Introduction

Denoj Sebastian “Prevalence of torch infections in pregnancy related complications in the malabar region of Kerala”, Department of Life Sciences, University of Calicut, 2006
Introduction
Every minute, one woman somewhere in the world dies from a complication related to pregnancy or childbirth with a total death toll of almost 6 lakh women a year. Ninety-nine percent of these deaths occur in developing countries. Of these, India accounts for 1.36 lakh maternal deaths. That is, in India, one woman dies every five minutes from a pregnancy-related cause. For every three deaths of women in their reproductive years in some developing countries, one is the result of complications from pregnancy and childbirth. About 15% of deaths of women in the reproductive age in India are maternal deaths. Complications related to pregnancy, childbirth and complications arising out of unsafe abortion are leading causes of death in adolescent girls. In India, 50 percent of maternal deaths of girls in the 15-19 years age group are due to complications arising out of unsafe abortion.

A successful pregnancy outcome, in any given community, is a powerful indicator of the health status of its women and of the quality of health care available to them during pregnancy and birth. Poor maternal health resulting in repeated miscarriages, stillbirths and early infant death is a well known fact. Fetal death in uterus, if not detected in time and followed up with prompt hospitalisation, can endanger the mother's life.

Negative pregnancy outcome means all pregnancy outcomes other than a live birth and a voluntarily terminated pregnancy. It includes spontaneous abortions (miscarriages), stillbirths, intrauterine growth retardation (IUGR), premature labor, eclampsia, and other complications.
intrauterine fetal death (IUD), early neonatal mortality and congenital disorders/malformations. **Miscarriage** is defined as the premature expulsion from the uterus of the product of conception before 28 weeks of pregnancy. The term **Intrauterine Growth Retardation (IUGR)** is the most common generic term that is used to describe the fetus with a birth weight at or below the 10th percentile for gestational age and sex.\(^6\) while **Intrauterine Fetal Death (IUD)** includes all fetal deaths occurring during pregnancy, after 28th week of gestation and during labour. Early neonatal mortality refers to death of the infant within the first seven days after birth.\(^5\) Stillbirths or IUD and early neonatal deaths are often associated with premature births and low birth weights. A **Congenital disorder/malformation** is a medical condition that is present at birth. Congenital disorder can be recognized before birth (prenatally), at birth, or many years later.\(^7\)

Pregnancy wastage rates in India are high when compared to a number of developing countries. Miscarriage usually happens in the first three months. Early miscarriage is mainly due to the fetus failing to develop normally. Later miscarriage is more likely to be the result of the placenta not functioning properly or a weak cervix. Symptoms include bleeding; but this is not always the case and about half of all women who bleed in the early stages of pregnancy do not go on to miscarry. Bleeding may also not be related to the fetus at all but could be caused by lesions in the vagina or cervix.

Almost eight million low birth weight infants were born in India every year, accounting for around 40 per cent of low birth weight infants in the world.\(^1\) It is also said that 75 per cent of neonatal deaths occurred in infants with low birth weight\(^1,8\). A study in the Tamilnadu district showed a rate of 1.35% stillbirth, 3.53% neonatal mortality and 4.2% perinatal mortality.\(^9\) The
stillbirth rate in India was 30 to 35 per 1,000 births and the perinatal mortality rate was around 60 to 70 per 1,000 live births.¹

In the rural part of Tamil Nadu, women had a controlled reproductive pattern. The excess neonatal mortality among girls constitutes about one third of the perinatal mortality rate as a result of their preference to sons.⁹ According to a joint study ¹ conducted by the World Health Organization (WHO), the National Neonatology Forum and the United Nations Children's Fund (UNICEF) on the "State of newborns in India," out of the 26 million newborns every year, 1.2 million die within the first four weeks. This constitutes 30 per cent of the 3.9 million neonatal deaths worldwide. Neonatal Mortality Rate of 44 deaths per 1,000 live births as reported by The Hindu in 2004 accounts nearly two-thirds of the global infant mortality and half of the global child mortality. The undivided States of Uttar Pradesh, Madhya Pradesh and Bihar together accounted for over 50 percent of neonatal deaths in India in the year 2000. This was roughly 15 per cent of the global neonatal deaths. The number of deaths was as low as 10 per 1,000 live births in Kerala, whereas, it was around 60 in Orissa and Madhya Pradesh. Infections, birth asphyxia, and premature birth were identified as the leading causes of neonatal deaths.

Congenital anomalies were another significant negative outcome of pregnancy. The ratio of major congenital anomalies to the total number of deliveries reported by Henry and Varma in 1996 was 1:400.¹⁰

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High-Risk Pregnancy and Risk Factors

Pregnancy in which the mother, fetus, or newborn is or will be at increased risk for morbidity or mortality before or after delivery is termed high risk pregnancy.

Most pregnancies proceed normally and result in a healthy baby. The most important step in ensuring a safe and healthy pregnancy is identifying women at risk of complications and the best time to identify them is before they get pregnant. Factors such as lifestyle, family health history and the mother’s overall health offer important information about potential risks. For instance, women over age of 35 are considered at higher risk than younger women for pregnancy-related complications. Chronic health problems such as asthma, diabetes, heart problems, lupus and Rh disease also require particular care during pregnancy. Some factors, such as the mother’s advanced age, anemia and bleeding in pregnancy are considered as low-risk.¹¹

Maternal Infections

Intrauterine Infections

Anne, T. and Elizabeth, R ¹² opined that serious infectious illness in the mother can have non-specific fetal or obstetric effects and lead to miscarriage, premature labor or fetal death. These infections must be treated as any other serious illness. Infections acquired in utero or during the birth process are a significant cause of fetal and neonatal mortality and an important contributor to early and later childhood morbidity.

Usually the fetus is infected by transplacental spread after maternal infection, in which the organism circulates in the mother’s blood. These

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infections, acquired in utero, can be severe enough to cause fetal loss or can result in intrauterine growth restriction, prematurity, or chronic postnatal infection. In most cases the maternal illness is mild but the impact on the developing fetus is more severe. The degree of severity is dependent on the gestational age of the fetus when infected, the virulence of the organism, the damage to the placenta, and the severity of maternal disease. For example, a primary maternal infection such as *Herpes simplex* is more likely to be vertically transmitted and cause a more severe disease than recurrence of same infection in the mother. Wilcox, A.J. et al., observed the difficulty to determine the percentage of fetal loss due to infection during early pregnancy.

Different workers listed the unique pathogenic mechanisms of these infections. Because of their relatively low virulence, the organisms involved seldom lead to fetal death beyond the earliest stages of embryogenesis. Since the fetus is essentially a graft of foreign tissue in the uterus, the placenta constitutes a protective immunologic barrier that shields the fetus from the mother's humoral and cell-mediated immune responses. This makes the fetus especially susceptible to infection during the first trimester and the perinatal period. Early in pregnancy, the most complex events in embryogenesis take place, making sensory organs such as the eyes and ears vulnerable. The immature fetus lacks the immunologic mechanisms necessary to completely eliminate an infecting organism. Therefore, a state of immunologic tolerance is often established, which results in persistence of organisms that ordinarily would be eliminated by a normal child or adult.

Clinical evidence of infection may be seen at birth, soon afterward, or not until years later. The infected newborn infant may display growth retardation, developmental anomalies, or multiple clinical and laboratory abnormalities. Progressive tissue destruction is seen in Rubella, HSV, CMV,
Toxoplasma and *Treponema pallidum* as the infective agents continue to survive and replicate in the tissues for months or years after initial infection. This is particularly unfortunate when treatment is possible. The sequelae of these diseases can also progress over time, *e.g.*, the hearing loss that is secondary to rubella infection can progress or develop even after years of normal hearing.\(^{13}\)

According to Anne, T. and Elizabeth,\(^{12}\) routine screening of pre-pregnancy or antenatal cases for the presence of, or susceptibility to, these infections and appropriate management can prevent adverse fetal or perinatal outcomes. This screening should include infection with TORCH agents, Hepatitis B virus, *Treponema pallidum*, and HIV.\(^{16}\) When a TORCH test or screening is ordered on a newborn, it is suspected that the child has been exposed in utero to one of several organisms that can cause mild or subclinical disease in the mother but devastating damage to the infant. Routine screening of pregnant women for TORCH titers at the first prenatal visit is commonplace in many parts of the world. However, the value of this testing has been questioned by workers like Garland, S.M *et al.*,\(^ {17,18}\) Khan, N.A *et al.*,\(^ {17,18}\) and many others, while screening of both maternal IgG and IgM antibodies are performed routinely.

The acronym TORCH has become one of the most recognized in the field of neonatal/perinatal medicine. The original concept of the TORCH perinatal infections was to group five infections with similar presentations, and etiology. These are the infections with;

- *Toxoplasma gondii*
- Other diseases (Paravovirus B19, *T. pallidum*, Varicella zoster)
- Rubella Virus
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- Cytomegalovirus (CMV)
- *Herpes simplex* Virus (HSV)

*Toxoplasma gondii*

*Toxoplasma*, an apicomplexan parasite, is an obligate intracellular parasite that has evolved a very different survival strategy from the extracellular trypanosomes. The life cycle of *Toxoplasma* is very complex but breaks down into two parts, one sexual and the other asexual.

The sexual cycle occurs exclusively in cats and is initiated when a cat eats an infected prey or accidentally ingests feces contaminated with oocysts. Following a typical process of gamete formation and fusion in the intestinal epithelium, the zygote is formed. This ultimately develops into an immature oocyst which, after being shed in the feces will mature into an extraordinarily resistant entity containing 8 sporozoites. The oocysts are highly infectious not only to other cats but to virtually any warm-blooded animal. Herbivorous grazing animals, of course, will be particularly susceptible but it's also found in strict carnivorous animals, as well. Once in the herbivore, the sporozoites are released from the oocyst in the intestine and invade the intestinal epithelium. There they differentiate into the rapidly dividing tachyzoite form which is capable of indefinite replication *in vivo* and *in vitro*. These disseminate through the host, infecting virtually any cell in any tissue. As the host's immune response rises to the challenge, the parasites encyst and differentiate to the very slowly dividing form, the bradyzoite. These are very stable and infectious if tissue from the animal is eaten.

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The most serious problems occur in either of two situations. First, if a woman becomes infected for the first time during pregnancy, her fetus is at risk of severe neurological problems, even death. Second, if a chronically infected person develops AIDS or is immuno-suppressed for any other reason, the disease can reactivate with the quasi-latent tissue cysts releasing the bradyzoites which rapidly differentiate back to tachyzoites. The resulting disseminated infection is of greatest concern when it enters the brain where encephalitis can ensue.

According to Hohlfeld, P. et al., toxoplasma infection during pregnancy can cause congenital infection and manifest as mental retardation and blindness in the infant. The infant is infected transplacentally after the parasites invade the placenta. Once acquired, the latent encysted organism will persist for life in the host. Like congenital syphilis and perinatally acquired HIV infection, congenital toxoplasmosis is usually not apparent at birth. Boyer, K.M. reported that 70 to 90% of the infants who appear normal at birth would develop significant clinical illness by young adulthood. These
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infants develop choreoretinitis that can lead to blindness, obstructive hydrocephalus, and intracranial calcifications that are associated with mental retardation, seizure activity, and motor and developmental delays.

In Europe, pregnant women are screened monthly as part of standard prenatal care as early as 18 weeks' gestation, by using polymerase chain reaction (PCR) amplification of the B1 gene of *Toxoplasma gondii* in a sample of amniotic fluid. Whether the fetus has actual organ damage is determined by serial ultrasound examinations.20

Early diagnosis is important because the disease is most severe in the fetus when the mother is acutely infected in the first trimester. However, the disease in the mother may be easily overlooked sometimes, as it is often asymptomatic. It may also be overlooked because the maternal physical findings, *e.g.*, fever, lymphadenopathy (swelling of one or more lymph nodes), headache, myalgia (muscle pain), stiff neck, and anorexia (decreased sensation of appetite), can easily be attributed to other more common infections.13

Others (*Treponema pallidum*, Varicella-Zoster Virus (VZV), Parvovirus B19, and Human Immunodeficiency Virus (HIV))

*Treponema pallidum*

According to Rawston,S, 21 congenital syphilis is caused by the transplacental transmission of the spirochete, *Treponema pallidum*, which has a 100% vertical transmission rate. Syphilis in the mother is characterized by three different stages: the primary stage, which is characterized by the appearance of the syphilitic chancre and lymphadenitis and the secondary
stage, which is the result of hematogenous dissemination. The newborn infant with congenital syphilis is considered to be in the secondary stage. During the tertiary stage which is either asymptomatic (late latent) or symptomatic (tertiary stage) neurological, cardiovascular, and gummatous lesions (granulomas of the skin and musculoskeletal system) are seen.

Syphilis is currently at its lowest incidence since reporting first began in 1941 and the decline in incidence of congenital syphilis is attributable mainly to mandatory serologic screening during pregnancy.  

Congenital syphilis which has classifications as early disease (seen in children before two years) and late disease (seen after two years) is more likely to be transmitted by women who are in the primary or secondary stages of the disease rather than in the latent phase. Azimi, P reported 40% fetal or perinatal deaths in the patients without treatment. If not detected or treated these live-born infants will display symptoms within weeks or months of birth.

Early manifestations of congenital infection involve multiple body systems. Infants may display hemorrhagic nasal discharge, hepatosplenomegaly (simultaneous enlargement of both the liver and the spleen), jaundice, increased liver enzymes, lymphadenopathy (swelling of one or more lymph nodes), hemolytic anemia, thrombocytopenia (presence of relatively few platelets in blood), Osteochondritis dissecans (a loose piece of bone and cartilage separates from the end of the bone because of a loss of blood supply and insufficient amounts of calcium) and periostitis (the inflammation of the periosteum), mucocutaneous rash, central nervous system (CNS) abnormalities, failure to thrive, choreoretinitis (inflammation of the choroid and retina of the eye), nephritis (inflammation of the kidney), and
nephrotic syndrome. Late manifestations result primarily from chronic inflammation of bone, teeth, and CNS.

Serologic tests are the main means of diagnosis. VDRL (Venereal Disease Research Laboratory) and RPR (rapid plasma reagin) tests detect antibodies against cardiolipin. While not specific for syphilis, these "nontreponemal" tests are useful in its diagnosis because the quantitative results of these tests correlate with disease activity. Thus, they are practical for screening purposes.

**Varicella-Zoster Virus (VZV)**

Varicella infection in the pregnant woman can cause severe consequences for both the mother and infant. Infection of the fetus in early pregnancy results in congenital varicella syndrome; transplacental infection at delivery results in neonatal varicella. Interestingly, recurrent VZV infection does not cause congenital varicella syndrome or neonatal varicella. But because most adults at present are immune as a result of childhood illness, the incidence of maternal varicella is low in United States. The clinical experience in Japan, which is now being substantiated by long-term studies in the United States, indicates that the immunity persists for prolonged periods after childhood immunization.

Arvin, A reported that about 25% of fetuses become infected when the mother has varicella but not every infected fetus is clinically affected. Infants infected later in pregnancy have less frequent and less severe involvement.

The anomalies associated with congenital varicella syndrome involve many organ systems. There are unusual cutaneous defects, with cicatrical skin

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scars, atrophy of extremities, and evidence of damage to the central nervous system.\textsuperscript{28}

\textbf{Parvovirus B19}

Most commonly, parvovirus B19 causes the childhood viral exanthem erythema infectiosum (Fifth disease or "Slapped cheek disease") that was first associated with the virus in 1983. By the age of 15 years, 50\% of adolescents have detectable IgG antibodies to the virus.\textsuperscript{29} Transmission is airborne or by droplet spread. Most infections in adulthood are asymptomatic or mild causing a subtle rash and arthralgia. Maternal infection during pregnancy can result in miscarriage or the development of nonimmune hydrops fetalis. The pathogenesis in the fetus is cardiac failure as a result of profound anemia caused by virus-induced arrest of red cell production. The risk of transplacental infection is about 30\% and the risk of fetal loss about 9\%, primarily in the second trimester. Chronic congenital infection with perinatal sequelae is rare.\textsuperscript{30} The best way to diagnose B19 infection in the fetus is the demonstration of viral DNA in amniotic fluid, fetal blood, or tissues by PCR method.

\textbf{Rubella Virus}

A benign self-limited viral illness, rubella, is characterized by an exanthem and posterior cervical lymphadenopathy. Much of its significant morbidity is secondary to the effects of the illness when contracted by the fetus in utero. The congenital rubella syndrome that was so common before 1969 included growth retardation, deafness, congenital heart disease, and mental retardation. Since the availability of mass immunization, the reported incidence of rubella has dropped from 57,686 cases in 1969 to 200 to 400 cases from 1992 to 1998.\textsuperscript{31} Although protection of women of childbearing age
is the goal of all immunization strategies, serological surveys in the United States continue to document 10 to 20% susceptibility in this population.

The gestational age at the time of infection determines the intensity of fetal involvement. Up to 20% of maternal infections occurring in the first eight weeks' of gestation result in miscarriage, spontaneous abortion, or stillbirth. Those fetuses infected before 11 weeks have multiple organ damage while those after 11 to 12 weeks are more likely to have only deafness and/or retinopathy. Fetal damage rarely occurs after 16 weeks' of gestation.32

Clinical manifestations can be evident at birth but more commonly result in a ‘normal’ newborn with late-onset sequelae. Early clinical manifestations can be transient or progressive. Transient early clinical manifestations of congenital rubella include generalized lymphadenopathy, hepatosplenomegaly, intrauterine growth restriction, hepatitis, jaundice, thrombocytopenic purpura (low platelet count), with petechiae (small red or purple spot on the body, caused by a minor hemorrhage) and ‘blueberry muffin’ lesions. These transient manifestations resolve in days or weeks usually without long-term sequelae. The most common permanent problems seen are sensorineural deafness, cataracts, peripheral pulmonary stenosis, mental retardation, central language defects, diabetes mellitus type I, and hypogammaglobulinemia (a type of immune deficiency, where the number of gamma globulins is greatly reduced).33

The infant should be evaluated for congenital rubella if there is a maternal history of rubella during the pregnancy or neonatal manifestations suggestive of a congenital infection, such as thrombocytopenic purpura or cataracts. Diagnosis of congenital rubella requires virologic or serologic confirmation. The virus can be isolated for up to one year or more from the...
nasopharynx, cerebrospinal fluid (CSF), urine, and buffy coat of the blood. Serologic confirmation is difficult. Although rubella-specific IgM can be measured in cord blood or neonatal serum, it is often associated with false positive and false negative results. It is recommended that serial measurements of IgG specific antibodies be done at three and six months to document persisting high antibody levels. Presence of rubella-specific hemagglutination inhibition (HAI) or enzyme immunoassay antibodies after nine months of age is diagnostic of congenital infection.\textsuperscript{33}

The prevention of congenital rubella obviously is dependent upon adequate early immunization, resulting in a high prevalence of immunity in women of childbearing age. Women should be screened for rubella immunity at the beginning of pregnancy and immunization is recommended for seronegative women immediately after delivery.\textsuperscript{34} Although inadvertent immunization of pregnant women has not resulted in fetal abnormality, postpartum immunization is considered safe.

**Cytomegalovirus (CMV)**

Currently CMV is the most common cause of congenital infection in the United States.\textsuperscript{35} Infected infants of 10 to 20 % may suffer from sensorineural hearing loss, ocular damage, or impairment of cognitive and motor function. CMV is common in all socioeconomic groups but congenital infection with significant impairment is seen at highest rates in populations in which pregnant women have the highest risk of acquiring primary infection. In addition to the transplacental route, CMV can be transmitted at delivery via the maternal genital tract, during the postpartum period in breast milk, and in transfused blood products. CMV is easily spread in daycare centers and in families with young children. The organism can cause significant illness by
endogenous reactivation among immunosuppressed individuals, including transplant recipients.

According to Pass, R\textsuperscript{36} approximately 40% of maternal primary infections are transmitted to the fetus. The likelihood of transmission is similar early as well as late in gestation. However, first trimester primary maternal infection is more likely to cause neonatal infection, which is evident at birth, and more likely to result in severe sequelae such as deafness and mental retardation. Transmission of CMV from the mother to fetus can occur even if the mother was infected long before conception. However, maternal infections that result from reactivation of the virus usually cause only asymptomatic viral shedding in the infant.

Congenitally infected infants are often divided into two groups: those with findings that are apparent in the neonatal period and those with signs of CNS damage that become apparent later in childhood. Those symptomatic at birth are most compromised. In addition to intrauterine growth restriction, over 70% have evidence of CNS involvement: microcephaly, lethargy, hypotonia, optic atrophy, decreased hearing, and intracranial calcifications. Such infants have a mortality rate of 12% by six months of age. Of infants who are asymptomatic at birth, 10 to 20% eventually will have CNS involvement. Congenital CMV is said to be the second leading cause of mental retardation in the United States and is currently the leading cause of sensorineural deafness. Hearing loss secondary to congenital CMV is progressive in childhood; even those with normal hearing at birth can develop hearing loss later.\textsuperscript{37}

Transmission of the virus requires direct contact with bodily fluids. Hand washing and other preventive hygienic measures can decrease spread in
daycare centers and at home. Pre-pregnancy titers can also identify at-risk women. In the future, a CMV vaccine currently being evaluated in young adults may be of use.\textsuperscript{38}

\textit{Herpes simplex Virus}

Though herpes simplex infections were recognized by the ancient Greeks, the association between herpes simplex type 2 as a cause of neonatal disease and genital herpes was not made until the 1960s.\textsuperscript{39} The recent development of antiviral therapy enables the reduction of mortality and morbidity. As herpes simplex is most often acquired during delivery rather than during gestation, it is more preventable and treatable compared with CMV or rubella. Like the varicella-zoster virus, the herpes simplex virus can persist in the latent state, resurfacing at any time during the individual's life span.

While there have been fluctuations in incidence of the disease, it is estimated that there are approximately 30 cases/100,000 live births in the United States. It is interesting that neonatal infection occurs far less frequently than might be expected given the high prevalence (1/5) of seropositivity to HSV-2 in childbearing women.\textsuperscript{40} The infant attack rate among women with primary infection is 33 to 50\%, while those with recurrent disease only show an attack rate of 1 to 3\%.\textsuperscript{41} Unfortunately, only 15 to 20\% of women whose infants develop neonatal herpes infection have a history of symptomatic disease and only 25\% have relevant symptoms at delivery.\textsuperscript{40}

Perinatal infection manifests itself during the first month of life with 9\% of infections occurring on day one and 40\% by the end of the first week of life. There are three major categories seen: (1) localized skin, eye and mouth

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infected; (2) encephalitis with or without skin, eye, and mouth disease; and (3) disseminated infection.\textsuperscript{42} Infants with disseminated disease have the worst prognosis. Many are born to mothers with a new herpes infection who have not developed or passively transferred antibodies against the virus to the infant. Multiple organs are involved and initial signs and symptoms include irritability, seizures, respiratory distress, jaundice, bleeding diathesis, and shock.

Arvin, A. & Whitley, R\textsuperscript{40} pointed out that the diagnosis of disseminated disease is difficult because symptoms are vague, nonspecific, and similar to those of bacterial sepsis or enteroviral infections. Nearly one-third of all infants with neonatal herpes have only encephalitis as the initial manifestation. As with disseminated disease, not all of these infants display the characteristic vesicular exanthem. Infection localized to the skin, eye, and/or mouth may seem benign but, in the absence of treatment, this onset is often associated with the subsequent development of the more serious disease manifestations. Skin vesicles are noted in 90\% and keratoconjunctivitis (inflammation ("itis") of the cornea and conjunctiva) may be evident with eye involvement. Long-term neurological impairment is frequent in children who have recovered from encephalitis.

Other Maternal Infections

Bacterial vaginosis

Bacterial vaginosis, previously known as nonspecific vaginitis or Gardnerella vaginitis, is the most common cause of vaginal discharge. It may be the cause of up to one half of cases of vaginitis \textsuperscript{43} in all women and the cause of from 10 to 30 percent of cases in pregnant women.\textsuperscript{44} This clinical
syndrome is now recognized as a polymicrobial superficial vaginal infection involving a loss of the normal lactobacilli and an overgrowth of anaerobes. While commonly found in increased numbers in women with bacterial vaginosis, *Gardnerella vaginalis* is not invariably present. *G. vaginalis* has been reported in from 16 to 42 percent of women with no signs or symptoms of vaginitis. Morbidity associated with bacterial vaginosis in pregnant women are amniotic fluid infection, clinical chorioamnionitis, postpartum endometritis, premature rupture of the membranes, preterm delivery and low birth weight.

**Urinary Tract Infection (UTI)**

The organisms that cause UTIs during pregnancy are the same as those found in nonpregnant patients. If untreated, 40% of the asymptomatic bacteriuria (ASB), will develop a symptomatic UTI. *Escherichia coli* accounts for 80 to 90 percent of infections. Other gram-negative rods such as *Proteus mirabilis* and *Klebsiella pneumoniae* are also common. Gram-positive organisms such as group B streptococcus and *Staphylococcus saprophyticus* are less common causes of UTI. Group B streptococcus has important implications in the management of pregnancy. Less common organisms that may cause UTI include enterococci, *Gardnerella vaginalis* and *Ureaplasma urealyticum*. Fetal risks associated with a UTI include abortion, preterm labour, low birth weight (LBW), fetal infection and perinatal death. Selective screening of pregnant women for ASB is recommended at the first antenatal visit.

**Other Maternal Factors**

Out of several pregnancy related maternal complications, a few important ones worth mentioning here are hypertension, pre-eclampsia and

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oligohydramnions etc. Hypertensive disorders of pregnancy result in 12% of maternal deaths globally, and up to 40% of maternal deaths in some countries. These conditions can also influence the health of the fetus or newborn and are responsible for up to 13% of stillbirths and 20% of early neonatal deaths in some areas of the world. The World Health Organization (WHO) estimates that 15% of women will have some degree of hypertension during pregnancy. Fortunately, most of these cases are benign and do not require treatment or result in complications. In some cases, however, the woman has a hypertensive disorder of pregnancy such as preeclampsia (hypertensive disorder of pregnancy with associated protein loss in the urine) and eclampsia, (serious complication of pregnancy characterised by convulsions) which can lead to serious complications or death. 49.

Pregnancy Induced Hypertension is also reported to have role in causing cerebral, cardiac, and renal complications, stillbirths and abruptio placentae in the mother. In the fetus, intrauterine growth retardation and hypoxia (lack of oxygen in tissues) due to superimposed pregnancy-induced hypertension are common. Managing hypertension during pregnancy is one of the most controversial areas of therapy in obstetric practice. 50

Oligohydramnios and polyhydramnios are found to be causing problems for mother and baby. The amniotic fluid that surrounds the baby in the uterus is a clear-colored liquid cushion and protects the baby, provides it with fluids and is crucial in normal development. The baby breathes this fluid into its lungs and swallows it; this helps promote the healthy growth of the lungs and gastrointestinal tract. Amniotic fluid also helps the baby move around, aiding in normal development of muscle and bone. The amount of amniotic fluid increases until about 28-32 weeks of pregnancy. The level stays about the same until about 38 to 40 weeks, i.e. during full term
pregnancy. After that, the level begins to decrease. In some pregnancies, there may be too little or too much amniotic fluid. These conditions are referred to as oligohydramnios and polyhydramnios respectively. Both can sometimes cause problems for mother and baby, or be a sign of other problems. In the majority of cases, however, the baby is born healthy. The level of the amniotic fluid is measured in terms of amniotic fluid index (AFI). This is done using ultrasonography. If the amniotic fluid depth is less than 5 centimeters (cm), it is called oligohydramnios. If the depth measures greater than 25 cm, the condition is called polyhydramnios.

About 8 percent of pregnant women all over the world have too little amniotic fluid. Oligohydramnios can develop at any time during pregnancy, though it is most common in the last trimester. Oligohydramnios that occurs in the first half of pregnancy is more likely to have serious consequences than if it occurs in the last trimester. Too little amniotic fluid early in pregnancy can compress fetal organs and cause birth defects, such as lung and limb defects. Oligohydramnios that develops in the first half of pregnancy also increases the risk of miscarriage, preterm birth and stillbirth. When oligohydramnios occurs in the second half of pregnancy, it may be associated with poor fetal growth. Women with oligohydramnios are more likely than unaffected women to need a cesarean delivery.
Relevance of the Study

Although pregnancy-related complications continue to take a huge toll on the lives of women and newborns, and despite a series of programmatic initiatives, there is little evidence that maternity has become significantly safer over the last 20 years. The links between pregnancy-related care and maternal mortality are well recognized. All pregnancies should be evaluated well at correct stage since many risk factors are involved in pregnancy. High risk pregnancies should receive extra attention and is to be referred to perinatal centers before delivery, thereby significantly decreasing neonatal morbidity and mortality rates. Proper antenatal care ensures at the end of pregnancy, a healthy mother and a healthy baby, but the coverage of antenatal care in India remains inadequate.

There are several factors, both maternal and others that influence pregnancy at various stages. The maternal health and the uterine environment have a good say in deciding the successful birth of a healthy infant. Infections by intrauterine pathogens have been found as significant in causing various pregnancy related complications all over the world, in various degrees. Similar studies conducted in India are reported as very few. In Kerala, especially in the northern region, it is doubtful whether such an attempt has ever been made, other than the individual screening for one or the other of these pathogens like Rubella etc, when the pregnant mother sometimes is

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suspected to have exposed to it, in order to rule out the doubt. Hence this study focuses on the incidence of intrauterine infections by TORCH pathogens and their role in causing common pregnancy associated complications like miscarriage, IUGR, IUD and congenital malformations among the pregnant women of Malabar area of Kerala state, where childhood marriages and teenage pregnancy have been represented as a common phenomenon. Parameters like PIH, Oligohydramnios, parity, gravida, age, diabetes, other factors that may contribute to pregnancy complications were also considered to eliminate their possible role.

Knowledge of the role of the intrauterine pathogens in causing pregnancy related complications is expected to contribute to the future pregnancy care in the area.