"A STUDY ON PREGNANCY OUTCOME IN WOMEN WITH FIRST TRIMESTER BLEEDING PER VAGINUM"

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For the award of the degree of

M.S. OBSTETRICS AND GYNAECOLOGY

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DECLARATION BY THE CANDIDATE

I here by declare that this dissertation entitled "**A STUDY ON PREGNANCY OUTCOME IN WOMEN WITH FIRST TRIMESTER BLEEDING PER VAGINUM"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.S.BAMA M.D.,D.G.O.** Associate Professor, Department of Obstetrics and Gynaecology, Coimbatore Medical College, Coimbatore .

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This is to certify that this dissertation entitled "**A STUDY ON PREGNANCY OUTCOME IN WOMEN WITH FIRST TRIMESTER BLEEDING PER VAGINUM"** is a bonafide research work done by **DR.S.SHANTHI SUSEENTHIRAN** in partial fulfillment of the requirement for the degree of **M.S in OBSTETRICS AND GYNAECOLOGY**, Coimbatore Medical College, Coimbatore.

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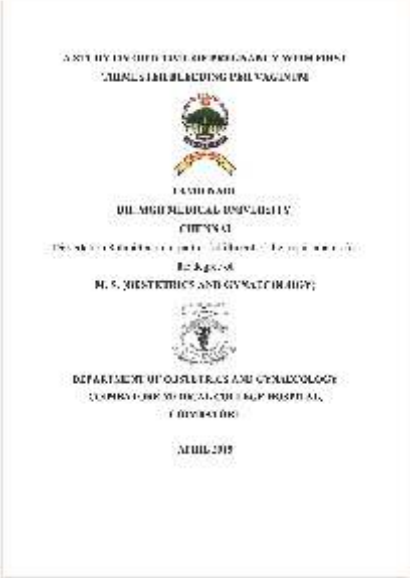

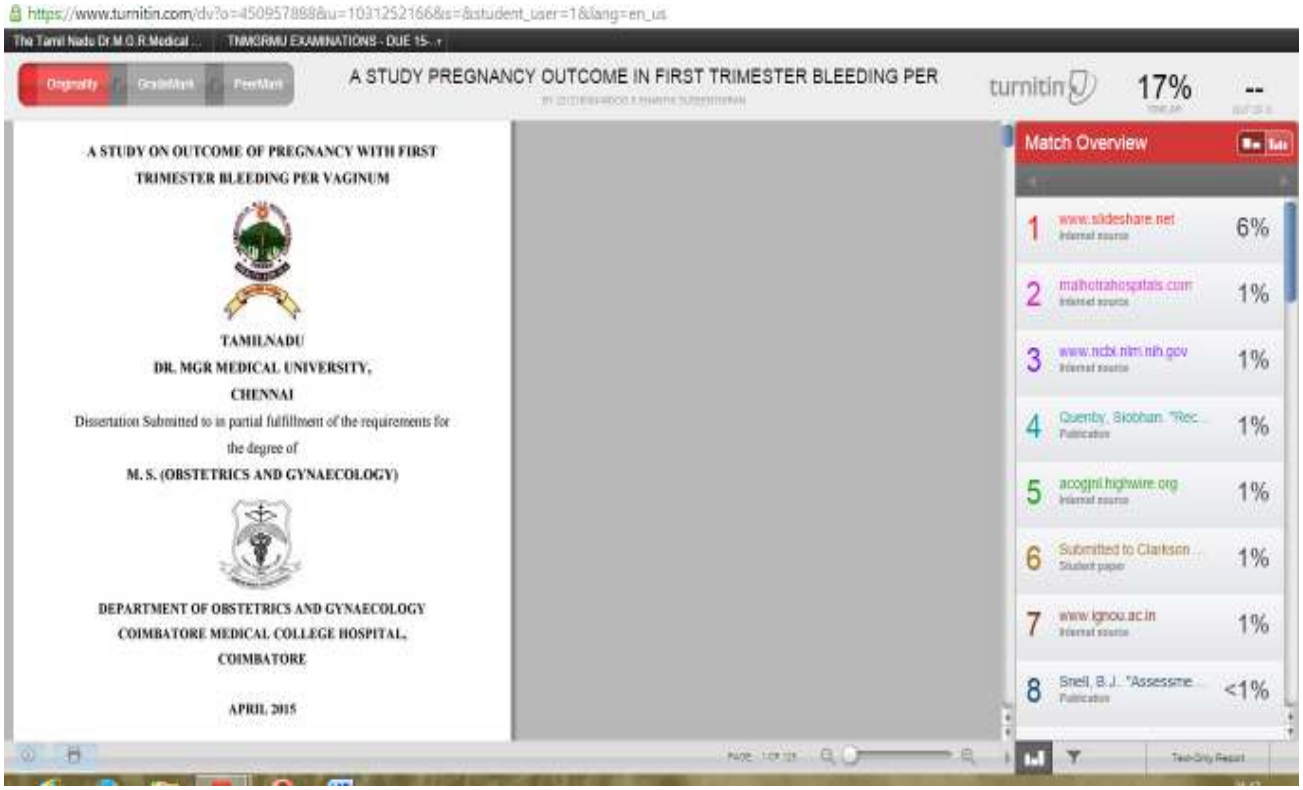
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LIST OF ABBREVIATIONS USED

USG	Ultrasonography
SCH	Sub chorionic hemorrhage
FTND	Full term normal delivery
PTND	Pre term normal delivery
MAS	Meconium aspiration syndrome
BA	Birth asphyxia
PREM	Pre maturity
NJ	Neonatal jaundice
D&C	Dilatation and curettage
F	Female
M	Male
APS	Anti phospolipid Antibody syndrome
PIH	Pregnancy induced hypertension
GDM	Gestational diabetes
LDA	Low dose aspirin
LH	Luteinizing hormone

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INTRODUCTION

Vaginal bleeding is common in early pregnancy , and has been associated with adverse pregnancy outcomes. Many studies of vaginal bleeding rely on maternal self-report during late pregnancy or after delivery. Prior studies have found that recalled data may be influenced by outcome and other events in pregnancy. No studies have evaluated the outcome of vaginal bleeding in pregnancy in first trimester briefly.

Threatened miscarriage is a common and poorly understood, adverse pregnancy outcome. Understanding the nature of common biological processes, symptoms, and behavioral changes that occur during early pregnancy may contribute to increased knowledge of miscarriage risk factors. The relationship between vaginal bleeding and miscarriage occurring in an estimated 20% of all pregnancies remains unclear.

Although vaginal bleeding occurs commonly in early pregnancy and may mark a miscarriage event, it is not always associated with imminent pregnancy loss. Few studies have investigated the prevalence and predictors of bleeding. Estimates

of bleeding prevalence in early pregnancy are imprecise and range from 7 to 24%.

Bleeding in the first trimester can originate from uterus, cervix, vagina or it can be extragenital. Thorough physical examination is essential to differentiate between genital and extra genital causes. It may be a normal sign of implantation of pregnancy, may be initiation of spontaneous miscarriage or may be the sign of a pathological condition such as ectopic pregnancy or Gestational trophoblastic disease.

Approximately 18% women with vaginal bleeding in first trimester have a sonographically demonstrable subchorionic hematoma.

This study analyzes the outcome of pregnancy with first trimester bleeding per vaginum.

AIM :

To find out the effect of threatened miscarriage on pregnancy outcome.

OBJECTIVES:

To find out the percentage of pregnant women with first trimester bleeding per vaginum which end up in first trimester miscarriage , second trimester miscarriage, preterm labour, and full term labour.

REVIEW OF LITERATURE

Vaginal bleeding at any stage of pregnancy is an alarming event that generates significant concern in both patient and doctors.

The introduction of USG into obstetrical practice has been extremely useful in providing a better understanding of the etiology of first trimester bleeding per vaginum and basis for its clinical classification and management.

CAUSES

- Idiopathic
- Vaginal and cervical pathology
- Inflammatory conditions
- Implantation bleeding
- Infection
- Subchorionic hemorrhage
- Luteal phase defect
- Maternal comorbidity like diabetes, Hypertension.

- Endocrine deficiency

- Iatrogenic-invasive procedures like amniocentesis and Chorionic Villus sampling.

PATHOPHYSIOLOGY

NORMALLY

Rapid growth of gestational sac during early pregnancy

↓

Tearing of blood vessels around the sac.

↓

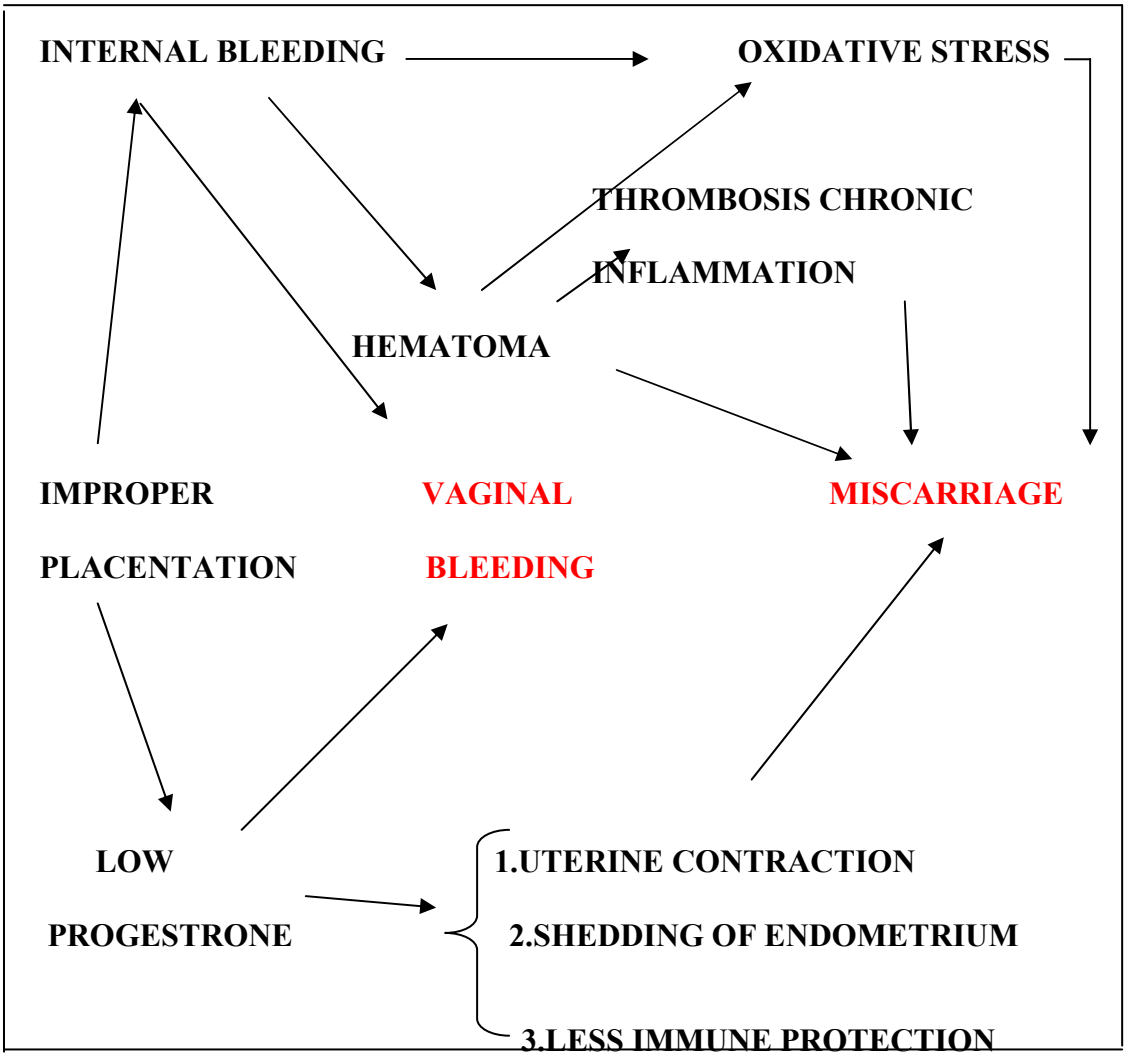
Bleeding

↓

Travels into vaginal canal and seen as spotting. This type of hemorrhage, physiologically usually clears up by second trimester of pregnancy.

In early pregnancy the rapid growth of the gestational sac tear the blood vessels around it and cause bleeding which travels into the vagina and seen as spotting which will clear up in due course.

Conceptual model outlining the relationship between bleeding and miscarriage, *Right From the Start*.



IMPLANTATION BLEEDING

The process of implantation begins approximately 6 days after fertilization. Vaginal bleeding can occur as a result of burrowing of the blastocyst into the uterine endometrium. During this preembryonic stage blastocyst implantation causes a disruption of the endometrial extracelluar environment. This results in bleeding generally a small amount but may be bright red in appearance.

In very early pregnancy, the corpus luteum produces progesterone. The shift to placental production of progesterone occurs by the 7^{th} week of pregnancy. If the decrease in progesterone during this transition period is substantial enough to mimic the progesterone drop at the end of the luteal phase of the menstrual cycle, this might trigger an episode of bleeding. Such an episode may suggest that the early placenta is not performing its function adequately.

The maternal spiral arteries are blocked by a trophoblastic shell during most of the first trimester, maintaining a low oxygen tension for fetal development. Evidence suggests that the early maternal spiral arteries are blocked until the last few weeks of

the first trimester , allowing the fetus to develop in an environment of low oxygen tension. Factors associated with the onset of circulation prior to the development of defence mechanisms against excess oxidative stress have been associated with bleeding.

SUBCHORIONIC HEMORRHAGE

Subchorionic hemorrhage is defined as collection of blood between the uterine wall and the chorionic membrane and may leak through the cervical canal. Believed to be caused by partial detachment of trophoblast from the uterine wall.

Subchorionic bleeding has guarded prognosis that is worse when its occurs before 9 weeks. The main complications are spontaneous miscarriage, premature rupture of membranes, preterm labour, placental abruption and fetal growth restriction.

There are no well defined indications of poor prognosis for the pregnancy but persistent and recurrent bleeding is a poor sign.

CLASSIFICATION OF HEMATOMAS ON THE BASIS OF LOCATION BETWEEN TISSUE LAYERS

1. Subchorionic hematoma – most common type, are between the chorion and endometrium.

2. Retroplacental hematoma – are entirely behind the placenta and not touching the gestational sac.

3. Subamniotic or preplacental hematoma – are contained within amnion and chorion.

FACTORS USED TO EVALUATE THE OUTCOME IN SCH

- Gestational age at which subchorionic hematoma was present <8 weeks or >8 weeks

- Maternal age at the time of diagnosis – advanced maternal age has more risk

DIAGNOSIS

USG: Best modality.

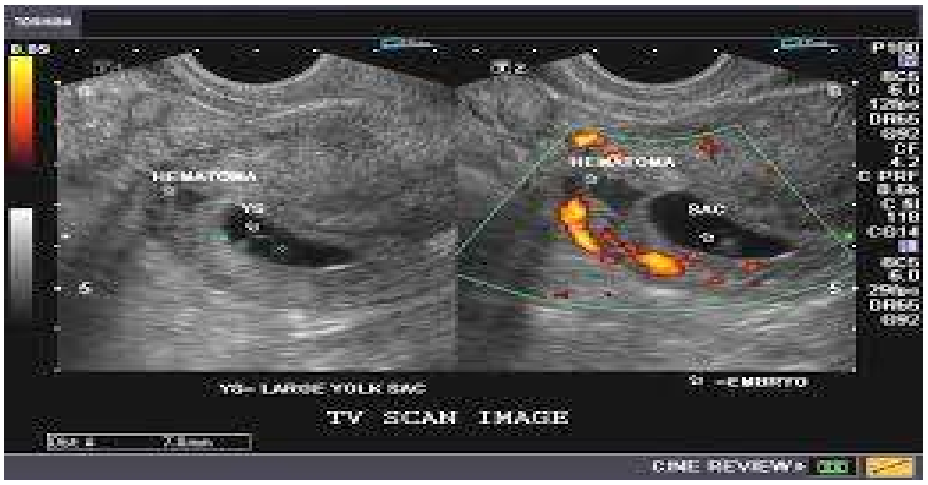


Fig1: USG showing SCH in 1st trimester

Methodius G. tuuli et al[1] conducted a cohort and case control study evaluating subchorionic hematoma and perinatal outcome and concluded that subchorionic hematoma was associated with an increased risk of early and late pregnancy loss, abruption, preterm, and premature rupture of membranes.

Geneviene L Bennett et al[2] retrospectively studied the effects of subchorionic hemorrhage size, gestational age, and maternal age on pregnancy outcome in patients with vaginal bleeding in first trimester of pregnancy and concluded that the spontaneous miscarriage rate was approximately twice as high for women aged 35 years or older against younger women and for

bleeding at 8weeks gestation or less compared with those with bleeding at > 8 weeks gestation.

Robert H ball et al[3] studied that there is no difference when pregnancies with USG documented SCH are compared with those without these hemorrhages.

Gianpaolo Maro et al[4] performed study on 248 cases and pregnancy outcome was correlated with hematoma volume , gestational age and maternal age and concluded the risk of spontaneous miscarriage was related to gestational age and significantly increased if diagnosed < 9 week

The size of the hematoma and the severity of initial bleeding episode have little prognostic value. [28,29]

Data from *Right From the Start* (RFTS), a prospective pregnancy cohort, was used to meet the aims of this project. RFTS enrollment is community-based and studied about early pregnancy. Data from this cohort will answer critical questions related to early pregnancy symptoms an important topic from the perspective of patients, clinicians, and researchers.

Right From the Start (RFTS) cohort.

Some attractive characteristics of this cohort include

1) Comprehensive bleeding episode assessment;

2) longitudinal data obtained on >4000 pregnancies over an 8 year period (2000-2008); and

3) Ascertainment of early pregnancy outcomes, including over 500 miscarriages

A comprehensive first trimester interview is conducted for all RFTS participants, which collects bleeding episode information. A subset of women completed prospective daily diaries, providing information about pregnancy symptoms and exposures prior to pregnancy and throughout the first trimester. Bleeding information collected at the first trimester interview will be compared to the diary reports. This cohort was well designed to answer a broad range of hypotheses related to miscarriage risk factors. It hypothesize that sporadic, light bleeding is a common occurrence of early pregnancy.

These hypotheses will be explored under the following specific aims:

1. Identify the maternal characteristics that predict bleeding in early pregnancy including maternal age, maternal comorbidities, prior birth outcomes and cycle characteristics.

2. Evaluate the association between patterns of bleeding in early pregnancy and the occurrence of miscarriage.

3. Evaluate the extent of agreement between bleeding episodes from retrospective first trimester interviews and prospectively collected data from daily diaries

EPIDEMIOLOGY

Pregnancy loss is the most common adverse pregnancy outcome. Approximately one-third of implanted embryos are lost prior to live birth. Between 20% and 40% of losses are pre-clinical losses, occurring prior to the first missed menstrual period.[26] Miscarriage is defined as the loss of a clinically detected pregnancy prior to twenty completed weeks of gestation. These losses, accounting for about 15% of all clinically recognized pregnancies, can be categorized as early or late losses, depending on whether the loss occurs in the first or second trimester ,

respectively. In addition to the pre-clinical and clinically recognized pregnancy losses discussed herein, it should also be noted that an unknown number of occult losses occur, comprised of conceptions that fail to implant.

These conceptuses, which do not survive more than a few days after conception, are undetectable due to the absence of an easily accessible, specific marker for the pre-implantation embryo. The total number of conceptions that are spontaneously lost before twenty weeks gestation may be as high as 70%.

Embryonic and endometrial characteristics associated with miscarriage:

DEVELOPMENTAL MILESTONES OF EARLY PREGNANCY

GESTATIONAL AGE AFTER LMP	EMBRYONIC MILE STONES
23days	Blastocyst Implantation
3.5 - 4.5 week	Decidual changes at

	Implantation site
4.5 -5.5 week	Exocoelomic cavity
	Of blastocyst
5 – 5.5 week	Yolk sac
5 - 6 week	Embryo
5 -6 week	Cardiac activity

The causes of miscarriage are unclear. About half of early losses are thought to be due to fetal chromosomal abnormalities leading to non-viability. Chromosomal abnormalities result from non-disjunction in gamete formation, resulting in early errors in zygote cell division and subsequent complications with blastocyst differentiation. Genetic abnormalities also stem directly from the maternal or paternal genotype as in the case of unbalanced translocations that are passed on from the sperm or egg.

Genetic factors may lead to structural or developmental aberrations in the embryo, slowing growth and progress towards subsequent stages in development such as implantation. Because the endometrial environment is dynamically changing throughout the cycle, if processes such as Implantation are delayed

substantially, the endometrial environment may not adequately support the embryo. A larger proportion of embryos that undergo late implantation (more than nine days after ovulation) are subsequently lost. Furthermore a chromosomally abnormal embryo that implants successfully may not express necessary factors or respond to signaling molecules at appropriate times during development, eventually leading to fetal demise.

An interplay between the chromosomal makeup of the embryo and the endometrial environment contributes to the occurrence of pregnancy loss. Factors affecting endometrial receptivity , particularly the uterine environment around the time of implantation, account for a proportion of those pregnancy losses that cannot be directly related to genetic abnormalities. Defects in the early processes of implantation, invasion into the myometrium or access to the uterine vasculature may contribute to pregnancy loss weeks or months after the event. The site of implantation in the uterus also plays a role in pregnancy viability. The implantations occurring in the middle and lower regions of the uterus more likely to miscarry.

To summarize the events and environment of very early pregnancy, influence the eventual outcome of the pregnancy, from

an epidemiologic and clinical perspective, it is difficult to assess the extent to which these early, often unobservable, events predispose to an outcome like miscarriage. Because of this, the majority of epidemiologic studies of the causes of miscarriage have focused on maternal characteristics that confer an inreased risk of miscarriage,

MATERNAL CHARACTERISTICS ASSOCIATED WITH MISCARRIAGE

Studies of maternal factors have uncovered a variety of characteristics associated with miscarriage. In general, miscarriage risk increases with increasing maternal age and number of prior miscarriages. These trends may be the result of an increased frequency of age-related errors in DNA replication, other aspects of oocyte and embryo quality or a uterine environment that is less amenable to the development of the embryo. Other maternal factors thought to affect the risk of miscarriage include structural uterine anomalies such as bicornuate uterus or benign tumors, such as fibroids. These structural malformations physically interfere with the ability of the conceptus to implant or grow in the uterus due to their spaceoccupying effect.

MATERNAL COMORBIDITIES

It has been investigated particularly in populations of women with recurrent miscarriage. Women with thyroid disturbances, autoimmune diseases, thrombophilic defects, and other systemic disorders such as polycystic ovarian syndrome have an increased risk of miscarriage and decreased fertility. Similarly maternal obesity and poorly controlled diabetes have also been linked to miscarriage. Other hormone alterations may also be related to miscarriage including luteal phase defects. This condition is characterized by low progesterone production by the corpus luteum, resulting in miscarriage or reduced fertility due to an inability to maintain pregnancy. These factors contribute to a suboptimal uterine environment and decreased endometrial receptivity.

POLYCYSTIC OVARIAN SYNDROME

Patient suffering from PCOS may have an increased risk for miscarriage. The mechanism of the pregnancy loss in women who have polycystic ovaries is uncertain. However it has been proposed that hyper secretion of LH causes premature ageing of

the oocyte. Ovulation induction and prevention of LH hypersecretion are mainstay of treatment.

ANTI THYROID ANTIBODIES

The presence of anti thyroid antibodies has been associated with a higher pregnancy loss rate. The underlying mechanisms of which are either auto immune or mild thyroid insuffiency.

Auto immune events are thought to account for 5 to 40% of recurrent pregnancy loss . The mechanism of this processs is loss of self tolerance. The vast majority of pregnancy losses are from anti phospholipid antibodies. Death occurs in the first trimester frequently after cardiac activity has been detected. The incidence of anticardiolipin antibody in recurrent pregnancy loss is 2 to 5% and positive LA is 0.3 to 2 %. There is a large amount of overlap with both of these antibodies being positive in 60% of the cases. It causes pregnancy loss by inhibition of the thrombolytic system, thrombosis, infarction of the placenta and abnormal prostacylin metabolism.

Anti phospholipid antibody syndrome has a prevalence of 15% in women with first trimester recurrent miscarriage.

Treatment options including low dose aspirin ,heparin, prednisolone and intravenous immunoglobulin. The systemic review showed that prednisolone and iv immunoglobulin do not improve pregnancy outcome and are associated with increased risk of diabetes and premature birth and concluded that low dose aspirin was not of significant benefit but a combination of LDA and unfrationed heparin reduced pregnancy loss by 54%. Thus LDA and heparin are the recommended treatment for women with recurrent miscarriage and APS. In clinical practice low molecular weight heparin are preferred as they have reduced risk of thrombocytopenia and only need once daily administration and levels need not be monitored

Menstrual cycle length and regularity may be related to pregnancy loss. Specifically long cycles have been associated with miscarriage. Short cycles and irregular cycles have also been associated with miscarriage. The relationships may be modified by other systemic factors such as obesity.

MATERNAL INFECTION

Infection may play a role in miscarriage. Asymptomatic bacterial vaginosis may be associated with second trimester miscarriage[5,6] and some evidence also indicates that oral infections

and placental inflammation may be related to late miscarriage. Maternal behaviours and occupational factors have also been suggested to increase the risk of miscarriage. Work schedule particularly working at night or working overtime during the first trimester has been associated with increased risk of miscarriage. Work -related stress have also been found to be related to a higher risk of miscarriage.

Both active smoking and exposure to environmental tobacco smoke have been associated with miscarriage. This may result from both reduced maternal fertility and altered endometrial receptivity in a population of women undergoing invitrofertilization, heavy smokers were at less chance to achieve pregnancy.

Maternal dietary exposures including alcohol and caffeine exposure have also been associated with increased risk of miscarriage. Additionally some studies found an increased risk of miscarriage for caffeine exposure that occurred prior to pregnancy regardless of consumption during pregnancy. Certain medication exposures have also increases the risk of miscarriage including non-steroidal anti-inflammatory drugs and some classes of anti-depressants.

PATERNAL CHARACTERISTICS ASSOCIATED WITH MISCARRIAGE

A smaller literature outlines the relationship between paternal characteristics and miscarriage. Because the sperm contributes half of the genetic make-up of the embryo, a substantial proportion of genetic abnormalities related to miscarriage are derived from paternal factors. Paternal factors may affect chromosomal and structural abnormalities in the sperm. Additionally some investigators have found a link between paternal age and miscarriage likely to mediated by sperm quality. Paternal environmental exposures and behaviours are also thought to play a role.

Previous studies:

Three reports attempt to describe early pregnancy bleeding patterns in a pregnant population by Yang et al[7], Harville et al[8], and Axelsen et al[9] Most participants were enrolled in conjunction with clinical care. These studies are limited by retrospective data collection, second trimester recruitment from prenatal clinics and small sample size.

The Yang analysis was based on a clinic-based population of pregnant women (n=2800) who reported their early pregnancy bleeding patterns at the end of the second trimester (26 to 30 weeks of pregnancy).1 This study found that 25% of women reported vaginal bleeding during pregnancy with peak incidence during the first completed month of pregnancy. Because bleeding assessment occurred in a telephone interview conducted around the 28th week of gestation, the extent to which these results are affected by recall error is unclear. The timing of bleeding was not assessed in detail with episodes reported in monthly intervals. Additionally only those women whose pregnancies continued to the mid/late second trimester (20 to 26 weeks) were included eliminating all women who had a miscarriage. This study focused on later pregnancy outcomes such as preterm birth.

The Harville analysis focused the reports of 14 women (9% of total n = ++151) who prospectively reported bleeding symptoms during the first eight weeks of pregnancy . Twelve of the fourteen women with bleeding continued to live birth. Bleeding was not associated with miscarriage in this study. This study also found no evidence for the presence of implantation

bleeding. Although the details obtained from this study are useful this study is limited by the small numbers of participants and data collection only through the eighth week of gestation. This study is the only prospective longitudinal description of daily bleeding patterns in very early pregnancy.

The Axelsen study analyzed a group of Danish women in prenatal care.[9] About 20% of participants (n=1091) reported bleeding in a 16 week questionnaire. The median week of first occurrence of bleeding was eight weeks. Although this study is population based (97% of women in their area receive care at the prenatal clinics) and has ~ 6800 participants, the analysis only includes data from 13 women whose pregnancies progress to live birth lacking a complete ascertainment of all pregnancies in the population (such as those ending in miscarriage or fetal death). The focus of this study is primarily on the relationship between recalled bleeding and later outcomes such as preterm birth.

Two additional studies published over 30 years ago provide some descriptive information about the incidence and patterns of vaginal bleeding in early pregnancy. These studies describe the timing of bleeding and maternal characteristics associated with increased bleeding occurrence.

One of the studies found that approximately 27% of pregnancies with vaginal bleeding result in miscarriage. Unfortunately, neither of these publications contain a complete method of selection, no details regarding data collection procedures or sample recruitment are provided making it difficult to assess the validity of their results.

Based on this review of the early pregnancy bleeding literature, it is clear that little data exists that would be relevant for miscarriage as an outcome. The prevalence of bleeding reported by these studies is wide (7-25%).This basic information needs to be clarified before undertaking additional analyses of the relationship between bleeding symptoms and pregnancy outcomes such as miscarriage or preterm birth.

Predictors of bleeding:

Most of which have evaluated predictors using unadjusted analyses. Only one previous study has systematically investigated the maternal predictors of bleeding in a general obstetric population.[7] This research found that women of advanced maternal age, with passive smoking exposure , prior preterm birth, multiple prior elective terminations and prior miscarriages were more likely to experience intense vaginal bleeding as measured

by several characteristics including heaviness, duration, and index of total blood loss.

Another recent study of emergency department visits for vaginal bleeding found that Hispanic and younger (ages 20-29) women had higher rates of Emergency Department visits than other subpopulations studied. This analysis was based on a national database of Emergency Department visits and may reflect national patterns in access to care[10]. Other studies have also reported unadjusted associations with increasing maternal age, minority race/ethnicity , prior obstetric outcomes (induced miscarriage, stillbirth, preterm delivery), or use of assisted reproductive technologies.

BLEEDING AND PREGNANCY OUTCOMES:

Although the underlying source of bleeding episodes remains unclear, the relationship between bleeding symptoms in pregnancy and various pregnancy outcomes have been investigated.[11] Studies of adverse outcomes in both early and late pregnancy are briefly reviewed. Reports of bleeding between 8 and 12 weeks of pregnancy were of interest because this is the time that maternal –fetal circulation begins to develop.

Miscarriage:

Women who present to the clinic or emergency department with early pregnancy bleeding are usually considered to have a 'threatened miscarriage'. Approximately 35 - 66% of women hospitalized with threatened miscarriage proceed to miscarriage.[12.] Women with threatened miscarriage and ultrasound detected fetal cardiac activity have a lower risk of miscarriage ranging from 5 to 23%[23,24] These reports of the risk of miscarriage are based on clinical populations whose symptoms and outcomes are collected retrospectively in obstetric clinics or emergency departments. Most studies have reported some relationship between early pregnancy bleeding and miscarriage.

CART analysis evaluated the relationship between miscarriage and the following characteristics of first trimester bleeding : heaviness , duration, color , timing , and associated pain.

Consistent with the hypothesis that bleeding is a marker for placental dysfunction, bleeding is most likely to be seen around the time of the luteal –placental shift.

Gracia and colleagues found that a complaint of bleeding was associated with miscarriage in their study population

recruited in an urban emergency department (OR 7.4, 95% CI 5.7, 9.4)[13]

Weiss et al. conducted a similiar analysis among women presenting for prenatal care in several sites throughout the country and reported an OR of 2.5 (95% CI 1.5, 4.3) for the relationship between light bleeding and miscarriage and an OR of 4.2 (1.6, 10.9) for heavy bleeding miscarriage[14]

Chung et al[15], who reported that vaginal bleeding similar to menses was associated with miscarriage (OR of 10.5; 95% CI 1.5, 74.4) when compared to light bleeding. This study was conducted among 1000 consecutive bleeding cases presenting to a university hospital.

Tongsong et al[16] reported a risk ratio (RR) of 2.9 (95% CI 1.1, 8.0) for the relationship between first trimester threatened miscarriage and miscarriage.

Strobino and Pantel –Silverman published a report showing that moderate or heavy bleeding was related to pregnancy loss of both a normal (OR 3.6; 95% CI 2.1, 6.2) and abnormal karyotype (OR 4.9; 95% CI 2.1, 11.6)[17] However slight bleeding was only associated with a miscarriage of a normal karyotype (OR 2.7;)

However these studies have important limitations. No uniform definition of bleeding has been used in the literature; some studies focused on bleeding quantified by number of pads used and other studies included light spotting in their bleeding definition. Most studies were prenatal clinic or hospital / emergency department based studies of pregnant women seeking care. Recruitment only from prenatal clinics is especially difficult for studies of miscarriage because many miscarriages occur before entry to prenatal care. Additional recruitment in a hospital setting only captures the most serious episodes of bleeding that occur as a direct consequence to miscarriage. Thus to have complete ascertainment of all women experiencing bleeding during pregnancy, a community or population based recruitment design is preferred, permitting enrollment of participants very early in pregnancy before entry to prenatal care.

Another drawback of the published literature is that presence of vaginal bleeding was an eligibility criterion in almost all of these studies. Because only women with bleeding were assessed the conclusions that can be drawn from these studies are limited. These studies do not have an appropriate comparison group to which the risk of miscarriage can be compared. Many

miscarriages are not associated with any symptoms of bleeding. Some of the studies categorized different 'types' of bleeding (such as light , heavy , etc.) in order to create different groups for comparison.

The study by Weiss and colleagues was the only analysis that used a general clinic based population of pregnant women rather than focusing only on those with bleeding symptoms [20] This study enrolled participants between 10 and 14 weeks of pregnancy not accounting for pregnancy losses occurring prior to that time. The reported results are for the relationship between bleeding in the month prior to enrollment and second trimester miscarriage and the overall focus of the study is primarily on the effect of bleeding and later pregnancy outcomes.

A case control study by Strobino and colleagues likewise only reports on the relationship between first trimester bleeding and second trimester fetal loss (defined as loss occurring up to 28 weeks of gestation).[17]

Later pregnancy outcomes:

Vaginal bleeding in early pregnancy has also been related to a variety of outcomes that occur later during pregnancy.

Studies have focused on preterm birth, small for gestational age births[18] low birth weight [2,19] placental abruption and rate of Caesarean section.[20.] This has been systematically reviewed.[11] It is clear that vaginal bleeding is of interest not only with regards to early pregnancy outcomes, but also for later outcomes [31,32,33.]

BIOLOGIC MECHANISMS :

A bleeding episode in pregnancy may be associated with a variety of pregnancy outcomes including miscarriage. Evidence from the basic science literature provides some insight into biologic mechanisms that may underlie between bleeding and miscarriage.

Miscarriage is a disorder of placentation. A hormonally functional placenta begins to produce sufficient amounts of progesterone to support the pregnancy around the 7th week of gestation. Progesterone plays a vital role in maintaining pregnancy by preventing uterine contractility , maintaining the endometrium, and altering the maternal immune response to prevent rejection of the embryo. If sufficient amount of progesterone are not produced miscarriage may result.

Some evidence also suggests that during the first ten weeks of gestation the fetus develops in a largely hypoxic environment.[21] The gestational sac serves as a barrier to prevent oxygen transfer to the fetus whose metabolism is largely anaerobic during this time. Additionally extravillous trophoblastic cells of the fetus migrate to the edge of the intervillous space during most of the first trimester to plug the spiral arteries and seal off the intervillous space. This creates a trophoblastic shell that protects the fetus from the maternal blood supply.

Furthermore at this time the spiral arteries are narrow high resistance vessels that inhibit blood flow. These barriers between the maternal and fetal circulation create a physiologically hypoxic environment during early pregnancy. Early onset of maternal and fetal circulation may expose the fetus to high level of oxidative stress.[21] Specifically free oxygen radicals interact with lipids proteins, and DNA to destroy membranes and contribute to cellular dysfunction and cell death. Overall oxidative stress damages fetal tissues, disrupts organogenesis and affects other developmental processes during this critical period of pregnancy.

At about ten weeks of gestation the plugs located at the periphery of the placenta begin to disintegrate and maternal, fetal

circulation begins in the intervillous space. The spiral arteries of the placenta transform into low resistance vessels to accommodate increased blood volume. By fourteen weeks of pregnancy maternal blood flows freely into the placenta permitting the exchange of nutrients and other essential factors. By this time fetal antioxidant enzymes are functional providing the fetus with additional defense mechanisms to maintain the balance of oxidative factors.

A proportion of miscarriages may result from premature onset of maternal blood flow and fetal exposure to oxidative stress. Due to defective placentation the trophoblastic shell may be fragmented and inadequately prevent the entry of maternal blood into the intervillous space. Premature onset of circulation exposes the fetus to the damaging effects of free oxygen radicals. Markers of oxidative stress were increased in miscarriage tissues compared to others.[21]

Bleeding into the intervillous space may also lead to subchorionic bleeding which may be clinically observed as vaginal bleeding or observed on ultrasound. Subchorionic bleeding has been associated with increased production of free oxygen radicals and may exert a mechanical space occupying effect that

interferes with fetal presence in the uterus. Furthermore, subchorionic bleeding may cause a chronic inflammatory reaction and uterine contractions that directly lead to miscarriage.[27,28,29]

Extra-cellular matrix degradation may also destabilize and weaken fetal membranes increasing the likelihood of pregnancy loss .These defects in placentation probably originate very early in gestation; the processes described may relate to anomalies of implantation or early fetal cell organization .

Miscarriage and bleeding associated with other physiological changes:

Bleeding could also lead to a cascade of other events that may be involved in the pathophysiology of miscarriage. A hematoma may result in an inflammatory reaction leading to uterine contractions and loss of pregnancy. Some studies have described links between cytokine imbalance and bleeding and miscarriage. Previous work suggested that a Th2 –based immune response may be characteristic of women with miscarriage or threatened miscarriage . Immune and inflammatory mediators may be altered during threatened miscarriage , although no definitive conclusions exist. Infection may mediate the relationship between bleeding and miscarriage. Infection during pregnancy has been

implicated as a factor underlying a variety of adverse outcomes including preterm birth and may predispose to some of the previously mentioned immune alterations.

Investigations of the role of infection in the early pregnancy have concluded that bleeding in pregnancy may be the only symptom related to a concurrent underlying infection of the reproductive tract by opening access to areas of the reproductive tract that were previously inaccessible to pathogens.

Endocrinological changes occurring in early pregnancy may also be associated with bleeding and loss. Alterations of levels of hormones and metabolic factors among women with miscarriage compared to women with continuing pregnancy. In these comparisons lower levels of human chorionic gonadotrophin and higher levels of thyroid hormone were found among women with bleeding. There may also be a relationship between alterations of thyroid hormone levels and immune function. It is clear that endocrinological changes occur within the maternal system in response to miscarriage and also in response to an episode of bleeding.

PUBLIC HEALTH SIGNIFICANCE

Although vaginal bleeding may be associated with pregnancy loss understanding the characteristics and distribution of first trimester bleeding will clarify its role in predicting adverse outcomes. This project will evaluate the relationship between vaginal bleeding experienced by some women in early pregnancy and outcome Based on the review of the identified literature on this topic to date, it is evident that there is little solid data in this area and that an analysis of RFTS data can contribute a great deal of knowledge to this field.

This research will inform that studies of early pregnancy with bleeding in the first trimester with outcome may provide clues of the gestational/ developmental stages at which bleeding may be most relevant. Clinically any research that gives insight to the processes and mechanisms operating during early pregnancy is useful.

ABNORMAL PLACENTATION

During normal placentation the spinal arteries undergo adaptive changes characterized by loss of the normal musculoelastic arterial wall and replacement by fibrinoid maternal

containing trophoblastic cells. The changes transform the narrow, thick walled arteries into widely open. The vascular channels that provide necessary blood flow for the developing conceptus. The lack of these changes has been named abnormal placentation which is the feature showed by patients with pre-eclampsia, severe growth restriction, preterm labour.

Abnormal placentation may occur in fetuses with normal or abnormal chromosomes. Patients with recurrent miscarriage due to abnormal placentation may occur in features with normal or abnormal placentation. Patients with recurrent miscarriage due to abnormal placentation who are able to prolong a pregnancy beyond the 2nd trimester remain at high risk for pre-eclampsia, preterm labour, and fetal growth restriction. There is evidence suggesting that transvaginal Doppler velocimetry may be useful.

The physical examination of women with second trimester vaginal bleeding may show uterine size in agreement with gestational age but it may be smaller than expected if a fetal demise happened several days before the examination or when fetal restriction is present. Routine lab investigations are usually within normal limits unless bleeding has been severe. Speculum examination may reveal cervical changes consistent with advanced

labour or with uncompetent cervix. In a few cases the medical history and physical examination will help to focus on a few etiology possible but in many occasions the history and the physical examination are unrevealing and it is necessary to make extensive use of lab testing.

Histologic and microbiologic examination of placenta is a fundamental part of evaluation of the patients. The placenta will show extensive acute inflammation changes in patients with ascending infection and typical lesions in patient with chorionic villi which is caused by CMV. Examination of the decidual tissue attached to the placenta will reveal physiologic changes in spinal arteries. Thrombosis of fetal and maternal vessels will be apparent in patients with thrombophilia, particularly antipo-spholipid antibodies and FVL mutations. A mother having a 2nd trimester fetal demise or miscarriage should have a Kleihauer Betke test to rule out the possibility of extensive fetomaternal hemorrhage. The search for connective tissue disorder includes an ANA with ACA and LA of any of these list is positive, further laboratory analysis will be necessary.

After the physical, speculum and laboratory examinations, the patient should have an ultrasound examinations using abdominal and endovaginal transducers.

MISCARRIAGE

Is the termination of pregnancy spontaneous or induced before the period of viability which is now accepted as 20 weeks of gestation or a birth weight of 500 gm. Spontaneous miscarriage is beyond the patient's control. A spontaneous miscarriage in its natural course goes through several stages – threatened , inevitable, incomplete and complete.

THREATENED MISCARRIAGE

The earliest stage is threatened miscarriage where the pregnancy is so far intact but there is an obvious risk to its continuation. The presenting symptom is that of bleeding, which usually precedes the onset of pain. The amount of bleeding is variable but usually never heavy. Pain is usually no more than a mild discomfort at this stage.

On vaginal examination-

The uterus is soft and corresponding to the period of gestation and the internal os is closed. It is in cases of mild bleeding that a conservative attitude is indicated. Vaginal examination may turn a threatened abortion into an inevitable one, or so it may be perceived, although an examination of this vigouris, neither indicated nor necessary. Ultrasound confirm the diagnosis. Any patient, however who continues to bleed, even though slightly, for more than 4 or 5 days must be examined vaginally and by speculum as otherwise from time to time, other pelvic pathological lesions may be missed for example a cervical polyp, erosion or even carcinoma.

MANAGEMENT OF THREATENED MISCARRIAGE

The management is conservative with bed rest and reassurance till the bleeding stops. Sexual intercourse is best avoided. 80% of pregnancies with threatened miscarriage will proceed upto term.

Follow up with ultrasound is essential to detect progression to inevitable or missed miscarriage. The presence of fetal cardiac activity on ultrasound produces a good outcome in 95% of these

women. However therapy with natural progesterone 400 mg in 2 divided doses orally or vaginally is sometimes given in an empirical basis , but there is no evidence to support this. Anti-D is given if the mother is Rh negative and the pregnancy is beyond 12 weeks. There is an increased incidence of prematurity and IUGR in those pregnancies which continue.

INEVITABLE MISCARRIAGE

Following the onset of bleeding , if the patient had pain , the miscarriage had most likely become inevitable.

Bleeding increases significantly as a rule bright , less and more profuse than in cases of ectopic gestation.

On vaginal examination

The uterus is still felt soft and enlarged but the internal os is open through which the products can be felt. If the products of conception can be withdrawn easily by means of a sponge forceps further bleeding may cease and it is even possible that little other than decidual remnants may be found on subsequent curettage. The management is immediate evaluation of the pregnancy. If the patient is in shock, immediate resuscitation with intravenous fluids and blood transfusions is imperative.

If duration of pregnancy is <12 weeks, suction evacuation can be done. If pregnancy exceeds 12 weeks, oxytocin infusion is started and the process of expulsion is hastened. Prophylactic antibiotics are given and anti-D if mother is Rh negative. The products are sent for histopathological examination

In case of inevitable miscarriage:

Management is aimed :

1. To accelerate the process of expulsion.

2. To maintain strict asepsis.

General measures :

Excessive bleeding should be promptly controlled and the blood loss is corrected by IV fluid therapy and blood transfusion.

Active treatment :

Before 12 weeks:

• Dilatation and evacuation followed by curettage of the uterine cavity by using analgesia or under general anaesthesia.

• Alternatively suction evacuation followed by curettage is done.

After 12 weeks :

• The uterine contractions are accelerated by oxytocin drip (10 units in 500 ml of NS) 40 -60 drops per minute. If the fetus is expelled and the placenta is retained it is removed by ovum forceps .

• If placenta is not separated, digital separation followed by its evacuation is to be done under general anaesthesia.

INCOMPLETE MISCARRIAGE :

Early miscarriage :

Dilatation and evacuation under analgesia or general anaesthesia is to be done.

Late miscarriage :

The uterus is evacuated under GA and the products are removed by ovum forceps or by blunt curette. In late cases check curettage to be done to remove the bits of tissue left behind.

The removed materials are subjected to a histological examination. Medical management may be tried .with Tab. Misoprostol 200 µg which should be kept in the posterior fornix every 4^{th} hourly.

COMPLETE MISCARRIAGE:

TVS is useful to see that uterine cavity is empty otherwise evacuation should be done.

MISSED MISCARRIAGE :

Less than 12 weeks :

- Expectant management – many women expel the conceptus spontaneously.
- Medical management – misoprostol 800µg kept vaginally in the posterior fornix and repeated if needed. Expulsion usually occurs within 48 hours.
- Suction evacuation and D& C is done as a definitive treatment or it can be done when the medical method fails. The risk of damage to the uterine walls and brisk haemorrhage during the operation should be kept in mind.

Uterus more than 12 weeks :

- Induction is done by the following methods – misoprostol 200 μg tablet kept into the posterior vaginal fornix every 4 hours for a maximum of 5 doses.
- Oxytocin -10 -20 units of oxytocin in 500 ml of normal saline at 30 drops per minute is started.
- Many patients need surgical evacuation following medical treatment.
- D &C was done once cervix become soft with use of PGE1. Otherwise cervical canal is dilated using the mechanical dilator.

MATERNAL THROMBOPHILIA

Congenital abnormalities of the hemostatic system are frequently implicated as a cause of spontaneous miscarriage, and several studies have indicated a higher risk for pregnancy loss in affected individuals in the second rather than in the first trimester. Evidence suggestive of this etiology exists for women heterozygous or homozygous for the factor V Leiden (FVL mutation, increased activated protein C (APC) resistance,

prothrombin promoter mutation, hyperhomocysteinemia, and protein S deficiency.

INFECTION

Overt intrauterine infection is not a frequent cause of first trimester miscarriage. . Intrauterine infection are usually ascending from the vagina. Patients with ascending infection usually develop temperature elevation and uterine cramps after 14 weeks. This is followed rather quickly by the onset of uterine contractions or by rupture of the membranes. Histologic examination of the placenta shows severe chorioamnionitis. Ascending infections frequently recur in subsequent pregnancies, suggesting the presence of a maternal immunologic deficiency or genetic predisposition.

GENETIC ABNORMALITIES

Chromosomal abnormalities are found in approximately 5-10% of second trimester abortions. In this case autosomal trisomies predominate but fetuses with triploidy and monosomy X are occasionally recognized. In a significant number of second trimester miscarriages extensive investigations including karyotype produce negative results. In these cases, it is suspected that a

lethal mutation undetectable with the presently available methods of analysis is responsible for the problem.

Fetal death may also be secondary to mutations affecting the genes that control the expression of other genes at the transcriptional level (homoeobox genes) or to mutations that cause excessive concentration of products toxic to the embryonic cells.

PROGESTERONE DEFICIENCY

Progesterone deficiency has been an obvious candidate as an etiologic factor of early pregnancy loss because of its well known effect in maintaining uterine quiescence. Unforutnately, progesterone deficiency is overdiagnosed and progesterone supplementation is overused. A vigorous diagnosis of corpus luteum defect requires histologic confirmation of an endometrium out of phase by 2 or more days during the secretory period of the menstrual cycle. Because endometrial biopsies are not obtained during pregnancy the only possible documentation of a corpus luteum deficiency during gestation is by measuring the serum progesterone concentration.

Another problem with the use of serum progesterone levels for the diagnosis of progesterone deficiency in patients with first trimester bleeding or with a history of multiple pregnancy losses is that patients with blighted ova have a low serum progesterone concentration. In these patients, low serum

Progesterone concentration is the result rather than the cause of the miscarriage[22].

DEVELOPMENT OF PLACENTA

The human placenta is discoid because of its shape; haemochorial, because of direct contact of the chorion with the maternal blood and decidua, because some maternal tissue is shed at parturition. The placenta is attached to the uterine wall and establishes connection between the mother and fetus through the umbilical cord. The placenta is developed from two sources. The principal component is fetal which develops from the chorion frondosum and the maternal component consists of decidua basalis.

When the interstitial implantation is completed on 11th day, the blastocyst is surrounded on all sides by lacunar spaces around cords of syncytial cells called trabeculae. From the trabeculae develops the stem villi on 13th day which connect the chorionic plate with the basal plate. Primary, secondary and tertiary villi are successively developed from the stem vill

Arterio-capillary-venous system in the mesenchymal core of each villus is completed on 21st day. This ultimately makes connection with the intraembryonic vascular system through the body stalk.

Simultaneously lacunar spaces become confluent with one another and by 3rd – 4th week form a multilocular receptacle lined by syncytium and filled with maternal blood. This space becomes the future intervillous space. As the growth of the embryo proceeds, decidua capsularis becomes thinner beginning at 6th week and both the villi and the lacunar spaces in the anembryonic area get obliterated converting the chorion into chorion laeve. This is however compensated by a exuberant growth and proliferation of the decidua basalis.

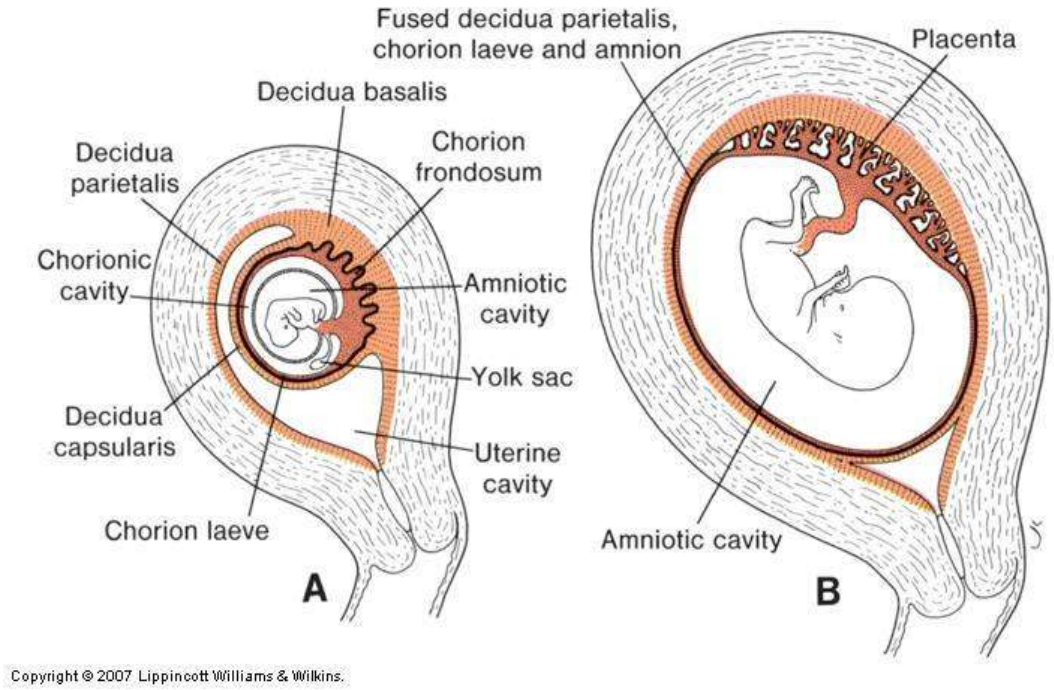


Fig 2 : The relation of the amniotic cavity, chorionic cavity and uterine cavity

THE PLACENTA AT TERM : GROSS ANATOMY

The placenta at term is almost a circular disc with a diameter of 15-20 cm and thickness of about 3 cm at its centre. It thins off towards the edge. It feels spongy and weighs about 500 gm, the proportion to the weight of the baby being roughly 1:6 at term and occupies about 30% of the uterine wall. It presents with two surfaces fetal and maternal and a peripheral margin.

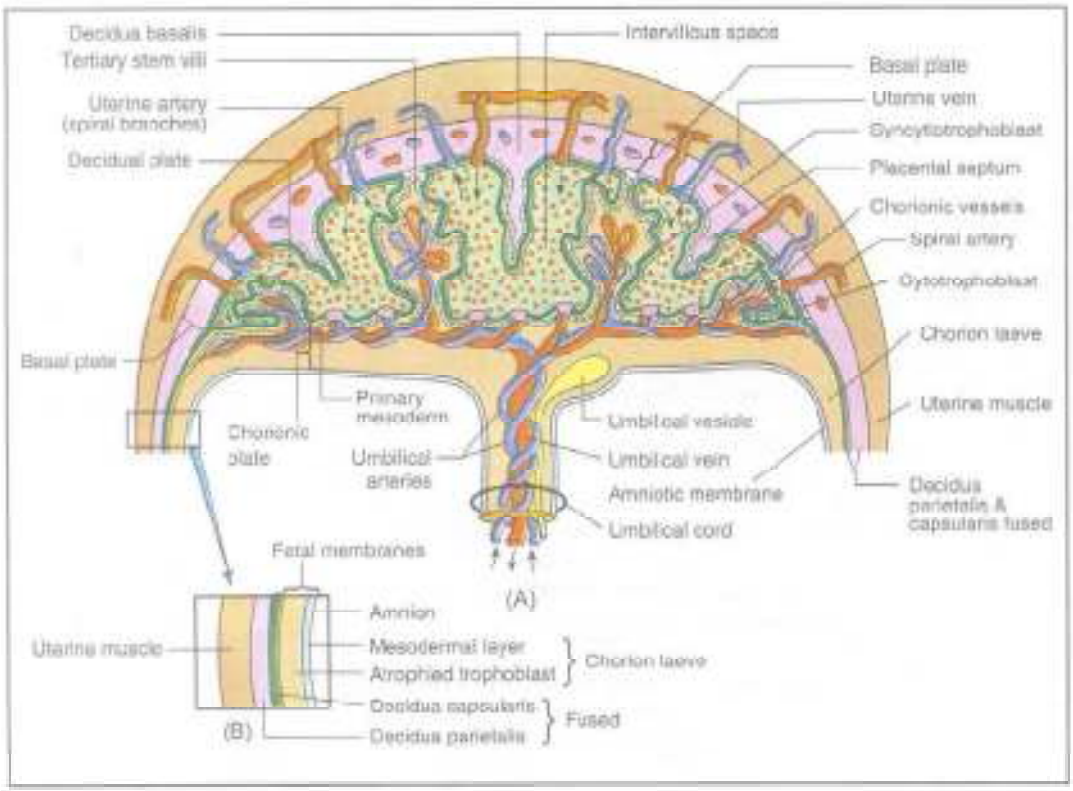


Fig. 3 placenta at term

Fetal surface :

The fetal surface is covered by the smooth and glishening amnion with the umbilical cord attached at or near its centre. Branches of the umbilical vessels are visible beneath the amnion as they radiate from the insertion of the cord. The amnion can be peeled off from the underlying chorion except at the insertion of the cord. At term about four-fifths of the placenta is of fetal origin.

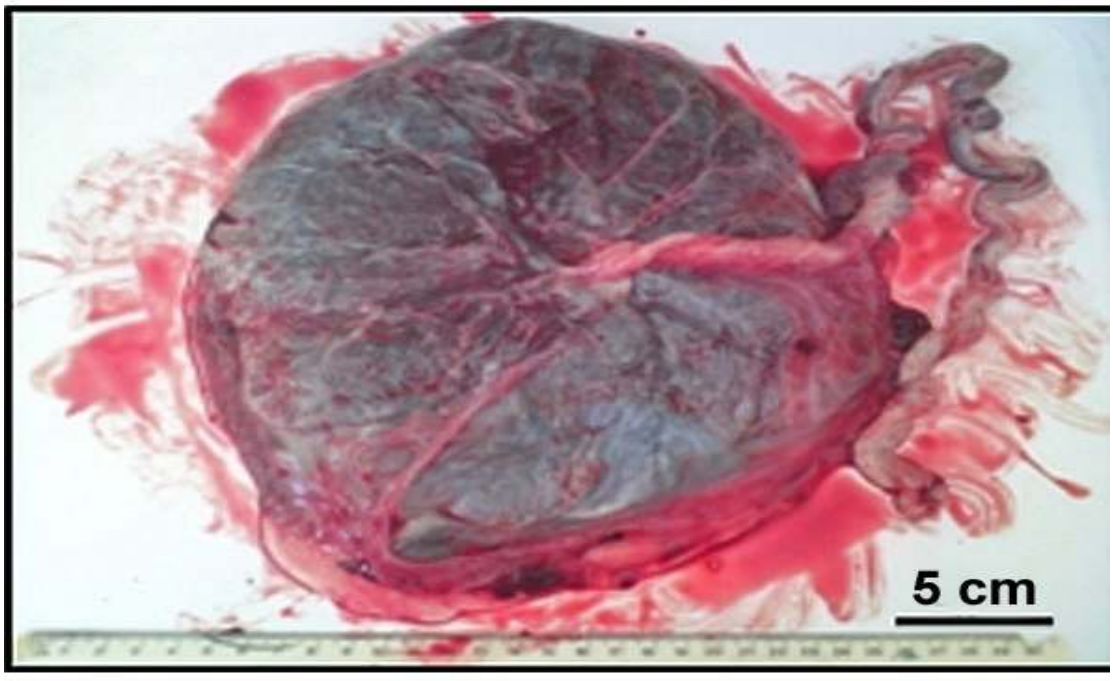


Fig 4 : fetal surface

Maternal surface :

The maternal surface is rough and spongy. Maternal blood gives it a dull red colour. A thin greyish somewhat shaggy layer which is the remnant of the decidua basalis (compact and spongy layer) and has come away with the placenta may be visible.

The maternal surface is mapped out into 15- 20 somewhat convex polygonal areas known as lobes or cotyledons which are limited by fissures. Each fissure is occupied by the decidual septum which is derived from the basal plate. Numerous small grayish spots are visible. These are due to deposition of calcium in the degenerated areas and are of no clinical significance. The maternal portion of the placenta amounts to less than one fifth of the total placenta. Only the decidua basalis and the blood in the intervillous space are of maternal origin.

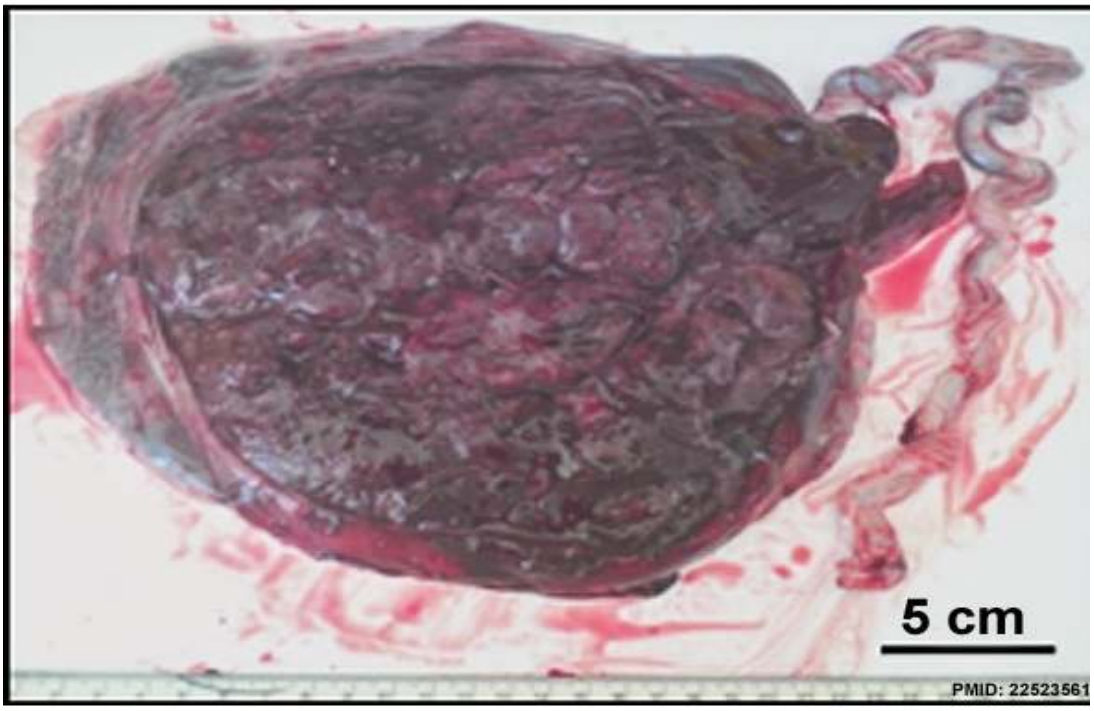


Fig 5 : maternal surface

Margin :

Peripheral margin of the placenta is limited by the fusion of basal and chorionic plates and is continuous with the chorion leave and amnion. Essentially the chorion and the placenta are one structure but the placenta is a specialised part of the chorion.

Attachment:

The placenta is usually attached to the upper part of the body of the uterus encroaching to the fundus adjacent to the anterior or posterior wall with equal frequency. The

attachment to the uterine wall is effective due to anchoring villi connecting the chorionic plate with the basal plate and also by the fused decidua capsularis and vera with the chorion leave at the margin.

Separation

Placenta separates after the birth of the baby and the line of separation is through the decidua spongiosum.

STRUCTURES

The placenta consists of two plates. The chorionic plate lies internally. It is lined by the amniotic membrane. The umbilical cord is attached to this plate. The basal plate lies to the maternal aspect. Between these two plates, lies the intervillous space, containing the stem villi with their branches and the space being filled with maternal blood.

AMNIOTIC MEMBRANE

It consists of single layer of cubical epithelium loosely attached to the adjacent chorionic plate. It takes no part in formation of the placenta.

CHORIONIC PLATE

From within outwards it consists of

(i) Primitive mesenchymal tissue containing branches of umbilical vessel.

(ii) a layer of cytotrophoblast and

(iii) syncytiotrophoblast.

The stem villi arise from the plate. It forms the inner boundary of the choriodecidual space.

BASAL PLATE :

It consists of the following structures from outside inwards.

(1) Part of the compact and spongy layer of the decidua basalis.

(2) Nitabuch's layer of fibrinoid degeneration of the outer syncytiotrophoblast at the junction of the cytotrophoblastic shell and decidua.

(3) Cytotrophoblastic shell.

(4) Syncytiotrophoblast.

The basal plate is perforated by the spiral branches of the uterine vessels through which the maternal blood flows into the intervillous space. At places placental or decidual septa project from the basal plate into the intervillous space but fail to reach the chorionic plate. The septum consists of decidual elements covered by trophoblastic cells. The areas between the septa are known as cotyledons (lobes) which are observed from the maternal surface numbering 15-20.

INTERVILLOUS SPACE

It is bounded on the inner side by the chorionic plate and the outer side by the basal plate limited on the periphery by the fusion of these two plates. It is lined internally on all sides by the syncytiotrophoblast and is filled with slow flowing maternal blood. Numerous branching villi which arise from the stem villi project into the space and constitute chief content of the intervillous space.

STEM VILLI

These arise from the chorionic plate and extends to the basal plate. With the progressive development primary, secondary and tertiary villi are formed. Functional unit of the placenta is

called a fetal cotyledon or placentome which is derived from a major primary stem villi. These major stem villi pass down through the intervillous space to anchor onto the basal plate. Functional subunit is called a lobule which is derived from a tertiary stem villi. About 60 stem villi persist in human placenta. Thus each cotyledon (totaling 15- 29) contains 3 -4 major stem villi. The villi are the functional unit of the placenta. The total villi surface for exchange approximately varies between 10 to 14 square metres. The fetal capillary system within the villi is almost 50 km long. Thus while some of the villi are anchoring the placenta to the decidua the majority are free within the intervillous space and are called nutritive villi. Blood vessels within the branching villi do not anastomose with the neighbouring one.

STRUCTURE OF A TERMINAL VILLUS

In the early placenta each terminal villus has got the following structures from outside inward :

(1) Outer syncytiotrophoblast

(2) Cytotrophoblast

(3) Basement membrane

(4) Central stroma containing fetal capillaries, primitive mesenchymal cells, connective tissue and a few phagocytic (Hofbauer) cells.

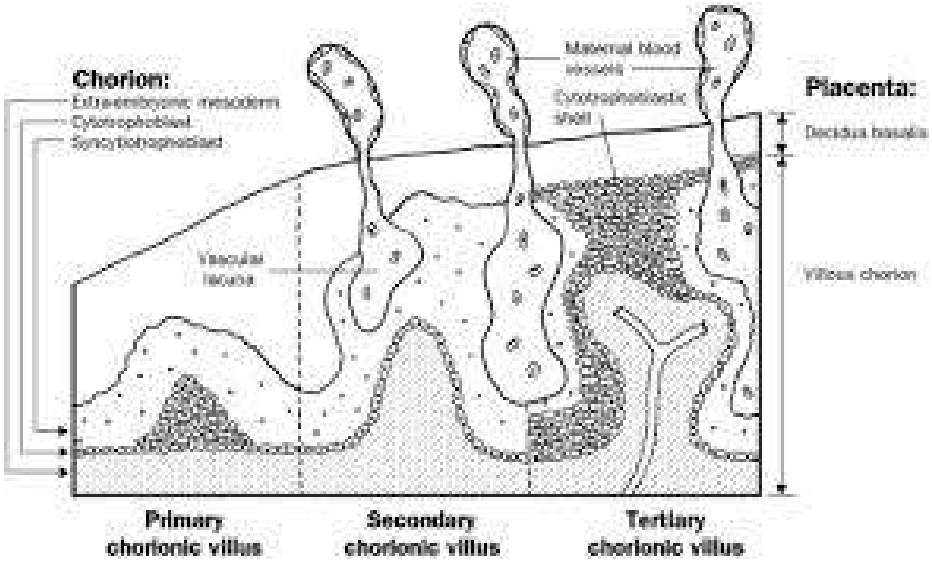


Fig. 6 : development of the villus (a) primary, (b) secondary, (c) tertiary

In placenta at term the syncytiotrophoblast becomes relatively thin at places overlying the fetal capillaries and thicker at other areas containing extensive endoplasmic reticulum. The former is probably the site for transfer and the latter the site for synthesis. The cytotrophoblast is relatively sparse. Basement membrane becomes thicker. Stroma contains dilated vessels along

with all the constituents and few Hofbauer cells. Hofbauer cells are round cells that are capable of phagocytosis and can trap maternal antibodies crossing through the placenta (immune suppressive). These cells can express class II MHC molecules.

PLACENTAL CIRCULATION

Placental circulation consists of independent circulation of blood in two systems :

- Uteroplacental circulation
- Feto-placental circulation

UTERO-PLACENTAL CIRCULATION

(Maternal circulation)

It is concerned with the circulation of the maternal blood through the intervillous space. A mature placenta has a volume of about 500 mL of blood . About 350 mL of blood being occupied in the villi system and 150 ml lying in the intervillous space. As the intervillous blood flow at term is estimated to be 500-600 ml per minute. The blood in the intervillous space is completely replaced about 3 to 4 times per minute. The villi depend on the maternal blood for their nutrition thus it is

possible for the chorionic villi to survive for a varying period even after the fetus is dead. The pressure within the intervillous space is about 10 to 15 mm Hg during uterine relaxation and 30-50 mm Hg during uterine contraction. In contrast the fetal capillary pressure in the villi is 20-40 mm Hg.

Arterial circulation :

About 120- 200 spiral arteries open into the intervillous space by piercing the basal plate randomly at numerous sites. Normally there is cytotrophoblastic invasion into the spiral arteries initially upto the intra decidual portion within 12 weeks of pregnancy. Not only the endothelial lining is replaced but also the musculo elastic media is destroyed and replaced by fibrinoid material. There is a secondary invasion of trophoblast between 12-16 weeks extending upto radial arteries within the myometrium. Thus spiral arteries are converted to large bore uteroplacental arteries. The net effect is funnelling of the arteries which reduces the pressure of the blood to 70-80 mm Hg before it reaches the intervillous space. It thus increases the blood flow.

Trophoblast cells that do not take part in villous structure are known as extravillous trophoblast (EVT).

EVT are of two types :

(i) Endovascular that migrates down the lumen of the spiral arteries and replaces the endothelium.

(ii) Interstitial that invades as far as the inner third of the myometrium. Further invasion is limited by the NK cells to prevent morbid adhesion of placenta (placenta accreta). Defects in trophoblast invasion and failure to establish maternal circulation correctly leads to complications of pregnancy (PIH, 1UGR)[33].

Venous drainage :

The venous blood of the intervillous space drains through the uterine veins which pierce the basal plate randomly like the arteries. This concept of uteroplacental circulation is based on the studies of Ramsey and coworkers (1963,1966). Intervillous haemodynamics is mentioned in table.

Summary of intervillous haemodynamics:

- Volume of blood in mature placenta 500 ml

- Volume of blood in intervillous space 150 ml
- Blood flow in intervillous space 500 -600 ml/minute
- Pressure in intervillous space-
    - During uterine contraction 30-50 mm Hg
    - During uterine relaxation 10-15 mm Hg
- Pressure in the supplying uterine artery 70-80 mm Hg
- Pressure in the draining uterine vein 8 mm Hg

USG. IN FIRST TRIMESTER

USG has been available to evaluate early pregnancy since 1980 and standard criteria have been adopted for its use. Improvement in USG have resulted in increasing use of this modality for management of obstetric patients.

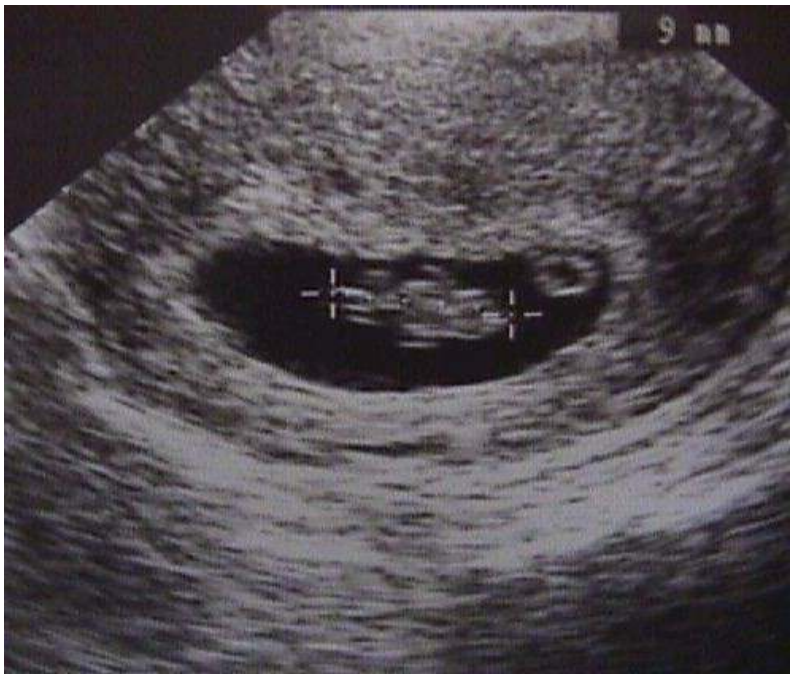


Fig 7 : TVS in early pregnancy

At approximately 4.5 weeks of gestation an intradecidual sac may be seen. The trophoblastic ring appears echogenic and eccentrically located within the endometrial cavity. Definite identification of intrauterine pregnancy is possible as early as 29 to 35 days of gestation. Identificatin of the gestational sac is

made by the double ring or double decidual sign. This refers to the interface between the decidua and chorion and appears as two distinct layers of the wall of the gestational sac.

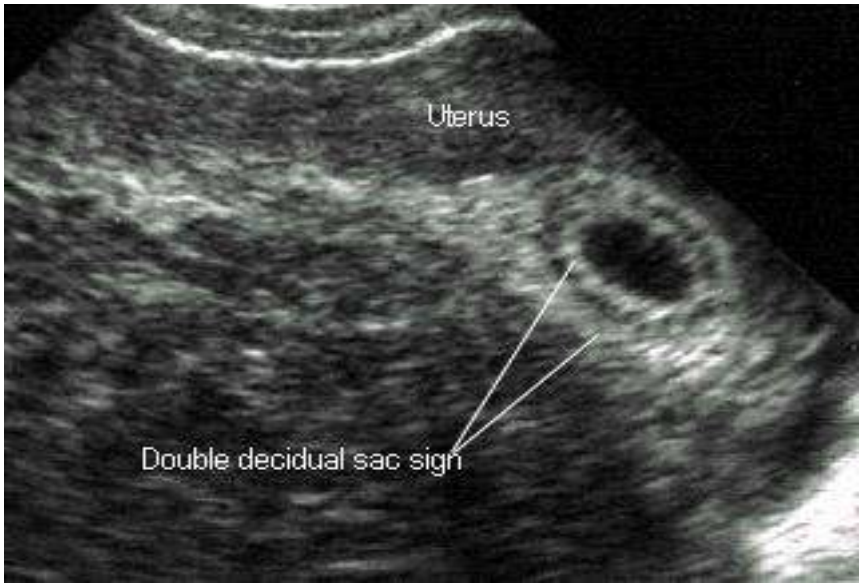


Fig 8 : early gestational sac with double decidual sign

Confirmation of intrauterine pregnancy with 100% certainty is aided by identification of the structures (yolk sac, fetal pole) within the gestational sac.

Early in gestation the endometrium appears prominent. Development of pseudogestational sac may occur before or after the onset of vaginal bleeding as the decidua begins to slough.

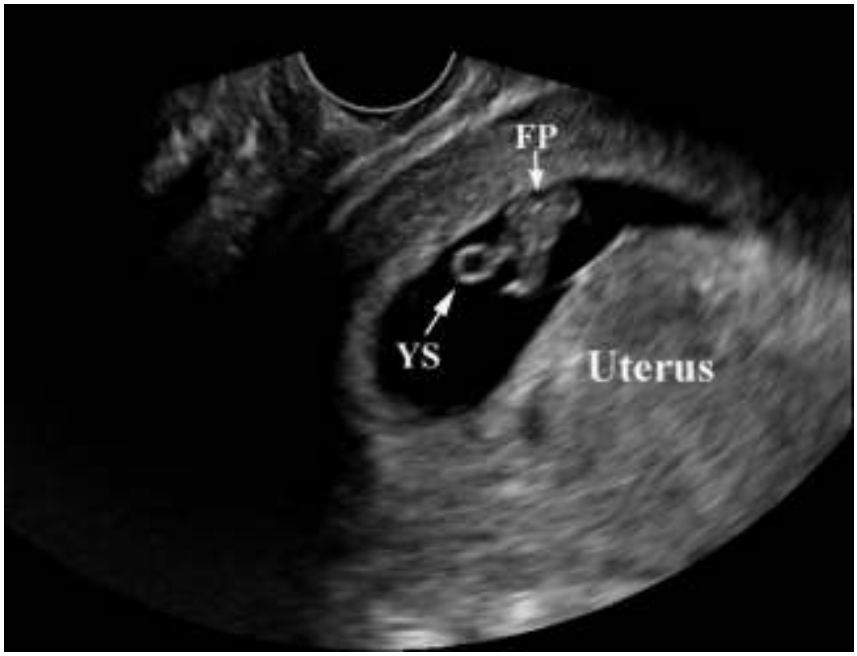


Fig 9 : early gestational sac with yolk sac and fetal pole

STRUCTURES - GESTATIONAL AGE VISUALISED BY TRANSVAGINAL ULTRASONOGRAPHY

Gestational age (Menstrual week)	Structures Visualised
5	Gestational sac, yolk sac
6	Foetal pole, cardiac activity
7	Lower limb buds, physiologic Midgut herniation
8	Upper limb buds, stomach
9	Spine, falx, choroid plexus

Differenciation between a pseudogestational sac or pseudosac and an early intrauterine gestational sac is important.The pseudosac tends to be centrally located in the uterus and may be irregularly shaped and there are no structures present within the pseudosac and double ring sign which signifies the chorion and the decidua interface is absent.

DETERMINE VIABILITY

The mean gestational sac size is determined by measuring the sac in three planes and averaging the values.

- Gestational sac > 12 mm→ yolk sac seen
- Gestational age > 17 mm→ Embryo seen
- Crown rump > 5 mm → Cardiac activity identified

The gestational sac should increase in diameter by 1.1 mm /day.

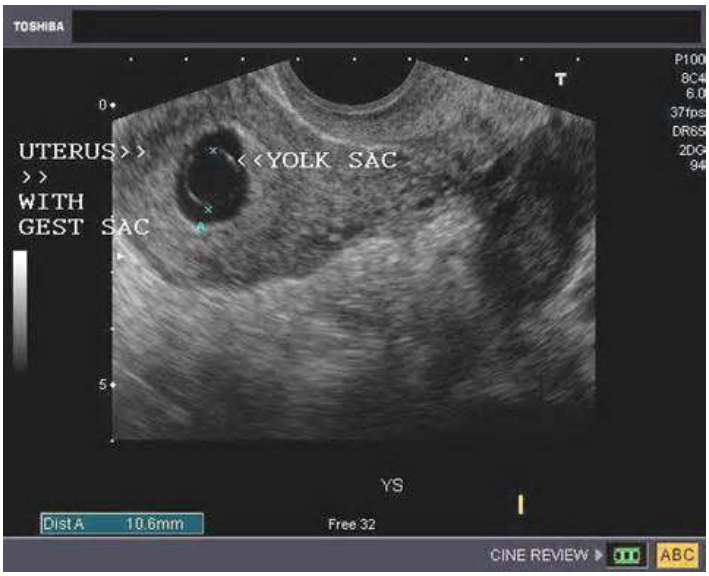
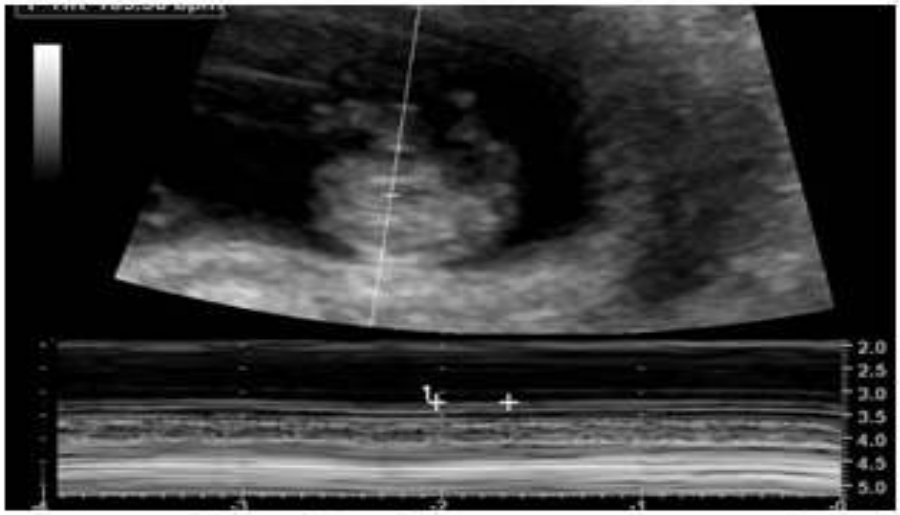


Fig 10 : early gestational sac with yolk sac

Once a fetal pole identified the presence of cardiac activity should be confirmed. The initial heart rate may be slow with a mean of 110 beats per minute at 5weeks of gestation.This rate gradually becomes higher with advancing gestation. In 95% of normal pregnancies cardiac activity detected when the fetal pole is less than 3 mm in length. The pregnancy should not be called abnormal unless absence of cardiac activity is noted, when the Crown rump length is 5 mm or more.



M-Mode of a first trimester fetus demonstrating cardiac activity

Fig 11 : documentation of fetal cardiac activity in doppler :

Once a gestational sac is identified an 11.5% loss rate is reported, the presense of a yolk sac decreases the loss rate to 8.5%, if an embryo is seen the loss rate is 7.2% provided the crown rump length is more than 5 mm (0.5% for embryos larger than 10 mm).

USG criteria of EPF include

- MSD of 13 mm and no yolk sac
- MSD of 18mm and no embroyonic pole
- Embroyo of 4mm and no cardiac activity

- Failure of the gestational sac or embryo to grow at expected rate of 1mm per day
- Loss of previously present cardiac activity

DATING THE PREGNANCY

The scan performed earlier in pregnancy are more accurate for dating is certainly true after 6 weeks of gestation .Dating a first trimester pregnancy by USG is most accurately done by crown rump length, gestational sac size is not as accurate.The crown rump length is more accurate between 7 to 12 weeks of gestation because small differences in caliper placement in early pregnancy may affect the gestational age assignment . Curvature of the fetus is more pronounced as the pregnancy progresses, limiting the accuracy of crown rump length measurement after 12 weeks of gestation

HISTORY

On admission to the labour room a complete but quick history was taken. Information regarding her age, address, socioeconomic status and dietary habits were taken. Enquiry was made regarding usage of drugs, history regarding her previous

antenatal checkups, the kind of antenatal care received and the number of antenatal visits were noted.

History of present complaint:

History was taken in detail so as to ascertain the amount and character of bleeding any initiating factors such as trauma or coitus, previous vaginal bleeding, association of abdominal pain or labour pains, duration between time of onset of bleeding and time of admission and distance traversed to get admitted to the hospital. Period of gestation was calculated from her last menstrual period (nearest number of weeks from the patient's last menstrual period to time of presentation) or previous ultrasonography .

Past History

Any history of associated gynaecological disorders like fibroid, ovarian cyst etc and any previous gynaecological surgery was taken.

Medical history and family history

Any history of hypertension , blood disorders, diabetes mellitus, jaundice etc. were noted .

Menstrual history

Patients last menstrual period, previous menstrual cycle, duration, amount of flow, associated dysmenorrhoea were noted.

Obstetric History

History was taken regarding the number of previous pregnancies and their outcome , number of live issues, complication during previous pregnancies , mode of delivery and complications during and after deliveries. Any previous history of bleeding per vaginum was specifically enquired.

General Physical Examination

- Measuring maternal pulse, blood pressure and respiratory rate.
- Looking for pallor, cyanosis and pedal oedema
- Examination of cardio vascular and respiratory systems

Abdominal examination included -

- Symphysio –fundal height, to ascertain whether the uterine fundus was compatible with the estimated gestational age
- The presence of uterine tenderness and uterine contractions

- The viability, the presentation and engagement of presenting part

- Fetal heart sound was auscultated for rate, rhythm and tone.

Management

All patients presenting with bleeding per vaginum were initially investigated and managed as outlined below, but subsequent management was determined according to the suspected cause, severity and type of bleeding and the 37 gestational age of the pregnancy.

The initial management included the following:

- Routine investigations like CBC, HIV, HBsAg, Blood grouping , BT, CT. were taken

- Depending upon the condition of the patient IV line started if needed.

- Ultrasound scan was done to establish the cause, when maternal and foetal conditions were stable. Presentation, Position of the placenta, Estimated fetal weight were noted.

Management options were -

The subsequent management, based on the above factors and according to

1) Amount of bleeding

2) Gestational age

3) Condition of the patient

4) Fetal viability.

In case of threatened miscarriage :

- The patient admitted in bed for few days until bleeding stops.
- Anxious patient requires some sedation. Relief of pain ensured by diazepam 5 mg tablet twice daily.
- Hormone therapy with IM progestational agents or micronized progesterone (oral or vaginal).
- Lifting heavy weights, sexual intercourse and powerful purgation must be avoided at least until safely past the sixteenth week.

- USG repeated after one to two weeks to ensure all is well with the fetus.

- Even though threatened miscarriages successfully treated the pregnancy continues at a greater risk than normal.

- There is no evidence that treatment with natural progesterone or synthetic progestins improve the prognosis.

- Patient were advised regarding frequent ANCs and scan was done after 1 week and was also explained the risk associated with the condition.

Incomplete / complete miscarriage

Pain and bleeding which are the prominent symptoms of an incomplete miscarriage usually settle down as the miscarriage becomes complete. The internal os was open on vaginal examination in case of an incomplete miscarriage. Once all the products have been expelled out , the os gradually closes down and the uterus can be felt to be much more firmer after a Complete miscarriage . Here 9 cases were ended in incomplete miscarriage.

Management of incomplete miscarriage

Suction evacuation was done under antibiotic coverage followed by USG confirmation

Complete miscarriage

Management is conservative. Anti-D is given because the mother is Rh negative in three cases and it was more than 12 weeks and 6 cases which ended in complete miscarriage.

MISSED MISCARRIAGE

There is intrauterine death of the embryo or non-viable fetus, which is then passively retained inside the uterus. There is a history of amenorrhea followed by vaginal bleeding . The signs and symptoms of early pregnancy slowly disappear. On examination the uterus size is smaller than the period of amenorrhea and the cervix is firm with a closed internal os. Ultrasound show gestational sac smaller than expected with irregular margins. There is fetus with no cardiac activity. If products are retained for a long time, it can lead to sepsis , so antibiotics are started.

Management :

As soon as USG shows missed miscarriage plan for emptying the uterus was decided. This reduces the psychological stress of carrying a dead fetus.

In cases with the uterine size is ≤ 8 weeks gestation PGE1 kept vaginally which results in spontaneous expulsion without any need for surgical intervention.

In cases with > 12 weeks gestation PGE1 kept 6^{th} hourly upto 4 doses results in spontaneous expulsion. In both the cases check curettage done and confirmed by USG.

In case of preterm labour

Following regimen may be instituted in an attempt to arrest premature labour –

- Bed rest - the patient is to lie preferably in left lateral position though the benefits are doubtful.
- Adequate hydration is maintained.
- Prophylactic antibiotic given.

- Tocolytic agents have been used to inhibit uterine contractions.

- Steroid therapy given to the mother to enhance fetal lung maturity such as Inj.Betamethazone 12mg Im 2doses given.

MANAGEMENT OF PRETERM LABOUR

Patient is put to bed to prevent early rupture of the membranes. Then to ensure adequate fetal oxygenation by giving oxygen to the mother by mask. Labour should be carefully monitored. Caesarean section is done for obstetric reasons only. The birth should be gentle and slow to avoid rapid compression and decompression of the head. Episiotomy may be done to minimize head compression if there is perineal resistance. The cord is to be clamped immediately at birth to prevent hypervolaemia and hyperbilirubinaemia. To shift the baby to neonatal intensive care unit under the care of a neonatologist.

PREMATURE RUPTURE OF THE MEMBRANES (PROM)

If pregnancy is less than 34 weeks – expectant management to continue for fetal maturity.

If pregnancy ≥ 34 weeks and <37 weeks to wait for spontaneous onset of labour for 24- 48 hours. If fails induction of labour with oxytocin.

If pregnancy ≥ 37 weeks to wait for spontaneous onset of labour for 24 hours .If fails induction of labour with oxytocin.

Cesarean section should be done for obstetric reasons for above gestations.

CESAREAN SECTION WAS DONE :

1. If unfavourable cervix

2. Other associated obstetrical conditions which themselves were indications for LSCS.

3. There was a appearance of fetal distress. In all cases left for vaginal delivery, labour was monitored strictly with careful watch for maternal vitals, FHS, amount of bleeding and progress of labour and appropriate intervention was done as and when required.

After delivery

Patient and the newborn were kept under close observation for early detection of any complications. Follow-up of

the neonate with regards to well being and complications like sepsis, anaemia, convulsions, respiratory distress syndrome (RDS), jaundice, and death in neonate were recorded till the time of discharge. Date of discharge and duration of hospital stay (mother and baby) was noted along with date of delivery.

METHODOLOGY

A prospective study of outcome of pregnancy with first trimester bleeding per vaginum was carried out at Coimbatore Medical College Hospital, Trichy road, Coimbatore, Tamilnadu. It is a teaching hospital and also a tertiary care centre which constitutes largest referral centre. The patient population comprises mainly of low socio-economic group from urban slums, referred patients from surrounding rural health services, private clinics and adjacent district hospital.

Pregnant woman admitted in labour ward directly or through the antenatal ward of respective units were taken for the study. 150 cases were taken during the period of one year from August 2013 – July 2014. A structured proforma was used to collect information and followed till they get discharged.

INCLUSION CRITERIA

Singleton pregnancy upto thirteen weeks with positive fetal heart motion.

EXCLUSION CRITERIA

Ectopic and molar pregnancy

Information regarding age, socio economic status, gravida, details about previous conception, complaints of present pregnancy, medical disorders are received in addition to routine antenatal history and type of antenatal care received and total number of visits and timing of 1st booking visit.

Examination included both general and obstetrical. Height weight and blood pressure were recorded. Routine analysis of urine (albumin, sugar) HB, blood grouping and Rh typing and VDRL, HIV were all taken during admission. Period of gestation was derived from history of LMP and clinical examination and confirmed by USG. First trimester miscarriage, second trimester, miscarriage, preterm labour, full term labour, mode of delivery, weight of the baby, maturity and Apgar were recorded.

RESULT

A prospective study was undertaken on pregnant women with bleeding per vaginum in first trimester attending Coimbatore Medical College, Coimbatore, from AUG 2013- JULY 2014.

Total of 150 patients were selected and followed up for the study.

PATIENT CHARACTERISTICS

Patient were distributed according to Gravida, Age, Outcome, presence of SCH.

STATISTICAL ANALYSIS

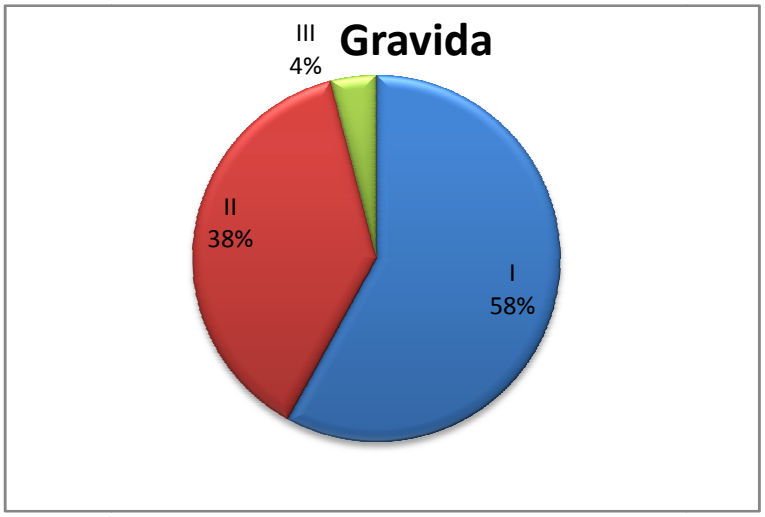
The collected data was analysed with SPSS 16.0 version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and for continuous variables the mean and S.D were used.

TABLE 1

DISTRIBUTION OF PATIENT ACCORDING TO GRAVIDA

GRAVIDA	FREQUENCY	PERCENT	VALID	CUMULATIVE
I	87	58.0	58.0	58.0
II	57	38.0	38.0	96.0
III	6	4.0	4.0	100.0
TOTAL	150	100.0	100.0	

Fig.12 DISRIBUTION OF PATIENTS ACCORDING TO GRAVIDA



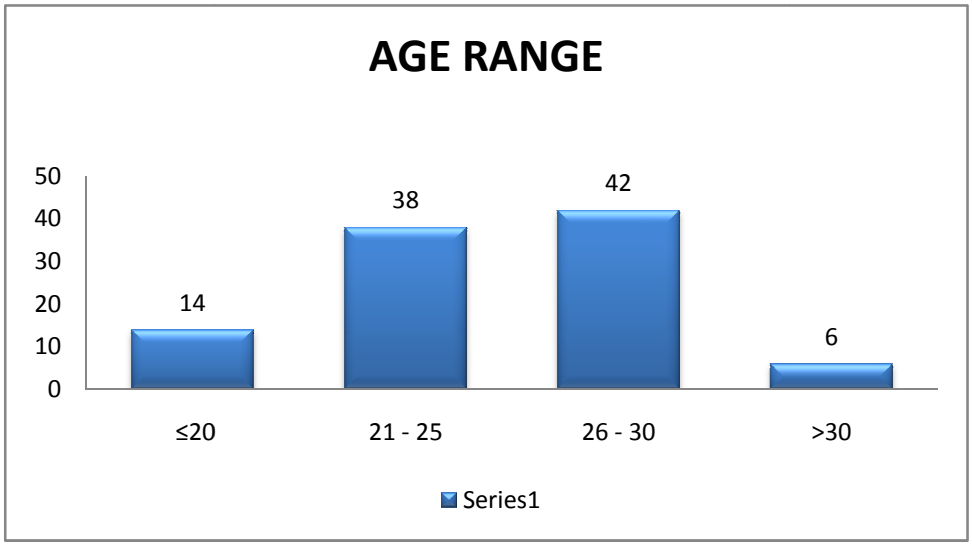
In my study there were 150 patients. Out of which there were 87 primi gravida which is 58 %, there were 57 second gravida which is 38 %, there were 6 third gravida which is 4 %. So first trimester bleeding is more common in primi gravida

TABLE 2

AGE DISTRIBUTION

AGE	FREQUENCY	PERCENT	VALID	CUMULATIVE
≤20	21	14.0	14.0	14.0
21 - 25	57	38.0	38.0	52.0
26 - 30	63	42.0	42.0	94.0
>30	9	6.0	6.0	100.0
TOTAL	150	100.0	100.0	

Fig.13 AGE DISTRIBUTION



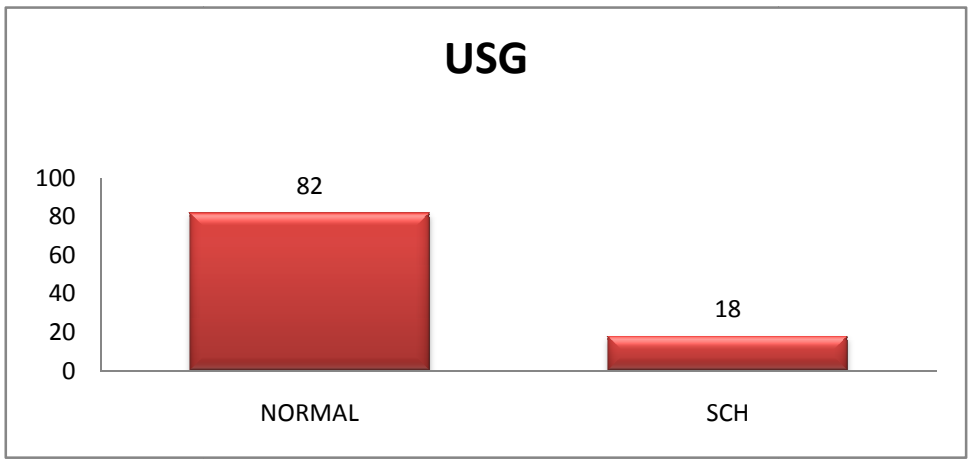
In my study ther were 14% in the age group of ≤20 yrs ,38% in the age group of 21 to 25 , 42 % in the age group of 26 to 30 and 6 % were > 30. So bleeding in the first trimester was more common in the age group of 26 to 30 years.

TABLE 3

DISTRIBUTION ACCORDING TO PRESENCE OF SCH

USG	FREQUENCY	PERCENT	VALID PERCENT	CUMULATIVE PERCENT
NORMAL	123	82.0	82.0	82.0
SCH	27	18.0	18.0	100.0
TOTAL	150	100.0	100.0	

Fig.14 DISTRIBUTION ACCORDING TO PRESENCE OF SCH



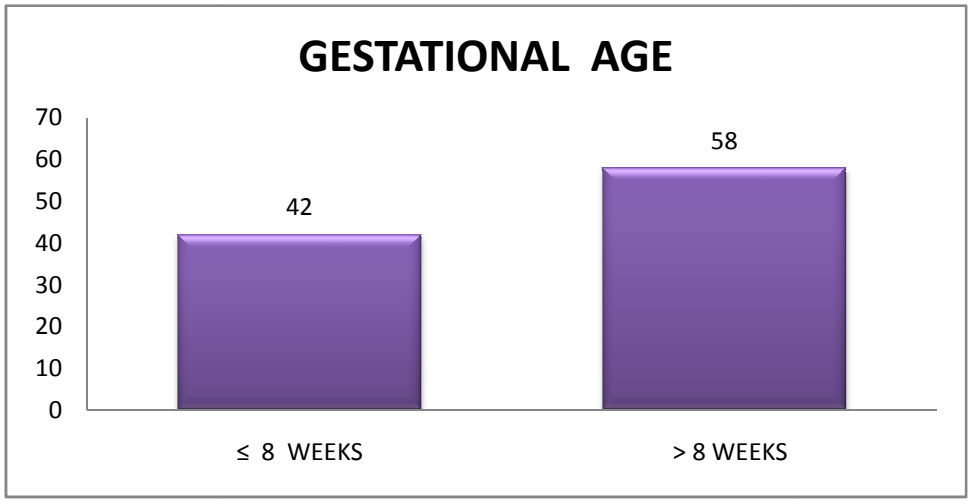
In my study there were 27 patients have USG evidence of SCH which was 18%.

TABLE 4

GESTATIONAL AGE AT THE TIME OF BLEEDING

GESTATIONAL	FREQUENC	PERCEN	VALID	CUMULATIV
≤ 8 WEEKS	63	42.0	42.0	42.0
> 8 WEEKS	87	58.0	58.0	100.0
TOTAL	150	100.0	100.0	

Fig.15 GESTATIONAL AGE AT THE TIME OF BLEEDING



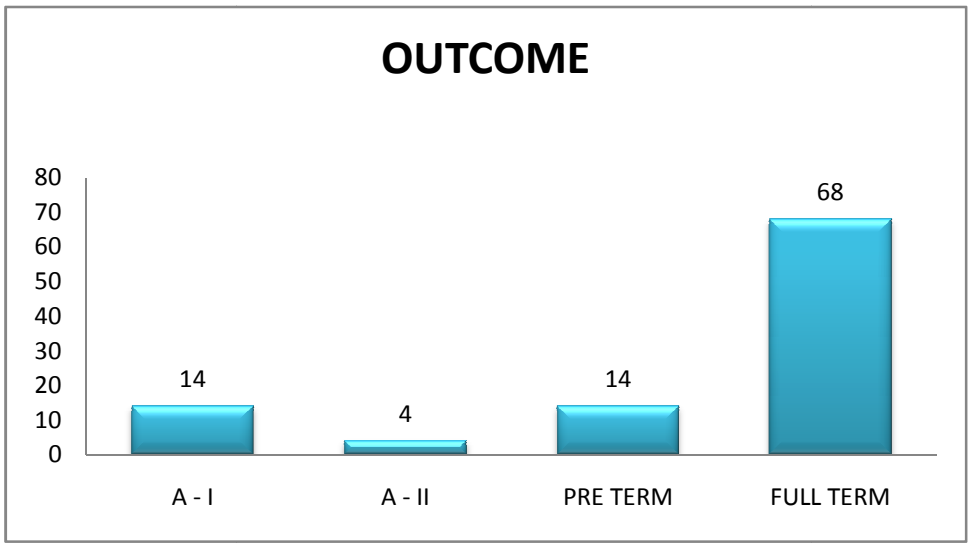
In my study there were 63 patients who had bleeding at ≤ 8 weeks which was 42 %, 87 patients who had> 8 weeks which was 58%. So bleeding is more common after 8weeks.

TABLE 5

OUTCOME OF PREGNANCY

	FREQUENCY	PERCENT	VALID	CUMULATIVE
A - I	21	14.0	14.0	14.0
A - II	6	4.0	4.0	18.0
PRE TERM	21	14.0	14.0	32.0
FULL	102	68.0	68.0	100.0
TOTAL	150	100.0	100.0	

Fig.16 OUTCOME OF PREGNANCY



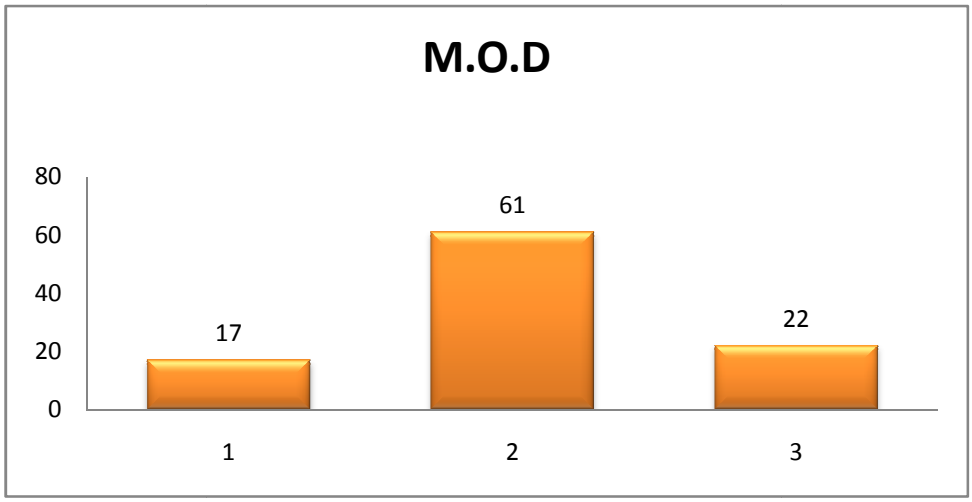
In my study there were 21 first trimester miscarriage which was 14% , 6 second trimester miscarriage which was 4%, 21 preterm birth, which was 14% 102 full term birth which was 68%

TABLE 6

DISTRIBUTION ACCORDING TO MODE OF DELIVERY

M.O.D	FREQUENCY	PERCENT	VALID	CUMULATIVE
PTND	21	17.0	17.0	17.0
FTND	75	61.0	61.0	78.0
LSCS	27	22.0	22.0	100.0
TOTAL	128	100.0	100.0	

Fig.17 DISTRIBUTION ACCORDING TO MODE OF DELIVERY



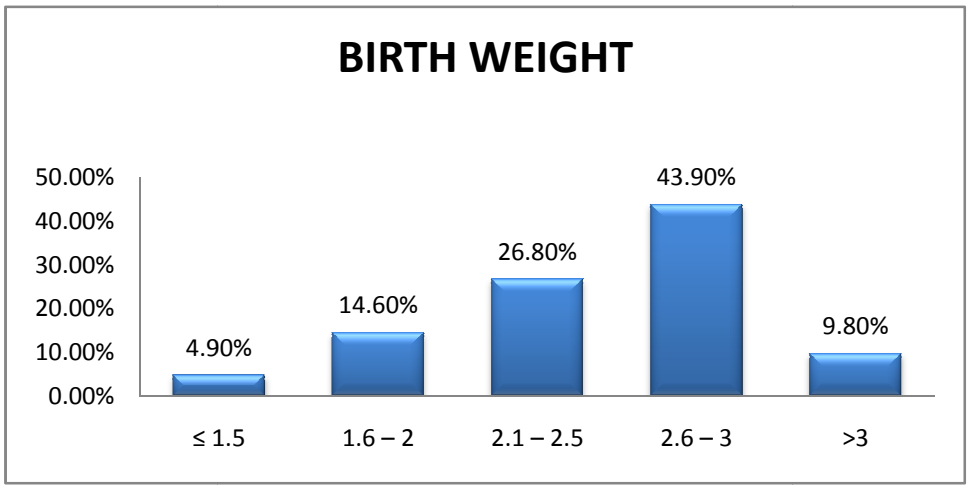
In my study out of 123 live birth there were 21 preterm vaginal delivery which was 14% ,75 full term vaginal delivery which was 50 % and 27 LS.CS which was 18%.

TABLE 7

BIRTH WEIGHT

S.No.	BIRTH WEIGHT	FREQUENCY	PERCNTAGE
1.	≤ 1.5	6	4.9%
2.	1.6 – 2	18	14.6%
3.	2.1 – 2.5	33	26.8%
4.	2.6 – 3	84	43.9%
5.	>3	12	9.8%
TOTAL		123	100.0%

Fig.18 BIRTH WEIGHT



In my study 44% had birth weight between 2.6 to 3 Kg.

TABLE 8

APGAR AT ONE MINUTE

APGAR	FREQUENCY	PERCENT	VALID	CUMULATIVE
< 5	9	7.3	7.3	7.3
5 - 8	87	70.7	70.7	78.0
>8	27	22.0	22.0	100.0
TOTAL	123	100.0	100.0	

Fig.19 APGAR AT ONE MINUTE

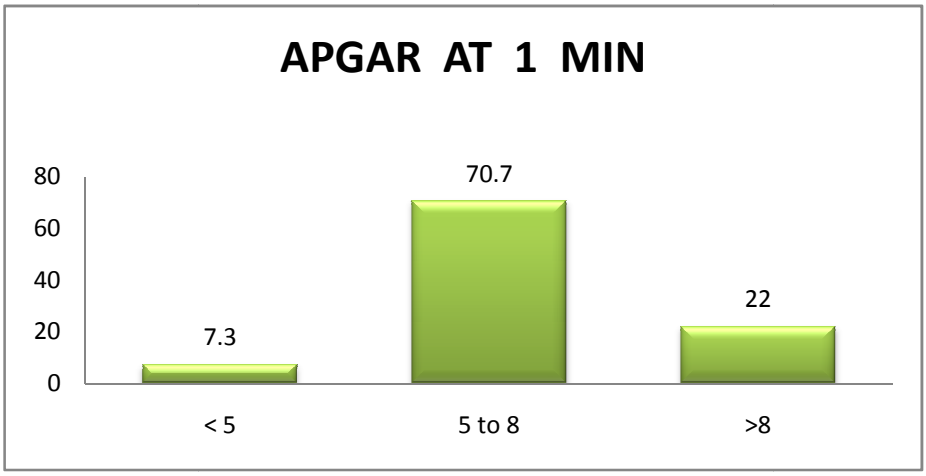
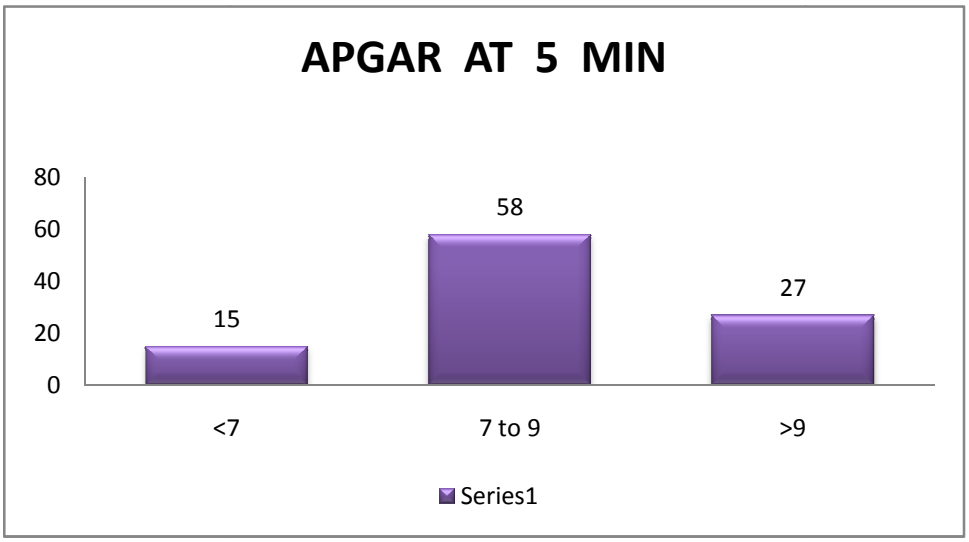


TABLE 9

APGAR AT FIVE MINUTE

APGAR	FREQUENCY	PERCENT	VALID	CUMULATIVE
<7	18	15.0	15.0	15.0
7 - 9	72	58.0	58.0	73.0
>9	33	27.0	27.0	100.0
TOTAL	123	100.0	100.0	

Fig.20 APGAR AT FIVE MINUTE



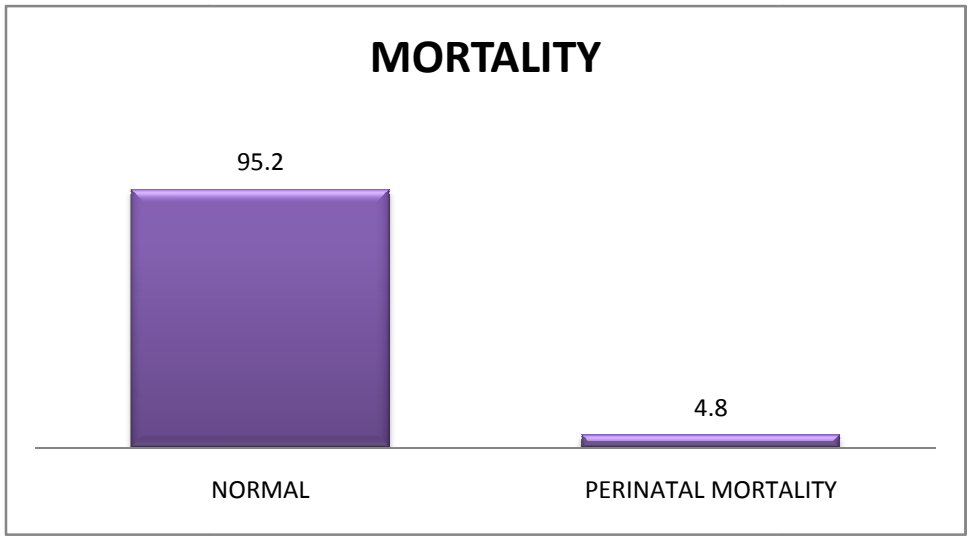
Apgar of the babies depend on various factors like MAS, Prematurity, neonatal jaundice, birth asphyxia.

TABLE 10

MORTALITY

	FREQUENCY	PERCENT	VALID PERCENT	CUMULATIVE PERCENT
NORMAL	117	95.2	95.2	95.2
PERINATAL MORTALITY	6	4.8	4.8	100.0
TOTAL	123	100.0	100.0	

Fig.21 MORTALITY



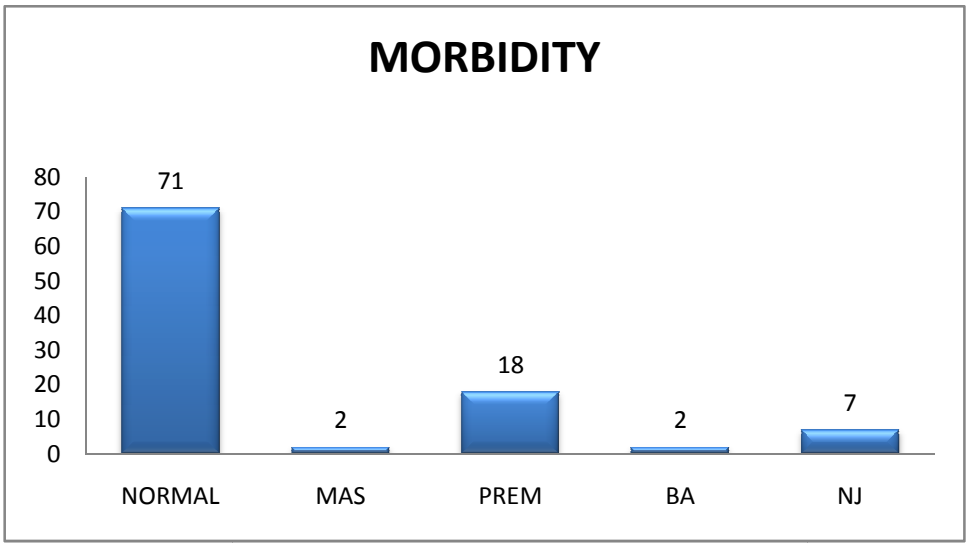
In my study there were 6 perinatal death due to birth asphyxia and prematurity which was 4.8%.

TABLE 11

MORBIDITY

	FREQUENCY	PERCENT	VALID PERCENT	CUMULATIVE PERCENT
NORMAL	87	71.0	71.0	71.0
MAS	3	2.0	2.0	73.0
PREM	21	18.0	18.0	91.0
BA	3	2.0	2.0	93.0
NJ	9	7.0	7.0	100.0
TOTAL	123	100.0	100.0	

Fig.22 MORBIDITY



The morbidity depend on various factors like MAS, PREM, BA NJ. In my study the morbidity due to MAS was 3 which was 2% PREM 21 which was 18 % BA 3 which was 2% NJ 9 which was 7%

DISCUSSION

Although first trimester vaginal bleeding is commonly considered a marker of a pregnancy at risk for adverse outcomes, few studies have vigorously investigated the prevalence and predictors of bleeding. Estimates of bleeding prevalence in early pregnancy are imprecise and range from 7-24%

First trimester bleeding per vaginum occures in 15-25%of all pregnancy. Medical disorder, Autoimmune disorder, Immunological disorder associated bleeding, eventhough the etiology is still unknown.

Mantoni and Pedersen first described the sonographic patterns of SCH which appears as an anechoic area that has a falciform shape and it is usually observed behind or below the gestational sac separating the chorion from the inner wall of uterus , small echogenic structures can be found in such areas and they are believed to be blood clots.

In our study there were 87 primigravida, 57 were second gravida, 6 were third gravida. So bleeding in the first trimester is more common in primigravida.

In our study 63 cases had bleeding in ≤8 weeks and 87 had bleeding more than 8 weeks. So bleeding is more common between 9-12 weeks.

Methodius conducted A cohort and case control study and concluded that SCH associated with increased risk of early and late pregnancy loss, abruption , preterm labour and PROM. Here in my study there were 27 patients who had SCH. Among this 6 ended with first trimester miscarriage, 3 second trimester miscarriage, 3 preterm labour, 15 fullterm labour . 22% ended with first trimester miscarriage 11% ended with second trimester miscarriage 11% ended in preterm 55% ended in full term. so the outcome not influenced by the prensence of SCH. It may be associated with the volume of SCH. The volume of SCH is not estimated.

Geneviene L Bennet studied effect of SCH and concluded that the spontaneous miscarriage rate was increased when SCH occured less than 8 weeks. In my study among the 27 cases with SCH, 18 cases had bleeding with SCH in ≤ 8 weeks. Among this 12 were ended in 1st trimester miscarriage which was 66%. So spontaneous miscarriage with SCH was more common if the bleeding occurs ≤ 8 weeks.

Gianpaolo maro performed study and concluded that the risk of spontaneous miscarriage is related to gestational age and is significantly increased if diagnosed <9 weeks and affect the outcome of pregnancy. In my study The incidence of miscarriage with SCH is more common in < 9 weeks and 1 patient ended in preterm Labour.

According to study the bleeding will be more common in advanced maternal age.In my study the presense of bleeding were

≤ 20yrs → 14 %

21 to 25 yrs → 38%

26 to 30 yrs → 42%

>30 yrs → 6%

The presence of bleeding is more common in the age group of 26 to 30 years.

According to Yang analysis 25% of women had reported bleeding with peak incidence during first completed month of pregnancy.In my study 18% had bleeding at the end of first completed month.

In Harvelle study the study group had bleeding at 8 weeks of gestation and study was limited by small number of participants , and dealt with preterm birth. Both these studies exclude the miscarriage.

In Alexan study, the study group had bleeding at 8 weeks and associates this with miscarriage and preterm birth. In my study 21 patients had bleeding at ≤ 8 weeks. Among these 5 ended in first trimester miscarriage and 1 second trimester miscarriage 15 full term. So the bleeding at 8 weeks with cardiac activity ended with good outcome.

In my study among the 150 patients there were 14 % ended with first miscarriage, 4 % second trimester miscarriage ,14 % preterm and 68 % full term birth . 82 % ended with live birth. So the outcome was not influenced by bleeding in first trimester.

Among the 123 live birth 21 PTND, 75 FTND, 27 LSCS. The indication for LSCS was obstetric cause only.

Among the 123 live birth 15 mothers had anaemia. But this was not related to bleeding.

Among the 123 live birth 44 % had birth weight between 2,5 to 3 Kg.

Apgar of the babies depend on various factors like prematurity, birth asphyxia, MAS.

In my study there were 6 perinatal deaths due to prematurity and birth asphyxia. So the perinatal outcome not influenced by bleeding in first trimester.

Morbidity depend on various factors like MAS, BA, PREM, NJ.

Thus this study showed that perinatal outcome is not influenced by bleeding in the first trimester.

SUMMARY

Threatened miscarriage occurs often and is a serious emotional burden fo women. Sonographic evaluation at presentation can usually differentiate between intra uterine and extra uterine pregnancy and offer some prognostic clues. Demonstration of fetal cardiac activity is generally associated with the successful pregnancy rate of 85 to 97% whereas an empty larger gestational sac or a discrepancy between menstrual and sonographic age of more than a week indicates poor prognosis. Serum beta HCG ,progesterone, inhibin A and CA -125 concentration may be helpful as predictors however these tests may not be useful in primary care settings.

The study was conducted on 150 patients . It is a prospective study, patients who had come to the hospital with first trimester bleeding were taken.

Cases were not dependent on the age, gravida. The study showed the results were not dependent on gravida and age of patient. It was seen that results were dependent on the gestational age of diagnosis.

Bleeding in the first trimester is more common between 9 to 12 weeks.

In 150 cases there were 27 miscarriage and 123 pregnancies who ended with live birth. Among the 123 live birthes there were 21 Preterm birth, term 102. Out of all 27 underwent cesarean section, rest of them delivered vaginally. There were total of 6 perinatal mortality because of birth asphyxia, prematurity, MAS.

Apgar of the babies dependent on various factors like prematurity, Birth asphyxia, MAS.

Thus the study showed that the perinatal outcome is not influenced by bleeding in first trimester.

CONCLUSION

From the study it can be concluded that the bleeding in the first trimester was more common in primi gravida . It was more common in the age group of 26 to 30 years. It was common between 9 to 12 weeks of gestation. There is no effect on perinatal outcome when the patient reached term. Though there were fifteen cases reported anaemia in the study it can not be said to be directly related to bleeding, because pregnancy itself is a state of hemodilution. Most of the patient present with anaemia, there by showing that the relation between maternal anaemia and bleeding is not significant unless the amount of bleeding is large. The outcome of the fetus was not influenced by bleeding in the first trimester.

Early diagnosis, Bed rest, Use of progesterone, Regular antenatal checkup will help in continuing pregnancy with good fetal outcome.

Limitations of the study

The study group include 150 patients only. If it will be included large number of patients it will be useful

There is no uniform definition of bleeding

The presense of bleeding in the first trimester is the only eligible criteria. Only women with bleeding in the firsr trimester were assessed. So if compare with appropriate comparative group it will be useful.

Hospital settings only captures the most serious episodes of bleeding. Thus to have complete ascestainment of all women experiencing bleeding during pregnancy a community or population based recruitment design is preferred.

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PROFORMA

Name :

Age :

Occupation :

Husband's Name :

Husband's Occupation :

Address :

Social economic status :

IP NO. :

Registered/unregistered :

Date of Admission :

Date of last menstrual period :

Expected date of delivery :

Period of gestation at time of admission :

Time interval between bleeding episode

and admission :

Duration of bleeding per vaginum :

Amount of blood loss :

Number of episodes of bleeding :

Bleeding associated with pain abdomen or not :

HISTORY

1st trimester : History of nausea/vomiting/ bleeding per vaginum/urinary

bowel trouble/Antenatal check up/ drug intake/fever.

2nd trimester : History of quickening, injection TT doses; Any complications, history of hypertension/Pedal edema/ Headache/ Giddiness/ Blurring vision. Intake of iron and calcium tablets were taken up.

MENSTRUAL HISTORY

Age or Menarche

Duration of flow

Amount of flow

Regular/Irregular

Date of last menstrual period

MARITAL HISTORY

Consanguineous or non Consanguineous

OBSTETRIC HISTORY

Gravida

Para

Abortion

Term/Preterm

Home/Hospital Deliveries

Normal delivery. Caesarean/Breech/Forceps

PAST HISTORY

Any history of tuberculosis, Diabetes mellitus, hypertension, drug allergy, jaundice, blood transfusion, bleeding diathesis.

History of bleeding in previous pregnancies.

PERSONAL HISTORY

Vegetarian or Non-vegetarian

GENERAL PHYSICAL EXAMINATION

General Condition

Pulse

Blood pressure

Temperature

Respiratory rate

Pallor/Icterus/Cyanosis/ Edema feet

Any other relevant findings

SYSTEMIC EXAMINATION :

Cardiovascular

Respiratory

LOCAL EXAMINATION:

Per abdomen examination

Fundal height

Tone of uterus-Tense/Tender/relaxed

Lie & presentation of fetus

FHS Present/Absent

Regular/Irregular/Slow

Per speculum examination

Per vaginal examination

INVESTIGATIONS

Hb

Urine complete examination

Blood grouping and Rh :

HIV, VDRL, HBsAg

BT, CT,

GCT

USG – for estimation of gestational age, placental localization, amount of liquor, evidence of SCH, any gross congenital malformation.

Labour- spontaneous/induced

Mode of delivery

Normal vaginal delivery

Induction delivery interval

Instrumental delivery

Caesarean section

FETAL OUTCOME:

Sex

Weight

Still born/Alive

APGAR score

Asphyxia/Respiratory distress

Condition of baby on discharge

Whether neonatal death or discharged well

Condition of mother on discharge

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் மகப்பேறு மருத்துவ துறையில் பட்ட பயிலும் மாணவி அவர்கள் மேற்கொள்ளும் "**முதல் மூன்று மாதத்தில் ஏற்படும் இரத்தப் போக்கால் பிரசவத்தில் ஏற்படும் பின்விளைவுகள்**" குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்தில் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

MASTER CHART

S. No.	NAME	AGE	IP.No.	GRAVIDA	G.A	USG	G.A AT DELIVERY	Hb	BT	CT	OUT COME	M.O.D	INDIC ATION	APGAR 1	APGAR 5	SEX	WEI GHT	MOR TALITY	MORBI DITY
1	DEVI	18	70512	I	8	SCH	8	9	2	4.5	A	-	-	-	-	M	-	-	-
2	RANI	19	69269	I	7	-	38	10	2.5	4	FT	FTND	-	7	8	F	2.3	-	-
3	AMUTHA	18	70123	I	9	-	39	12	3	4	FT	FTND	-	8	9	F	2.6	-	
4	VANI	21	70187	I	6	-	38	13	3	5	FT	LSCS	FD	4	5	F	2.8	-	MAS
5	KALVANI	22	69789	I	9	-	37	9	2	6	FT	FTND	-	7	9	M	2.6	-	-
6	KANNAHA	21	71102	I	10	SCH	34	8	2	6	PT	PTND	-	6	7	M	1.5	-	PREM
7	JANSI	23	71202	I	11	-	38	12	2.5	7	FT	FTND	-	8	9	F	2.2	-	-
8	RAJEE	23	71615	I	12	-	37	13	3	5	FT	FTND	-	8	10	M	2.9	-	-
9	MALIKA	20	71025	I	7	SCH	7	11	2	6	A	-	-	-	-	-	-	-	-
10	PADMA	20	70891	I	7	-	38	13	2	4.5	FT	FTND	-	7	8	M	2.6	-	-
11	VINITHA	36	70192	II	8	SCH	8	12	2	5.5	A	-	-	-	-	-	-	-	-
12	PREETHA	37	71202	III	9	-	33	9	3	5	PT	PTND	-	4	6	F	1.6	PREM	PREM
13	VIGI	30	72055	II	7	-	8	9	2.5	5	A	-	-	-	-	-	-	-	-
14	RUPA	31	72089	I	10	-	34	8	3	6.5	PT	PTND	-	7	7	M	2	-	PREM
15	CHANDRA	25	72123	I	9	-	38	12	2	6	FT	FTND	-	4	6	M	2.5	BA	BA
16	FARTHIMA	26	72185	II	8	-	39	11	2	5	FT	FTND	-	9	10	F	2.8	-	-
17	SAMEENA	28	72555	II	8	-	37	13	2.5	5	FT	FTND	-	8	10	M	2.9	-	-
18	RUBINI	29	72565	III	11	-	33	9	3	4	PT	PTND	-	9	10	F	1.5	-	PREM
19	SEETHA	25	72111	I	8	-	38	12	3	4	FT	LSCS	CPD	7	8	M	3.2	-	-
20	RATHA	26	72105	II	8	SCH	22	13	2.5	5	A	-	-	-	-	-	-	-	-
21	THULASI	20	1212	I	8	-	37	8.5	2	4.5	FT	FTND	-	9	10	F	2.6		-
22	RANJANI	21	4587	I	9	-	38	11	2	5	FT	FTND	-	8	9	F	2.7	-	-
23	JANANI	24	5208	I	7	-	34	9	2.5	6.5	PT	PTND	-	6	8	F	2.8	-	PREM
24	REGINA	24	9874	I	12	-	20	10	3	6	A	-	-	-	-	-	-	-	-
25	RAGHAVI	22	9956	I	10	-	38	10	3	6	FT	LSCS	FD	8	9	F	2.1		NJ
26	ANU	21	1023	I	7	SCH	37	12	2.5	7	FT	FTND	-	9	10	M	2.7	-	-

27	PRIYA	20	1200	I	10	-	38	8	2.5	7	FT	FTND	-	8	9	F	2.6	-	-
28	BAKKYA	23	1315	I	11	-	39	13	2	5.5	FT	LSCS	FD	9	10	F	2.8	-	NJ
29	DEVIKA	21	1909	I	6	-	6	11	2	5	A	-	-	-	-	-	-	-	-
30	LALITHA	20	2000	I	12	-	38	7	3	4	FT	FTND	-	9	10	F	2.6	-	-
31	MAMTHA	28	3210	II	9	-	37	10	3	4	FT	FTND	-	7	8	F	2.5	-	-
32	NIVETHA	29	2505	II	8	-	38	9	2.5	5	FT	LSCS	FD	9	10	M	2.6	-	-
33	BABITHA	26	2771	I	12	-	12	12	2	5.5	A	-	-	-	-	-	-	-	-
34	ABIRAMI	25	3303	II	10	SCH	37	13	2	6	FT	FTND	-	8	9	F	2.1	-	-
35	MEENA	26	3337	I	9	-	38	10	2	6	FT	FTND	-	8	9	M	2.4	-	-
36	SHANTHI	27	3908	II	9	-	37	9	3	4	FT	FTND	-	7	9	M	2.7	-	-
37	GRACE	28	3795	II	11	-	11	13	3	5	A	-	-	-	-	-	-	-	-
38	SARANYA	29	11205	II	12	-	39	12	3	6.5	FT	LSCS	CPD	7	9	F	3.5	-	-
39	RAMANI	25	12000	I	9	-	38	12	2.5	4.5	FT	FTND	-	8	9	F	2.5	-	-
40	RAMA	28	12079	II	8	-	37	11	2.5	5	FT	LSCS	FD	5	6	F	2.6	-	-
41	SOFIA	25	13135	II	8	SCH	38	10	3	5	FT	FTND	-	7	8	F	2.3	-	-
42	GEETHA	26	52202	I	9	-	33	10	2	4.5	PT	PTND	-	5	6	M	2	-	PREM
43	SABINA	27	39745	II	8	-	37	12	2.5	5.5	FT	FTND	-	8	9	M	1.9	-	-
44	SANGEETHA	28	45457	II	6	-	38	13	2	4	FT	FTND	-	9	10	M	2.4	-	-
45	PUJA	29	54987	II	11	-	37	9	2	5	FT	FTND		9	10	F	2.7	-	-
46	DEEPA	29	21325	II	6	-	38	9	3	5.5	FT	FTND	-	8	9	F	2	-	-
47	LATHA	28	24568	II	12	SCH	39	12	2	6	FT	FTND	-	5	6	F	3.2	-	NJ
48	SREEMATHI	26	60605	I	11	-	38	13	3	5	FT	LSCS	CPD	7	8	M	3.3	-	-
49	SAROJA	27	70524	II	10	-	34	10	2	5	PT	PTND	-	7	9	F	2.3	-	PREM
50	SELVI	25	40405	I	9	-	38	12	2.5	4.5	FT	LSCS	FD	7	8	F	2.8	-	-
51	ARASI	18	41300	I	8	SCH	8	9	2	4.5	A	-	-	-	-	M	-	-	-
52	BANU	19	31290	I	7	-	38	10	2.5	4	FT	FTND	-	7	8	F	2.3	-	-
53	PARANI	18	1960	I	9	-	39	12	3	4	FT	FTND	-	8	9	F	2.6	-	
54	ANJALI	21	3500	I	6	-	38	13	3	5	FT	LSCS	FD	4	5	F	2.8	-	MAS

55	SAMSET	22	9827	I	9	-	37	9	2	6	FT	FTND	-	7	9	M	2.6	-	-
56	SATHYA	21	10205	I	10	SCH	34	8	2	6	PT	PTND	-	6	7	M	1.5	-	PREM
57	RUBINI	23	11367	I	11	-	38	12	2.5	7	FT	FTND	-	8	9	F	2.2	-	-
58	DARANI	23	12487	I	12	-	37	13	3	5	FT	FTND	-	8	10	M	2.9	-	-
59	VASUMATHI	20	13987	I	7	SCH	7	11	2	6	A	-	-	-	-	-	-	-	-
60	ANU	20	18340	I	7	-	38	13	2	4.5	FT	FTND	-	7	8	M	2.6	-	-
61	NARMATHA	36	19367	II	8	SCH	8	12	2	5.5	A	-	-	-	-	-	-	-	-
62	KIRTHIKA	37	26487	III	9	-	33	9	3	5	PT	PTND	-	4	6	F	1.6	PREM	PREM
63	THAVASI	30	24600	II	7	-	8	9	2.5	5	A	-	-	-	-	-	-	-	-
64	JANAKI	31	24978	I	10	-	34	8	3	6.5	PT	PTND	-	7	7	M	2	-	PREM
65	JAMUNA	25	25478	I	9	-	38	12	2	6	FT	FTND	-	4	6	M	2.5	BA	BA
66	LEELA	26	29650	II	8	-	39	11	2	5	FT	FTND	-	9	10	F	2.8	-	-
67	LAILA	28	27654	II	8	-	37	13	2.5	5	FT	FTND	-	8	10	M	2.9	-	-
68	RAMZAN	29	32654	III	11	-	33	9	3	4	PT	PTND	-	9	10	F	1.5	-	PREM
69	SARATHY	25	31876	I	7	-	38	12	3	4	FT	LSCS	CPD	7	8	M	3.2	-	-
70	AKILA	26	35784	II	8	SCH	22	13	2.5	5	A	-	-	-	-	-	-	-	-
71	GRACE	20	54378	I	8	-	37	7.5	2	4.5	FT	FTND	-	9	10	F	2.6		-
72	SUDAR	21	60564	I	9	-	38	11	2	5	FT	FTND	-	8	9	F	2.7	-	-
73	QUINE	24	62541	I	7	-	34	9	2.5	6.5	PT	PTND	-	6	8	F	2.8	-	PREM
74	AGNAS	24	51890	I	12	-	20	10	3	6	A	-	-	-	-	-	-	-	-
75	MANJU	22	37864	I	10	-	38	10	3	6	FT	LSCS	FD	8	9	F	2.1		NJ
76	MALAR	21	45682	I	7	SCH	37	12	2.5	7	FT	FTND	-	9	10	M	2.7	-	-
77	SURYA	20	52684	I	10	-	38	8.5	2.5	7	FT	FTND	-	8	9	F	2.6	-	-
78	BAMA	23	21987	I	11	-	39	13	2	5.5	FT	LSCS	FD	9	10	F	2.8	-	NJ
79	MANGAI	21	7892	I	6	-	6	11	2	5	A	-	-	-	-	-	-	-	-
80	PARAMU	20	8865	I	12	-	38	8.5	3	4	FT	FTND	-	9	10	F	2.6	-	-
81	RUPA	28	11259	II	9	-	37	10	3	4	FT	FTND	-	7	8	F	2.5	-	-
82	MAARI	29	15364	II	8	-	38	9	2.5	5	FT	LSCS	FD	9	10	M	2.6	-	-

83	AMMANI	26	17890	I	12	-	12	12	2	5.5	A	-	-	-	-	-	-	-	-
84	MANASI	25	17862	II	10	SCH	37	13	2	6	FT	FTND	-	8	9	F	2.1	-	-
85	MERCY	26	19854	I	9	-	38	10	2	6	FT	FTND	-	8	9	M	2.4	-	-
86	NANCY	27	25495	II	9	-	37	9	3	4	FT	FTND	-	7	9	M	2.7	-	-
87	FERSIA	28	35492	II	11	-	11	13	3	5	A	-	-	-	-	-	-	-	-
88	VASAVI	29	48672	II	12	-	39	12	3	6.5	FT	LSCS	CPD	7	9	F	3.5	-	-
89	SAMEEMA	25	61524	I	9	-	38	12	2.5	4.5	FT	FTND	-	8	9	F	2.5	-	-
90	FELISIA	28	58942	II	8	-	37	11	2.5	5	FT	LSCS	FD	5	6	F	2.6	-	-
91	VANAJA	25	48321	II	8	SCH	38	10	3	5	FT	FTND	-	7	8	F	2.3	-	-
92	PETRISIA	26	67542	I	9	-	33	10	2	4.5	PT	PTND	-	5	6	M	2	-	PREM
93	VANI	27	51871	II	7	-	37	12	2.5	5.5	FT	FTND	-	8	9	M	1.9	-	-
94	SUSI	28	32465	II	6	-	38	13	2	4	FT	FTND	-	9	10	M	2.4	-	-
95	DILSATH	29	56482	II	11	-	37	9	2	5	FT	FTND		9	10	F	2.7	-	-
96	THAGEERA	29	28654	II	6	-	38	9	3	5.5	FT	FTND	-	8	9	F	2	-	-
97	MAMTHA	28	23486	II	12	SCH	39	12	2	6	FT	FTND	-	5	6	F	3.2	-	NJ
98	USHA	26	31498	I	11	-	38	13	3	5	FT	LSCS	CPD	7	8	M	3.3	-	-
99	DIVYA	27	41657	II	10	-	34	10	2	5	PT	PTND	-	7	9	F	2.3	-	PREM
100	POOJA	25	61497	I	9	-	38	12	2.5	4.5	FT	LSCS	FD	7	8	F	2.8	-	-
101	SRIDEVI	18	52348	I	8	SCH	8	9	2	4.5	A	-	-	-	-	M	-	-	-
102	JEERINA	19	10846	I	7	-	38	10	2.5	4	FT	FTND	-	7	8	F	2.3	-	-
103	JANU	18	64259	I	9	-	39	12	3	4	FT	FTND	-	8	9	F	2.6	-	
104	TRISHA	21	11650	I	6	-	38	13	3	5	FT	LSCS	FD	4	5	F	2.8	-	MAS
105	NARAYANI	22	31890	I	9	-	37	9	2	6	FT	FTND	-	7	9	M	2.6	-	-
106	JOTHI	21	51742	I	10	SCH	34	8	2	6	PT	PTND	-	6	7	M	1.5	-	PREM
107	PUNUTHA	23	29500	I	11	-	38	12	2.5	7	FT	FTND	-	8	9	F	2.2	-	-
108	CHARULATA	23	35684	I	12	-	37	13	3	5	FT	FTND	-	8	10	M	2.9	-	-
109	PREMA	20	43291	I	7	SCH	7	11	2	6	A	-	-	-	-	-	-	-	-
110	AMUTHA	20	59684	I	7	-	38	13	2	4.5	FT	FTND	-	7	8	M	2.6	-	-

111	FARTHIMA	36	55789	II	8	SCH	8	12	2	5.5	A	-	-	-	-	-	-	-	-
112	AMEERTHA	37	13567	III	9	-	33	9	3	5	PT	PTND	-	4	6	F	1.6	PREM	PREM
113	ROSHNI	30	14957	II	7	-	8	9	2.5	5	A	-	-	-	-	-	-	-	-
114	SUBA	31	27650	I	10	-	34	8	3	6.5	PT	PTND	-	7	7	M	2	-	PREM
115	SINDU	25	18354	I	9	-	38	12	2	6	FT	FTND	-	4	6	M	2.5	BA	BA
116	NEELA	26	24590	II	8	-	39	11	2	5	FT	FTND	-	9	10	F	2.8	-	-
117	SIJI	28	45681	II	8	-	37	13	2.5	5	FT	FTND	-	8	10	M	2.9	-	-
118	SUJATHA	29	38940	III	11	-	33	9	3	4	PT	PTND	-	9	10	F	1.5	-	PREM
119	MEENAKSHI	25	58742	I	8	-	38	12	3	4	FT	LSCS	CPD	7	8	M	3.2	-	-
120	GOPIKA	26	61852	II	8	SCH	22	13	2.5	5	A	-	-	-	-	-	-	-	-
121	MEERA	20	65790	I	8	-	37	7	2	4.5	FT	FTND	-	9	10	F	2.6		-
122	SAREETHA	21	21654	I	9	-	38	11	2	5	FT	FTND	-	8	9	F	2.7	-	-
123	GOKILA	24	35248	I	7	-	34	9	2.5	6.5	PT	PTND	-	6	8	F	2.8	-	PREM
124	RAJULA	24	42698	I	12	-	20	10	3	6	A	-	-	-	-	-	-	-	-
125	SAFIA	22	56231	I	10	-	38	10	3	6	FT	LSCS	FD	8	9	F	2.1		NJ
126	POONGUDI	21	55874	I	7	SCH	37	12	2.5	7	FT	FTND	-	9	10	M	2.7	-	-
127	ANISHA	20	49651	I	10	-	38	8	2.5	7	FT	FTND	-	8	9	F	2.6	-	-
128	KANNAMA	23	48251	I	11	-	39	13	2	5.5	FT	LSCS	FD	9	10	F	2.8	-	NJ
129	NEERAJA	21	36521	I	6	-	6	11	2	5	A	-	-	-	-	-	-	-	-
130	SHARMILA	20	39572	I	12	-	38	7	3	4	FT	FTND	-	9	10	F	2.6	-	-
131	LESLY	28	18965	II	9	-	37	10	3	4	FT	FTND	-	7	8	F	2.5	-	-
132	KAMATHCHI	29	34561	II	8	-	38	9	2.5	5	FT	LSCS	FD	9	10	M	2.6	-	-
133	ARUNA	26	55631	I	12	-	12	12	2	5.5	A	-	-	-	-	-	-	-	-
134	SHALLY	25	45592	II	10	SCH	37	13	2	6	FT	FTND	-	8	9	F	2.1	-	-
135	FARINA	26	32781	I	9	-	38	10	2	6	FT	FTND	-	8	9	M	2.4	-	-
136	SOOLAI	27	19587	II	9	-	37	9	3	4	FT	FTND	-	7	9	M	2.7	-	-
137	ROOKMANI	28	12654	II	11	-	11	13	3	5	A	-	-	-	-	-	-	-	-
138	PAKYA	29	42563	II	12	-	39	12	3	6.5	FT	LSCS	CPD	7	9	F	3.5	-	-

139	YOGA	25	36581	I	9	-	38	12	2.5	4.5	FT	FTND	-	8	9	F	2.5	-	-
140	SEETHA	28	62548	II	8	-	37	11	2.5	5	FT	LSCS	FD	5	6	F	2.6	-	-
141	RAMANI	25	53698	II	8	SCH	38	10	3	5	FT	FTND	-	7	8	F	2.3	-	-
142	PRIYANKA	26	17235	I	9	-	33	10	2	4.5	PT	PTND	-	5	6	M	2	-	PREM
143	MARIA	27	16548	II	8	-	37	12	2.5	5.5	FT	FTND	-	8	9	M	1.9	-	-
144	VALARMATI	28	32415	II	6	-	38	13	2	4	FT	FTND	-	9	10	M	2.4	-	-
145	SUSILA	29	62594	II	11	-	37	8.5	2	5	FT	FTND		9	10	F	2.7	-	-
146	SUMITHRA	29	63254	II	6	-	38	9	3	5.5	FT	FTND	-	8	9	F	2	-	-
147	NAZEMA	28	42563	II	12	SCH	39	12	2	6	FT	FTND	-	5	6	F	3.2	-	NJ
148	ANANDHI	26	24561	I	11	-	38	13	3	5	FT	LSCS	CPD	7	8	M	3.3	-	-
149	ROHINI	27	31562	II	10	-	34	10	2	5	PT	PTND	-	7	9	F	2.3	-	PREM
150	LAVANYA	25	45286	I	9	-	38	12	2.5	4.5	FT	LSCS	FD	7	8	F	2.8	-	-