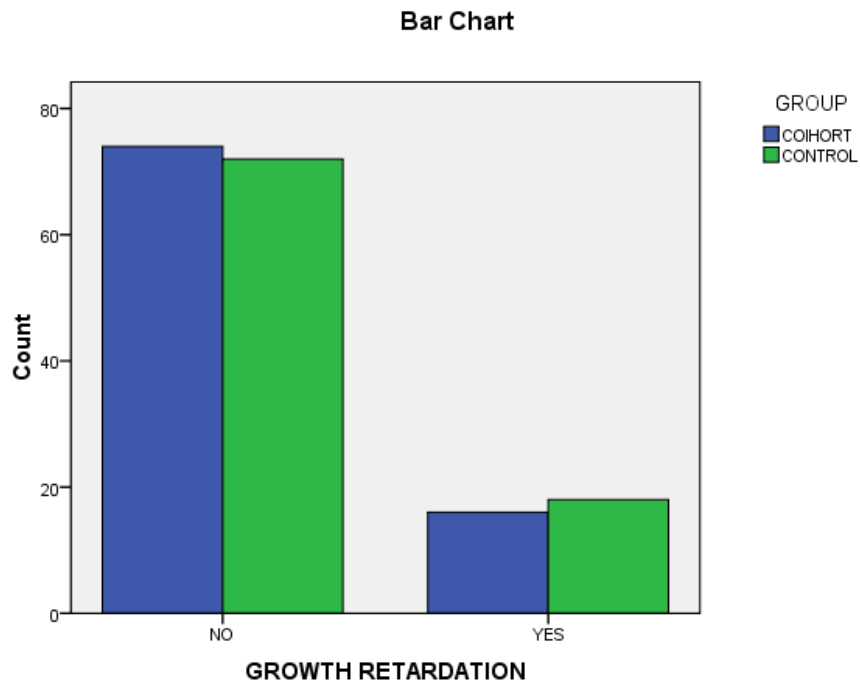


For neonatal sepsis the chi square value is 12.804 and p value is **0.000** which is statistically significant. Neonatal sepsis was more common in cohort when compared to controls. It is more common in patients exposed to fever.

INTRA UTERINE GROWTH RETARDATION

			GROUP		
			COHORT	CONTROL	Total
GROWTH RETARDATION	NO	Count	74	72	146
		% within GROUP	82.2%	80.0%	81.1%
	YES	Count	16	18	34
		% within GROUP	17.8%	20.0%	18.9%
Total		Count	90	90	180
		% within GROUP	100.0%	100.0%	100.0%

As regards intrauterine growth retardation the chi square value is 0.145 and the p value is 0.743 which is statistically not significant.



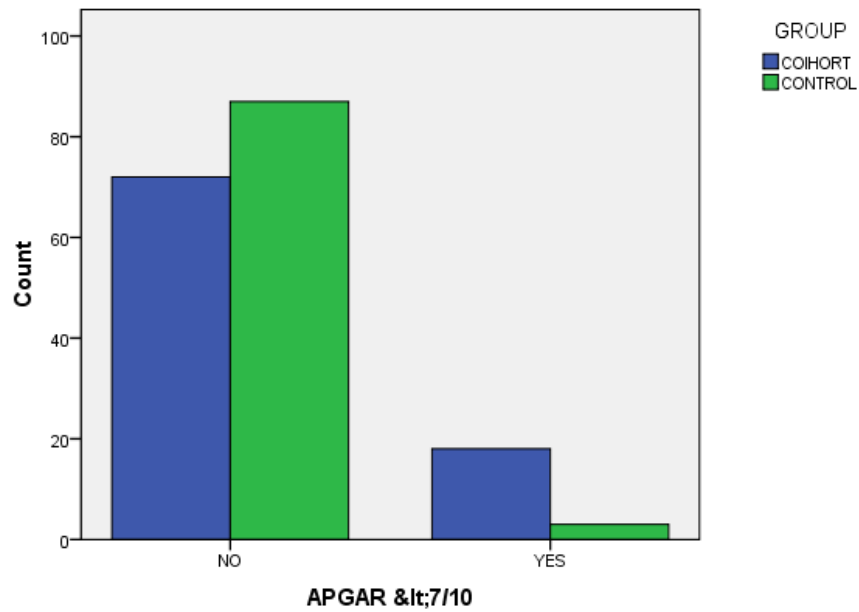
APGAR <7/10 * GROUP

Crosstab

			GROUP		
			COIHORT	CONTROL	Total
APGAR <7/10	NO	Count	72	87	159
		% within GROUP	80.0%	96.7%	88.3%
	YES	Count	18	3	21
		% within GROUP	20.0%	3.3%	11.7%
Total	Count	90	90	180	
	% within GROUP	100.0%	100.0%	100.0%	

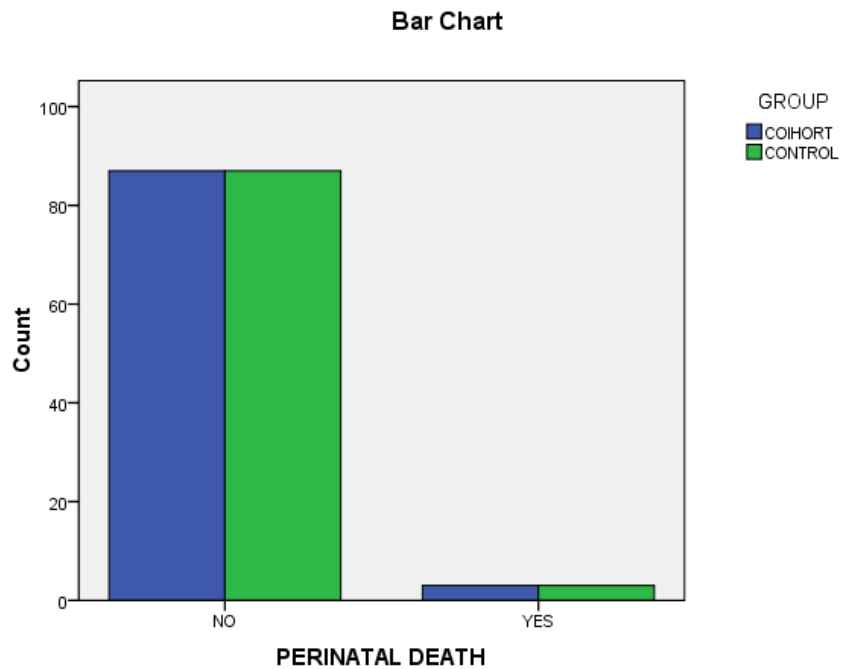
A regards APGAR the chi square value is 12.129 **and p value is 0.000** which is statistically very significant. Low APGAR score are more common in patients who had fever during labor and they need neonatal resuscitation.

Bar Chart



PERINATAL DEATH

			GROUP		
			COIHORT	CONTROL	Total
PERINATAL DEATH	NO	Count	87	87	174
		% within GROUP	96.7%	96.7%	96.7%
	YES	Count	3	3	6
		% within GROUP	3.3%	3.3%	3.3%
Total	Count	90	90	180	
	% within GROUP	100.0%	100.0%	100.0%	

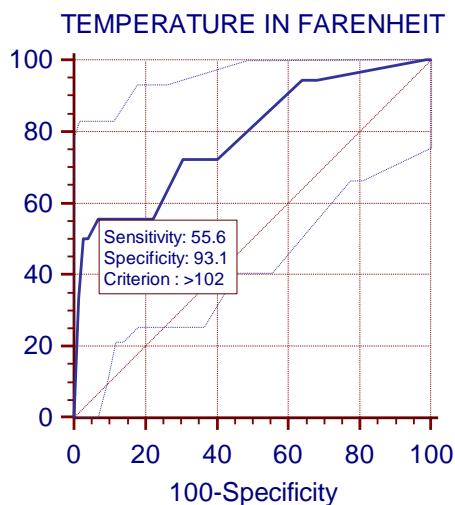


There are 2 perinatal deaths in cohort group and 2 in control group. The 2 perinatal deaths in cohort group had occurred in patients who had high fever during delivery. The two babies died due to meconium aspiration syndrome. In control group one baby died due to congenital anomalies and the other baby died due to perinatal asphyxia.

ROC

It is used to find out the optimum cut off value of fever (fahrenheit) values with respect to neonatal complications.

APGAR LESS THAN 7/10

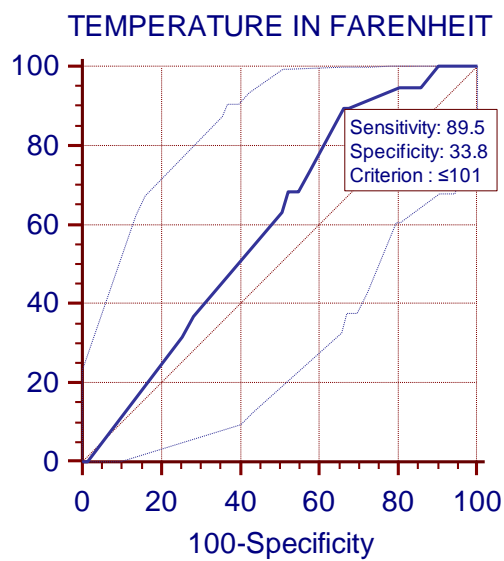


ROC curve

Variable	TEMPERATURE_IN_FARENHEIT TEMPERATURE IN FARENHEIT	
Classification variable	APGAR_7_10 APGAR <7/10	
Sample size		90
Positive group :	APGAR <7/10 = 1	18
Negative group :	APGAR <7/10 = 0	72
Area under the ROC curve (AUC)	0.785108	
Standard Error ^a	0.0649	
95% Confidence interval ^b	0.685944 to 0.864734	
z statistic	4.395	
Significance level P (Area=0.5)	<0.0001	

For APGAR < 7/10, sensitivity is 55.6 and specificity is 93.1. The area under the curve is 0.785 and the significance level is <0.0001 which is statistically significant.

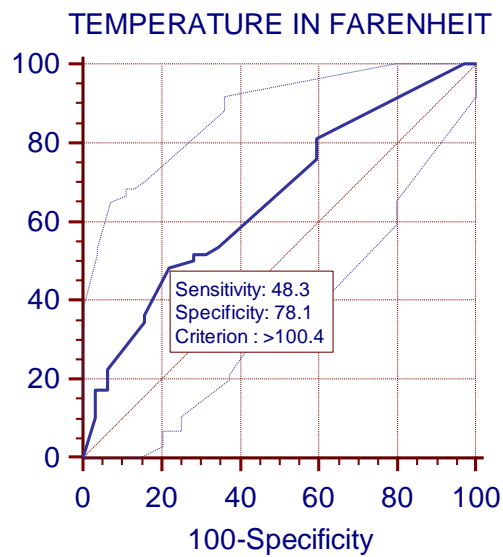
PRETERM LABOUR



Variable	TEMPERATURE_IN_FARENHEIT TEMPERATURE IN FARENHEIT	
Classification variable	PRETERM	
Sample size		90
Positive group :	PRETERM = 1	19
Negative group :	PRETERM = 0	71
Area under the ROC curve (AUC)	0.600074	
Standard Error ^a	0.0664	
95% Confidence interval ^b	0.491416 to 0.701955	
z statistic	1.508	
Significance level P (Area=0.5)	0.1317	

For preterm labor, sensitivity is 89.5 and specificity is 33.8. Area under the curve is 0.600074 and the significance p value is 0.1317.

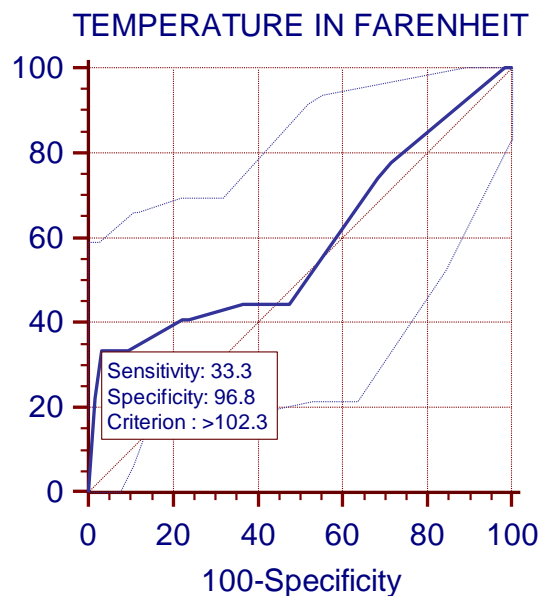
MATERNAL COMPLICATIONS



Variable	TEMPERATURE_IN_FARENHEIT TEMPERATURE IN FARENHEIT	
Classification variable	MATERNAL_COMPLICATIONS_GROUP MATERNAL COMPLICATIONS GROUP	
Sample size		90
Positive group :	MATERNAL COMPLICATIONS GR OUP = 1	58
Negative group :	MATERNAL COMPLICATIONS GR OUP = 0	32
Area under the ROC curve (AUC)	0.655711	
Standard Error ^a	0.0592	
95% Confidence interval ^b	0.548120 to 0.752736	
z statistic	2.630	
Significance level P (Area=0.5)	0.0086	

Sensitivity is 48.3 and specificity is 78.1 .The area under the curve is 0.655 and the significance p vaue is 0.008.Maternal complications are more in cohort when compared to control group.

NEONATAL SEPSIS



Variable		TEMPERATURE_IN_FARENHEIT TEMPERATURE IN FARENHEIT
Classification variable		NEONATAL_SEPSIS NEONATAL SEPSIS
Sample size		90
Positive group :	NEONATAL SEPSIS = 1	27
Negative group :	NEONATAL SEPSIS = 0	63

Area under the ROC curve (AUC)	0.586420
Standard Error ^a	0.0715
95% Confidence interval ^b	0.477704 to 0.689284
z statistic	1.208
Significance level P (Area=0.5)	0.0071

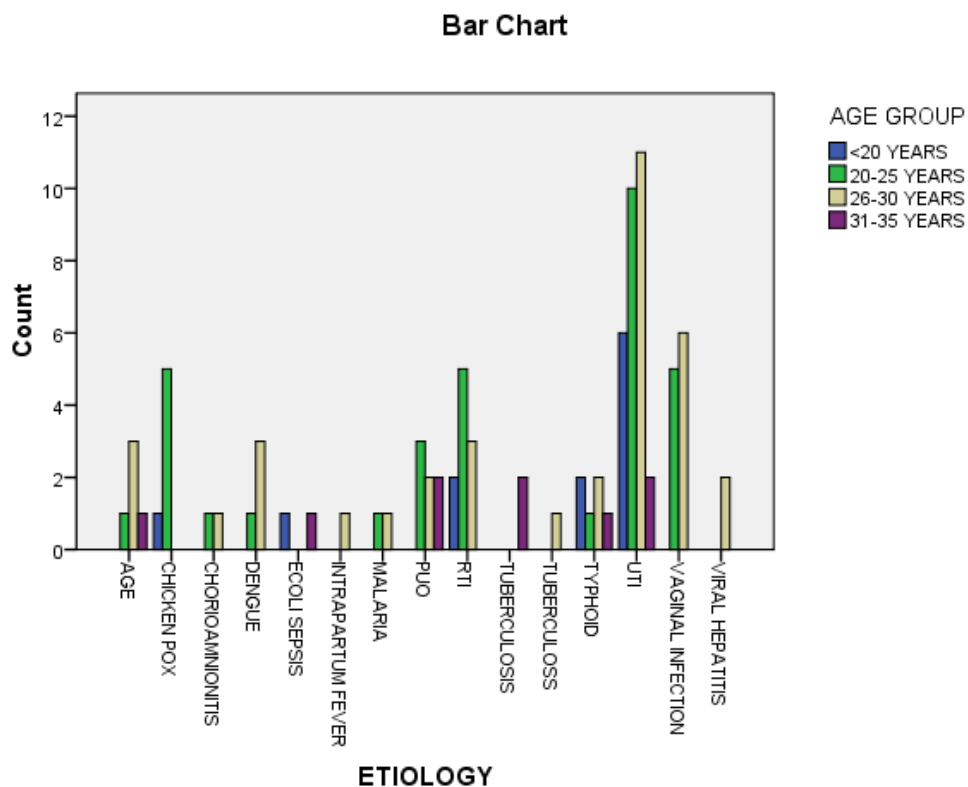
Sensitivity is 33.3% and specificity is 96.8. Area under the curve is 0.586 and the significance p value is 0.007 which is statistically more significant. Neonatal sepsis was more common in persons exposed to fever.

RESULTS

In this study we studied 180 pregnant women. Of these 90 women are exposed to fever beyond 28 weeks of gestation (cohort) and 90 women not exposed to fever (control).

ETIOLOGY OF FEVER:

1. Urinary tract infection n=29(32.2%)
2. Vaginal infections n=11 (12.2%)
3. Respiratory tract infection n=10 (11.1%)
4. Pyrexia of unknown origin n=7 (7.8%)
5. Typhoid (enteric fever) n=6 (6.7%)
6. Chicken pox n=6 (6.7%)
7. Acute gastro enteritis n=5 (5.6%)
8. Dengue fever n=4 (4.4%)
9. Chorioamnionitis n=3 (3.3%)
10. Tuberculosis n=3 (3.3%)
11. Malaria n=2 (2.2%)
12. Viral hepatitis n=2 (2.2%)



In our study the most common cause of fever was urinary tract infection followed by vaginal infections and respiratory tract infections.

Most of the patients are in the age group of 20 – 25 and 26- 30 years and most of them are primigravida.

MATERNAL COMPLICATIONS IN STUDY GROUP

1. Atonic PPH n=5 (5%)
2. Diarrhea n=5 (5.6%)
3. Consolidation of lungs n=2(2.2%)
4. Hypoglycemia n=1 (1.1)
5. DIVC n=1 (1.1% %)

6. Pleural effusion n=1 (1.1%)
7. Pneumonia n=5 (5.6%)
8. Puerperal sepsis n=4 (4.4%)
9. Thrombocytopenia n=4 (4.4%)
10. Wound infection n=5 (5.6%)
11. Anemia n= 9 (10%)
12. Oligohydromnios n=3 (3.3%)

The maternal complications related to the fetus includes preterm labor, pre labor rupture of membranes (PROM) (22.2%) ,preterm pre labour rupture of membranes (PPROM)(1.1%) and oligohydromnios.

64.4% (n=58) of the cohort had maternal complications and 35.6 % (n=32) of the cohort had no complication. Chi square test was applied to find the association and the value is 57.084 and the **significant p value is 0.000** which is statistically very significant. Relative risk is 6.444. Patients exposed to fever during their antenatal and intrapartum period had more complications when compared to those patients not exposed to fever.

We applied chi square test to find the association between maternal complications and demographic characters like age, parity and socio

economic status. No statistical association was between maternal complications and demographic characters.

Adverse fetal outcomes like low birth weight, intrauterine growth retardation (IUGR), preterm delivery, Apgar score <7 at 5 minutes after birth, neonatal sepsis and perinatal mortality were studied for these pregnancies with antepartum febrile illness. All deliveries were singleton and the mean birth weight was 2.5 kg.

Neonatal sepsis was found in 30% of the babies in the study group and 8% in the control group. We applied chi square test to find the association and the value is **12.804 and the significant p value is 0.000**. (p value <0.05 is significant). Relative risk is 3.3750. Neonatal sepsis are more common in patients exposed to fever when compared to controls.

20 % of babies born in the study group and 3.3% of babies in control group had **APGAR <7 at 5 minutes** of birth. We applied chi square test to find the association and the value is **12.129 and the significant p value is 0.000 which is statistically significant. Relative risk is 6.000**. Intrapartum fever patients had more adverse pregnancy outcome.

35.6% of babies born in the study group and 25.6% of babies born in the control group had low birth weight. The gestational age at birth determines the birth weight of the baby. Most of the patients in the study group had preterm labour and they had low birth weight babies. Chi square test is applied and the value is 2.121 and the significant p value is 0.145. Relative risk is 1.3913.

17.8% of babies in the study group and 20% of babies in the control group had intra uterine growth retardation. Chi square value is 0.145 and the p value is 0.703 which is statistically not significant. Relative risk is 0.8889.

3.3% in the study group and 3.3% in the control group had perinatal death. Chi square value is 0.00 and the p value is 1.000. Relative risk is 1.000. There were 2 perinatal deaths in the study group who had high fever during pregnancy. The two babies died due to meconium aspiration syndrome.

21.1% in the study group and 12.2% in the control group had preterm labour. The chi square value is 2.560 and the p value is 0.110. Relative risk is 1.7273 and 95% confidence interval is 3.4186. Preterm labour was more in the study group compared to the control group.

DISCUSSION

We studied 180 pregnant females, 90 females exposed to fever and 90 females not exposed to fever beyond 28 weeks of gestation. The maternal and fetal outcome of pyrexia in pregnancy was studied. The etiology of fever in the study group was made out. Urinary tract infection was the most common cause of fever in the study group.

The maternal and fetal outcome of pyrexia in pregnancy depends upon the etiology, duration of fever, gestational age at which it occurs and the extent of temperature rise.

Jhuma Biswaset al, Kaushik Banerjee et al in 2015 at Kolkatta studied the fetomaternal outcome of pyrexia in pregnancy. They studied 183 pregnant women with fever. In their study malaria was the most common cause of fever. In my study urinary tract infection was the most common cause of fever. In their study most of the patients are in the age group of 20-30 years and most of them were primigravida. In my study also the most of the patients are in the age group of 20-30 years. Low birth weight was the most common fetal outcome followed by low apgar score and preterm labour. In my study neonatal sepsis and low apgar score at birth was more sensitive and specific. Low birth weight, preterm labor, IUGR are more common in the study group compared to control group

Maternal complications are more common in the study group when compared to the control group **and the p value is 0.000**. Neonatal sepsis and low apgar score at birth are more specific and sensitive fetal outcome and their **p value is 0.000** which is statistically more significant. There were 2 perinatal deaths in the study group.

There were 4 cases of dengue fever. Of these one patient had severe thrombocytopenia corrected with platelet transfusion and intravenous fluids during labour. There are 2 malaria positive cases, of these one was plasmodium vivax positive and the other is plasmodium falciparum positive. Anaemia was the most common complication of malaria positive cases. Low birth weight was the most common fetal outcome in malaria positive cases.

Intrapartum fever had more severe consequences on maternal and fetal outcome. If there is high fever during labor, there are more chances for meconium stained liquor, fetal tachycardia, non reactive cardiotocography, fetal distress, perinatal asphyxia, intrauterine fetal demise and these patients need emergency cesarean section. Neonatal complications include perinatal mortality, neonatal seizures, poor APGAR score at birth, need for resuscitation after delivery, intraventricular haemorrhage, periventricular leucomalacia and cerebral palsy. Intrapartum antibiotics reduces the risk of neonatal morbidity. A

study conducted by **Lieberman et al** showed a strong association between intrapartum fever and low Apgar score, increased requirement of resuscitation and neonatal seizures in the first 24 hours following birth. One patient had high temperature during delivery (106 *F) and she had disseminated intravascular coagulation after delivery treated with blood and blood products.

5.5% of patients had puerperal infection and sub involution of uterus and they were treated with intravenous antibiotics. 2 persons in the study group had viral hepatitis and they had jaundice and hypoglycemia as complications. 4% of persons in the study group had post operative wound infections and they had fever in the post partum period. They are treated with culture specific antibiotics and wound re suturing was done for 2% of these patients.

32.2% had urinary tract infections and they had premature rupture of membranes(12.2%) and preterm labour as their complications. They are treated with antibiotics according to their culture and sensitivity . The most common organisms causing UTI were Escherichia coli, Klebsiella species, Enterococcus species and Proteus species. If left untreated it may lead to acute pyelonephritis ,low birth weight, PPRM, PROM, preterm labour and intrauterine growth retardation. 11% of patients in the study group had vaginal infections. The most common organism causing

vaginal infections were *Candida albicans*, *Trichomonas vaginalis*, *Klebsiella* species and *Enterococci* species.

In the study group 9 patients had anemia , of these 3 patients had haemoglobin < 6 grams and they were treated with blood transfusion.

Our study focused on fever in the antenatal period. Hyperthermia, typical effect of infectious agents, inflammatory reaction compounded by maternal complication had profound effect on the fetus. While maternal complications were very much dependent on the etiology of fever, fetal outcomes overlapped irrespective of the cause of fever. Moreover, adverse fetal outcome was more numerous than maternal complication. Hence it can be concluded that hyperthermia related changes in the uterine environment can affect fetal well-being.

CONCLUSION

A wide range of maternal and fetal complications can occur due to pyrexia in pregnancy from various causes. These complications can be preventable if the patient present to the hospital at an early time. The maternal and fetal complications can be avoidable if the cause for the fever is diagnosed and treated accordingly.

They should be treated with antipyretics and antibiotics according to their etiology to prevent the adverse maternal and fetal mortality and morbidity.

Hence standard methods for infection control in homes, communities and health care settings should be emphasized.

LIMITATIONS

1. The sample size is very small. Further studies can be done with large sample size.
2. We studied maternal and fetal complications beyond 28 weeks of gestation. Before 28 weeks many complications can occur due to pyrexia.
3. Maternal and fetal outcome for specific etiology of fever can be studied.

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PROFORMA

NAME : MOBILE NUMBER;

AGE :

IP NO :

ADDRESS :

SOCIOECONOMIC STATUS :

OBSTETRICS HISTORY :

1. Gravida / Parity / Gestational age

2. Regular ANC

3. LMP EDD

4. Living children

5. Abortion

6. Still birth

COMPLAINTS :

MENSTRUAL HISTORY : Regular / Irregular

MARITAL HISTORY : Married since

PAST HISTORY

1. Diabetes

2. Hypertension

3. Heart disease

4. Tuberculosis

5. Epilepsy

GENERAL EXAMINATION

TEMPERATURE : HEIGHT
PALLOR : WEIGHT :
PEDAL EDEMA : BMI :

VITALS :

PULSE :

BP :

CVS :

RS :

PER ABDOMEN :

PER VAGINAL

BLOOD INVESTIGATIONS :

TOTAL COUNT,DIFFERENTIAL COUNT,ESR

HAEMOGLOBIN

PLATELET COUNT

PERIPHERAL SMEAR FOR MALARIAL PARASITES :

WIDAL TEST :

URINE CULTURE AND SENSITIVITY :

BLOOD CULTURE AND SENSITIVITY :

DENGUE Ig G &Ig M :

SPUTUM AFB

FETAL OUTCOME;

BIRTH WEIGHT

GESTATIONAL AGE AT BIRTH

PERINATAL DEATH

APGAR SCORE AT 5 MINUTES

GROWTH RETARDATION

MATERNAL COMPLICATIONS:

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF GUIDE

DATE :

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு MATERNAL AND FETAL OUTCOME OF PYREXIA IN PREGNANCY BEYOND 28 WEEKS OF GESTATION

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது :

பங்கு பெறுபவரின் எண் :

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்

இடம் :

தேதி :

பங்கேற்பவரி ஆய்வாளரின் கையொப்பம்

ஆய்வாளரின் கையொப்பம்

