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COMPARATIVE STUDY OF ETIOLOGICAL FACTORS OF *FIRST ONE EARLY PREGNANCY LOSS* WITH THAT OF RECURRENT PREGNANCY LOSS

IJSER

A DISSERTATION SUBMITTED TO

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(An Institute of National Importance by an Act of Parliament,
under Ministry of Health and Family Welfare, Government of India)

DHANVANTRI NAGAR, PUDUCHERRY, INDIA-605006.

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(An Institute of National Importance by an Act of Parliament under Ministry of Health and Family Welfare, Government of India)

CERTIFICATE

This is to certify that this dissertation entitled **"Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss"** is a bonafide record of work done by **Dr. SONAL GARG** under our guidance and supervision in the Department of Obstetrics & Gynaecology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, during the period of her postgraduate study for the degree of **M. S. OBSTETRICS AND GYNAECOLOGY (Branch - II)** from **July 2017** to **June 2020**.

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ABBREVIATIONS

ACCP	-	American College of Chest Physicians
ACLA	-	Anticardiolipin antibodies
ACOG	-	American College of Obstetricians and Gynecologists
ANA	-	Antinuclear antibodies
APS	-	Antiphospholipid syndrome
APLA	-	Antiphospholipid antibodies
APTT	-	Activated partial thromboplastin time
ASRM	-	American Society of Reproductive Medicine
AT III	-	Antithrombin III
CMV	-	Cytomegalovirus
CSF	-	Colony stimulating factor
DM	-	Diabetes Mellitus
ESHRE	-	European Society of Human Reproduction & embryology
FSH	-	Follicle stimulating hormone
FVL	-	Factor V Leiden
GDM	-	Gestational Diabetes Mellitus
GTT	-	Glucose tolerance test
HbA1c	-	Glycosylated haemoglobin
HELLP	-	Haemolysis, elevated liver enzymes, low platelet count
HIV	-	Human immunodeficiency virus
HCG	-	Human chorionic gonadotropin
HLA	-	Human leucocyte antigen
HSG	-	Hysterosalphingography
IGFBP-1	-	Insulin like growth factor binding protein-1
IL	-	Interleukin
IUGR	-	Intrauterine growth retardation
IVIG	-	Intravenous immunoglobulin

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JIPMER	-	Jawaharlal Institute of Postgraduate Medical Education & Research
LA	-	Lupus anticoagulant
LIF	-	Leukemia inhibitory factor
LMP	-	Last menstrual period
LMWH	-	Low molecular weight heparin
LPS	-	Lipopolysaccharide
MTHFR	-	Methylene tetrahydrofolate reductase
MUC-1	-	Mucin-1 (glycoprotein)
NICE	-	National Institute for Health & Care Excellence
NICU	-	Neonatal intensive care unit
NK cell	-	Natural killer cell
OPD	-	Outpatient department
PAI-1	-	Plasminogen activator inhibitor-1
PC	-	Protein C
PCOS	-	Polycystic ovary syndrome
PGF2α	-	Prostaglandin F2α
PS	-	Protein S
RCOG	-	Royal College of Obstetricians and Gynaecologists
RCT	-	Randomized controlled trial
RPL	-	Recurrent pregnancy loss
SLE	-	Systemic Lupus Erythematosus
SHG	-	Sonohysterography
TORCH	-	Toxoplasmosis, Rubella, Cytomegalovirus, Herpes virus
TFT	-	Thyroid function test
TPO antibodies	-	Thyroid peroxidase antibodies
TSH	-	Thyroid stimulating hormone
USG	-	Ultrasonography
VTE	-	Venous thromboembolism

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INTRODUCTION

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Pregnancy loss is a distressing condition for both the patient and Obstetrician. It can occur at any gestational period but most commonly during early pregnancy. The etiology for early pregnancy loss and late pregnancy loss are most often different. Early pregnancy loss is defined as a non-viable intrauterine pregnancy with either an empty gestational sac or a gestational sac containing an embryo or fetus without cardiac activity within the first 12+6/7 weeks of gestation. In the first trimester the terms miscarriage spontaneous abortion and early pregnancy loss are used interchangeably as there is no consensus on terminology in the literature[1].

Early pregnancy loss occurs in 10% of all clinically recognized pregnancies and approximately 80% of all cases of pregnancy losses occur within the first trimester[2]. Pregnancy loss when occurs repeatedly is termed recurrent pregnancy loss (RPL). According to American society of reproductive medicine (ASRM), recurrent pregnancy loss is a distinct disorder defined by two or more failed clinical pregnancies[3]. Guidelines recommend evaluation only for RPL as a wide variety of etiological factors have been described in the literature and evaluation of RPL revealed causes only in 50%[4]. But there are no recommendations for initiation of investigations after first or single pregnancy loss.

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Whenever a woman suffers pregnancy loss an explanation is sought for the same from the treating Obstetrician. ***Sometimes women approach the clinicians after having suffered pregnancy loss and request for investigations, but the clinical practice recommendations are to investigate after two or more pregnancy losses.***

A significant proportion of women (20%) who experience a miscarriage become symptomatic for depression and anxiety.[5] This warrants diagnostic work-up and interventions. There are no studies with regard to initiation of investigations after first early pregnancy loss. ***In this context,this study aims to find out the etiological factors in women with first earlypregnancy loss in comparison to women with two or more than two early pregnancy losses (RPL).*** This study will establish the need, if any, to investigate a woman after one pregnancy loss for possible etiological factors. This will also find out the common causes of early pregnancy loss in this population and ensure adequate timely intervention for treatable causes without waiting for the subsequent pregnancy loss.

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AIMS AND OBJECTIVES

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AIM-

To find out the etiological factors in women with first early pregnancy loss.

Primary objective-

To determine the identifiable causes and their proportion in women with first early pregnancy loss.

Secondary Objective-

To compare the causes of first early pregnancy loss with that of women with two or more than two early pregnancy losses (RPL).

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REVIEW OF LITERATURE

Development of normal pregnancy

A. Implantation

Implantation is a crucial step after fertilization for the development of normal pregnancy. All the metabolic functions of the fetus are carried out by placenta. Thus the anatomical relationship of uterine interface, placenta and fetus mediated by the process of implantation is necessary for the development and maintainence of normal pregnancy.

Implantation begins 6-7 days after fertilization. This entire process of implantation is divided into three phases, apposition, adhesion and invasion. An appropriately primed receptive endometrium is required for successful implantation, which is optimum till day 20-24 of the menstrual cycle. Implantation involves interaction of cell-surface receptors at the implantation site with blastocyst receptors. After day 24 of cycle, anti adhesive glycoprotein is synthesized which interferes with the receptor interactions. A specific type of integrins called endometrial integrins which are hormonally regulated play a role in cell adhesion to extracellular matrix proteins.[6]

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B. Embryo

Zygote is unicellular and it undergoes mitosis to form multicellular embryo. Zygote undergoes cleavage and forms blastocyst. Blastocyst is further differentiated into the inner cell mass and trophoectoderm. It produces IL-1α, IL- 1β, HCG which influences endometrial receptivity. Receptive endometrium in turn activate the signaling pathways by producing leukemia inhibitory factor (LIF) and follistatin further enhancing the receptivity. It also produces colony

stimulating factor-1 (CSF-1) which plays a role in implantation by immunomodulatory and proangiogenic actions.[6]

C. Fetus

Embryo is termed as fetus approximately 9 weeks after LMP and 7 weeks after fertilization. At this time, fetus measures approximately 24 mm in length and most of the organ systems have developed and fetus undergoes growth and maturation till term.[6] Abnormal fetal development will lead to early pregnancy loss.

D. Factors affecting normal fetal development

Corpus luteum

The main function of the corpus luteum is secretion of progesterone which is essential to maintain normal pregnancy. After ovulation, the residual follicular granulosal and thecal cells form corpus luteum. It is composed of two steroidogenic cell types, small luteal cells and large luteal cells. Small luteal cells are thecal in origin and they respond to LH. LH activates the protein kinase A and stimulate the secretion of progesterone from these cells. Large luteal cells are granulosa cell origin and mediate the luteolytic actions of PGF2α in case pregnancy doesn't occur. The pregnant uterus secretes HCG, antiluteolytic factor and activates a neuroendocrine reflex arc to signal the corpus luteum about the conceptus. HCG maintains the corpus luteum and it secretes progesterone to maintain pregnancy and decays by 12th week period of gestation to become corpus albicans.[7] During this period, corpus-luteal placental shift will occur. Any deficiency of progesterone or presence of luteolytic factors will lead to pregnancy loss.

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Placenta

Human placenta formation starts with the trophoectoderm leading to the formation of trophoblast cell layer. Trophoblasts promote implantation, play a nutritive role, help in physiological adaptations and maintenance of pregnancy. Trophoblasts get differentiated into an outer syncytiotrophoblast and inner cytotrophoblast after initial implantation. Cytotrophoblast can undergo DNA synthesis, while this function is lacking in syncytiotrophoblast which performs transport functions. Trophoblasts further differentiate into villous and extravillous trophoblasts. Placenta is the organ of transfer of oxygen and nutrients from mother to the fetus, whereas CO2 and metabolic wastes are transferred out from fetus to the mother. Villous trophoblasts form chorionic villi which transports oxygen and nutrients primarily, while extravillous trophoblasts are further classified as interstitial and endovascular trophoblasts. The interstitial trophoblasts invade the decidua while endovascular trophoblasts penetrate the spiral artery lumen.[6]

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Early pregnancy loss

Early pregnancy loss is defined as a “nonviable intrauterine pregnancy with either an empty gestational sac or a gestational sac containing an embryo or fetus without heart activity within the first 12 6/7 weeks of gestation”. In the first trimester, the terms miscarriage spontaneous abortion and early pregnancy loss are used interchangeably as there is no consensus on terminology in the literature.[1]

According to American society of reproductive medicine (ASRM), “recurrent pregnancy loss is a distinct disorder defined by two or more failed clinical pregnancies”.

[3]

According to National health portal data published in june 2018, spontaneous abortion occurs in 15-20% of all clinically recognized pregnancies, out of which 80% are first trimester abortions.[8]

Ameet Patki et al did an epidemiological study to determine the prevalence and risk factors associated with recurrent spontaneous abortion in India. They concluded that the prevalence of RPL in Indian women was 7.46%. The probability of subsequent abortion after first abortion was 0.25, while it was 0.34 and 0.22 after second and third abortion respectively.[9] Data from a study conducted in Bangladesh published in 2019 showed that relative risk of subsequent abortion after one spontaneous abortion was 23.2 and 1.8 within 6 months and 7-14 months respectively.[10]

Most of the clinicians start investigating for the cause of pregnancy loss after 2 or 3 abortions. There are no studies done so far to determine the significance of initiating investigation after one pregnancy loss.

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Evaluation

As per **ESHRE 2018,** following investigations are recommended for the patients of RPL [11] . There are no studies/ recommendations for the evaluation of first pregnancy loss (Table 1).

Table 1: ESHRE recommendations for RPL (2018)

ETIOLOGY	DIAGNOSTIC EVALUATION
Genetic	Parental karyotype (not routinely recommended) Genetic analysis of pregnancy tissue (not routinely recommended)
Thrombophilia	Lupus anticoagulant Anticardiolipin antibodies (IgG and IgM) ß2 glycoprotein I antibodies and hereditary thrombophilia (can be considered)
Endocrinological	TSH TPO antibodies Assessment of PCOS, fasting insulin and fasting glucose, testing for ovarian reserve may be considered
Anatomic	3D Ultrasound Sonohysterography (SHG) Hysterosalphingography (HSG)
Male factor	Sperm DNA fragmentation (can be considered)

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Investigations not recommended are:

1. HLA determination
2. Anti HY antibodies
3. Cytokine testing and polymorphisms
4. NK cell testing
5. Anti- HLA antibodies
6. Serum prolactin
7. Luteal phase insufficiency
8. Androgen
9. LH

10. Homocysteine plasma levels

RCOG Guidelines for evaluation of RPL [12]

1. Antiphospholipid syndrome

   It is one of the preventable cause of RPL. Fifteen percent of the patients with RPL have antiphospholipid antibodies (APLA). RPL patients with APLA antibodies without pharmacological intervention can have live birth rate as low as 10%. So all women with RPL should be screened before pregnancy for anti- phospholipid antibodies.

2. Karyotype

   Cytogenetic analysis should be performed on products of conception of the third and subsequent consecutive miscarriage. Parental peripheral blood karyotyping of both partners should be performed in couples with recurrent miscarriage if the products of conception show an unbalanced structural chromosome abnormality.

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3. Anatomical factors

   Pelvic ultrasound to be done in all women with recurrent first and second trimester miscarriages. To confirm the diagnosis, 3D ultrasound, hysteroscopy and laparoscopy can be used.

4. Thrombophilia

   Women with recurrent second trimester miscarriage should be screened for APS and inherited thrombophilia including protein S deficiency, factor V leiden mutation, factor II (prothrombin) gene mutation.

5. Infectious agents

   Toxoplasmosis, rubella, cytomegalovirus, herpes and listeria infections are not capable of persisting in the genital tract to cause RPL. So the routine TORCH screening should be avoided.

6. Endocrine

   Women with diabetes with high HbA1c levels in the first trimester are at a risk of pregnancy loss and fetal malformation. However, well controlled diabetes mellitus is not a risk factor.

ETIOLOGY

1. ENDOCRINE CAUSES

Endocrine factors contribute as a cause of RPL in 8-20 % of the patients [13] [14]

There are no Indian studies to look for endocrine causes as a cause of RPL.

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A.THYROID DYSFUNCTION

Sarkar et al reviewed the literature for RPL in patients with thyroid dysfunction and concluded that even minimal hypothyroidism can adversely affect pregnancy outcome and can cause miscarriage and cognitive abnormalities in the child later [15]

The exact mechanism by which thyroid hormone affects fertility is unclear. However, in the literature , it has been proposed that it has a potential role in the development of reproductive system thus having an impact on fertility. There might be an association of subclinical hypothyroidism with ovulatory dysfunction. [16]

In pregnancy there is a shift from T helper -1 to T helper -2 lymphocyte state. Thyroid autoimmunity increases the rate of abortion. The incidence of thyroid peroxidase antibodies in women at 14 weeks of gestation was found to be 10%.It increases the risk of abortion,gestational thyroid dysfunction and predisposes to postpartum thyroiditis. Thyroxine supplementation does not seem to confer benefit [17].

Thyroid antibodies were proposed to alter fertility by targeting zona pellucida and HCG receptors. The presence of these antibodies leads to activation of immune system and exaggerated autoimmune response against the fetus and placenta. The exact mechanism remains unclear though. Thirty one precent of pregnancies in women with thyroid antibodies ended in miscarriage , which was shown with sensitive Hcg assays, two third of them occurring before the usual clinical detection [18].

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Thyroid antibodies can exert their effect in both TSH dependent and TSH independent manner. Vitamin D deficiency has already been studies as a predisposing factor for autoimmune diseases and its levels were found to be low in women with thyroid antibodies [19].

Most of the studies show an association of thyroid antibodies and increased rates of abortion but patients with high titres of antibodies do not have higher rates of abortion than patients with low titres of antibodies. It is still controversial to give thyroxine supplementation before or during pregnancy in euthyroid women with autoimmune thyroid disease [20]. Various etiological factors of RPL are discussed in Table 2.

Table 2: Etiology of RPL

S. No.	Causes	Author	Percentage contribution to RPL
1.	**Endocrine** DM	Shetty MB et al[21](2017)	26%
	Hypothyroidism	Shetty MB et al[21] (2017)	12.8%
	PCOS	Li TC et al[22] (2000)	7.8%
2.	Anatomical factors Uterine anomaly	Salim R et al[23] (2003)	5.3%
3.	Chromosomal abnormalities Robertsonian and reciprocal translocation and inversion	De BM & Dao TN et al[24] (1990)	4.7%
4.	Infections	Ford HB et al[14] (2009)	0.5-5%
5.	Unexplained	El Hachem et al[25] (2017)	50%
	Thrombophilia amongst unexplained RPL Acquired Inherited	Patil R et al[26] (2015) Patil R et al[26] (2015)	24% 16%

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B. DIABETES MELLITUS

Diabetes mellitus is a known cause of pregnancy loss. Type 1 diabetic women with poor glycaemic control are found to have increased rates of miscarriage.

Todorova et al performed a study in women with type 1 diabetes to find out the correlation between glycemic control and occurrence of spontaneous abortions. They did a prospective study in 75 women over a period of one year. The women were divided into 3 groups , first: 30 pregnant women with type 1 DM with normal

outcome. Second: 16 pregnant women with type 1 DM with spontaneous abortion. Third: 29 healthy pregnant controls. They measured the levels of glucosylated hemoglobin, glutathion peroxidase enzyme levels and selenium in these patients. ***The levels of selenium were found to be lower in all three groups. The activity of glutathion peroxidase enzyme was found to be increased in diabetic pregnant women with spontaneous abortions probably due to increased antioxidative defence of the cell.*** So it was proposed that spontaneous abortion might be due to high level of pre-prandial glycemia and the ineffective antioxidant defense [27].

Casson et al did a 5 year population based cohort study to find out the outcomes of pregnancy in insulin dependent diabetic women. They included 462 pregnancies in 355 women with insulin dependent diabetes from 10 different centers over 5 years. The incidence of spontaneous abortion was found to be 17 % in their study group [28].

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Miodovnik et al performed a study to find out the glycemic control in insulin dependent diabetic women with spontaneous abortion. They measured the levels of glycosylated hemoglobin in women with insulin dependent diabetes with spontaneous abortion in immediate post abortal period and in the first trimester for those with normal outcome. They evaluated 116 pregnancies in 75 insulin dependent diabetic women. Twenty six pregnancies ended up in spontaneous abortion before 20 weeks. The levels of glycosylated hemoglobin in women with normal pregnancy outcome at 8-9 weeks period of gestation was < 12%, whereas it was > 12% in women with spontaneous abortion indicating poor glycemic control [29].

C. PCOS

PCOS women are at increased risk of miscarriage . The incidence of early pregnancy loss was found to be 30-50% in PCOS women as compared to 10-15% in normal women [30]. Homburg et al proposed that the incidence of spontaneous abortion was higher in PCOS women who conceived with ovulation induction as compared to those who conceived spontaneously [31]. Several mechanisms have been proposed to cause increased risk of abortions in PCOS women.

Elevated LH levels have been linked to cause early pregnancy loss in women with PCOS. Regan L et al performed a prospective study in 193 women to study the effect of increased LH levels on infertility and miscarriage. ***They concluded that 65% of pregnancies in high LH group ended in abortion whereas only 12% in the normal LH group had abortion*** [32]. Clifford K et al in their randomized controlled trial concluded that pre-pregnancy suppression of high levels of LH in ovulatory women with RPL does not improve the pregnancy outcome [33].

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Apparao KB et al proposed that increased incidence of abortions in PCOS women might be due to increase in serum androgens and endometrial androgen receptor. The endometrial receptivity may be reduced by the combination of these as they found down regulation of alpha(v)beta3 integrin in these patients [34]. Glycodelin and insulin like growth factor binding protein-1 (IGFBP-1) play an important role in implantation and maintenance of pregnancy. The levels of both of these endometrial secretary proteins are found to be lower in PCOS women [35].

Glueck et al postulated that plasminogen activator inhibitor-1 (PAI-1) activity is an independent risk factor for miscarriage in PCOS women. It causes increased

thrombosis of placental bed leading to placental insufficiency due to impaired fibrinolysis [36].

D. HYPERPROLACTINEMIA

In vitro studies done in the past have shown that there is a role of prolactin the maintenance of corpus luteum and progesterone production in early pregnancy. Tal J et al conducted a study to find the effect of stress related hormones like prolactin on the production of beta HCG from placental products grown in vitro culture. They found that prolactin helps in early implantation by causing inhibition in the pulsatile secretion of HCG [37].

2. THROMBOPHILIA

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Vora S et al performed a study to find out the role of congenital and acquired thrombophilia as a cause of unexplained pregnancy loss. They included 380 women with two or more abortions for a period of 6 years in their study group. The investigations included lupus anticoagulant (LA), IgG and IgM anticardiolipin antibodies (ACLA), beta 2 glycoprotein 1 (beta2GP1) and annexin V. The genetic markers studied were protein C (PC) , protein S (PS) , antithrombin III (AT III), factor V leiden (FVL), PT gene G20210A, MTHFR C677T, EPCR 23 bp insertion and PAI 4G/5G polymorphisms.

They found a strong association of pregnancy loss with ACLA, annexin V and LA in the descending order. They did not find association between antiphospholipid antibodies and time of pregnancy loss except for LA which was associated more with early pregnancy loss than late pregnancy loss. Amongst heritable thrombophilia, the risk was highest with PS deficiency followed by PC deficiency [4].

There are no studies done so far to find out the association of thrombophilia with first early pregnancy loss.

A. INHERITED THROMBOPHILIA

Rey E et al published a meta-analysis of 31 studies to know the association between thrombophilia and pregnancy loss [38]. The following factors were analysed in this study

FACTOR V LEIDEN MUTATION

Factor V Leiden mutation was found to be most common cause of inherited thrombophilia. In this meta-analysis, factor V leiden was associated with early recurrent pregnancy loss (OR 2.01, 95% CI 1.13-3.58), late recurrent pregnancy loss after 22 weeks (OR 7.83, 95% CI 2.83-21.67) , late non recurrent pregnancy loss after 19 weeks (OR 3.26, 95% CI 1.82-5.83).

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PROTHROMBIN GENE 20210A MUTATION

Prothrombin gene mutation was found to be associated with early recurrent pregnancy loss (OR 2.56, 95% CI 1.04-6.29) and late non recurrent pregnancy loss (OR 2.30, 95% CI 1.09-4.87).

PROTEIN S DEFICIENCY

Protein S deficiency was associated both with recurrent pregnancy loss (OR 14.72, 95% CI 0.99-218.01) and late non recurrent pregnancy loss (OR 7.39, 95% CI 1.28-42.63).

PROTEIN C AND ANTITHROMBIN III DEFICIENCY

Protein C and antithrombin III deficiency were not found to be significantly associated with fetal loss

Hyperhomocysteinemia from genetic polymorphism of MTHFR gene C6777, methylene tetrahydrofolate mutation, folate and vitamin B12 deficiency were not found to be associated with fetal loss.

CQUIRED THROMBOPHILIA

Antibodies detected in women with RPL include :

1. ANA
2. LA
3. ACLA
4. Beta 2 glycoprotein 1

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ANTIPHOSPHOLIPID ANTIBODY SYNDROME

The diagnostic criteria followed worldwide was presented first in International antiphospholipid symposium 1999 in Sapparo, Japan. At least one of the following clinical criteria and one of the laboratory criteria must be present.

The clinical criteria include:

1. ≥ 1 unexplained death of a morphologically normal fetus at or beyond the 10^{th} week of gestation, with normal morphology documented by ultrasound or direct examination of fetus
2. One or more premature births before 34 weeks of gestation as a consequence of severe preeclampsia (or) placental insufficiency
3. ≥ 3 consecutive spontaneous miscarriages before 10 weeks of gestation

The laboratory criteria include:

The presence of APLA, on two or more occasions at least 12 weeks apart and not more than 5 years prior to clinical manifestations, as demonstrated by one or more of the following

1. IgG and/or IgM ACLA in moderate or high titre (>99th percentile)
2. Anti beta 2 glycoprotein of IgG/IgM at moderate to high titre (>99th percentile)
3. Lupus anticoagulant was detected by screening method of APTT

Clinically APS is divided into

Primary APS

Secondary APS

PRIMARY APS

Occur as a primary condition in women with no other recognizable autoimmune disease.

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SECONDARY APS

Occurs in patients with underlying autoimmune disease . Example, SLE, RA, sjogren syndrome.

Henk J Out[39] et al studied the obstetric risks in pregnant women with antiphospholipid antibodies. They found that APLA positive women had low birth weight babies.

Fosca A et al[40] reviewed the literature for antiphospholipid syndrome during pregnancy. There is increased chance of preterm delivery, oligohydramnios, pre- eclampsia, eclampsia, HELLP syndrome, IUGR, fetal or neonatal thrombosis. There is increased risk of venous thrombosis in pregnancy and post-partum period (0.61-1.72 per 1000 deliveries).

3. UTERINE CAUSES

A. UTERINE ANOMALIES

Saravelos SH et al done a study to find out the pattern of pregnancy loss with congenital uterine anomalies and RPL. They included 665 women with recurrent pregnancy loss and screened them for uterine anomalies using 2D ultrasonogram and hysterosalpingography. All the patients who were suspected to have congenital uterine anomalies were classified using a combined hysteroscopy/laparoscopy procedure. They compared the pregnancy outcome for each type of uterine anomaly with a control group of women with unexplained recurrent miscarriage. ***They concluded that the second trimester miscarriage rate in women with septate and bicornuate uterus was 13.2% and 13.8% as compared to controls (1%). However, the rates of biochemical pregnancy loss were lower in women with congenital uterine anomalies as compared to controls. It was 9.5% in women with arcuate uterus as compared to controls (30.4%).***[41]

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According to a systemic review of the prevalence of congenital uterine anomalies in unselected and high risk populations, uterine anomalies were found in 13.3% of women with a history of miscarriage and 24.5% of those with miscarriage and infertility. They reviewed 94 observational studies involving 89,861 women.[42]

B.UTERINE PATHOLOGY

FIBROID UTERUS

Saravelos SH et al concluded that uterine fibroids increase mid trimester pregnancy loss among women with RPL. The prevalence of fibroids in their study was found to be 8.2%. Resection of submucosal and intramural fibroids can prevent abortions and double the live birth rates, whereas no intervention is required in subserosal fibroid as they don't distort cavity.[43]

According to ASRM committee review, myomectomy may be considered in asymptomatic infertile women with cavity distorting fibroid to prevent pregnancy loss.[44]

Different mechanisms have been proposed as a cause of abortion due to fibroid like uterine cavity is distorted by submucosal fibroid, implantation of embryo at a poorly decidualised endometrium or sometimes due to rapid fibroid growth, blastocyst may get expelled before the implantation of placenta owing to increased uterine irritatability and contractility.

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INTRAUTERINE ADHESIONS

Heinrich Fritsch in 1894 published the first case of intrauterine adhesions. However, only after 54 years Ashermann syndrome was fully described by Joseph Ashermann when he identified this pathology in 29 women who presented with amenorrhoea and were found to have cervical stenosis and later he postulated that this manifestation might be due to endometrial trauma [45] . Conforti et al reviewed the literature in 2013 and concluded that it is a cause of menstrual disturbances, infertility and placental abnormalities. [46]

4. CERVICAL CAUSES

A. CERVICAL INCOMPETENCE

It is a well known cause of mid trimester abortions. Patients give typical history of painless cervical dilatation and expulsion of fetus. The incidence is reported to be 1%.[47]

Bartolucci et al proposed the following criteria described in Table 3 for the diagnosis of cervical incompetence.[48]

Table 3. Criteria for diagnosis of cervical incompetence

Cervical length	- Less than 3 cm
Internal os width	- More than 1.5 cm in first trimester More than 2 cm in second trimester
Lower uterine segment and internal os shape	-T/Y/V/U
Thinning of anterior part of lower uterine segment	-Less than 0.6 cm
Morphology of membranes in internal os and endocervical canal	-Bulging / Funneling
Cervical Index*	- ≥0.52

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(* Cervical Index = Funnel length +1 / Endocervical length (described by Gomez et al. [49])

B. CERVICAL INFECTIONS

Giakovumelou et al reviewed the literature for the role of infections in miscarriage. The effects of Chlamydia trachomatis, Ureaplasma urealyticum, Toxoplasma gondii, HPV, herpes simplex virus is still controversial. Further studies are required to prove the definite relationship of certain infections with miscarriage and whether screening of these in pregnancy would confer improvement in reproductive outcome.[50]

5. CORPUS LUTEAL DYSFUNCTION

Luteal phase insufficiency is due to inadequate production of progesterone. Progesterone is necessary for transformation of endometrium to secretory to aid in implantation and maintenance of early pregnancy[51]. It can also occur due to suboptimal response of endometrium to normal amount of progesterone.

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Tamura et al studied the changes in the corpus luteum blood flow during the luteal phase and early pregnancy. In normal women, there was high resistance index (RI) during late follicular phase. But the RI was low in the mid luteal phase indicating increase in blood flow, while there was increase in RI with the regression of corpus luteum. During first 7-8 weeks of pregnancy , the RI remains low till the corpus luteum regresses [52].

The incidence of luteal phase defect was found to be 17.4 %- 28% amongst women with RPL[53]. Hey NA et al studied that MUC1 which is a cell surface and secretary product of endometrial epithelium was found to be lower in uterine flushings of RPL women. Thus, they concluded that MUC1 may be involved in very early stage of implantation[54].

6. GENETIC CAUSES

Suzumori N et al in their review article concluded that aneuploidy in fetus occurs in 5-10 % of all pregnancies and most of these fetuses end up in abortion. Parental chromosomal aberrations is a major pre-disposing factor. Maternal or paternal translocation is one of the most important cause of RPL. Recently many genetic polymorphisms have been found to be implicated in causing RPL [55]

De Braekeeler M et al reviewed database of 22,199 couples for genetic cause in patients of RPL. They found that in 4.7% of cases with RPL, there was one carrier of chromosomal aberrations. They also concluded that only translocations (both reciprocal and robertsonian) and inversions were associated with pregnancy loss in patients of RPL [24]

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Sheth et al did a retrospective cytogenetic study in india on 4859 patients with history of RPL. They found chromosomal aberrations in 3.5% of the patients. Inversion of Y chromosome was commonest (57.7%) followed by chromosome 9 (32.05%). Reciprocal translocations constituted 24.7% of cases, while robertsonian translocations were detected in 17.64% of the cases [56].

Ogasawara M et al performed a retrospective study in 1309 patients with recurrent first trimester abortions to find out the frequency of chromosomal abnormalities in products of conception in relation to the number of abortions.Patients with previous normal karyotype were found to have more subsequent abortions than those with abnormal karyotype. Thus they proposed that a normal fetal karyotype in previous pregnancy may be a predictor of next abortion [57].

7. INFECTIONS

Giakoumelou et al reviewed the literature for the role of infections in miscarriage. They concluded that there is an associated increased risk of miscarriage with systemic infections with malaria, brucellosis, CMV, HIV, dengue fever, influenza virus and vaginal infection with bacterial vaginosis. Q fever, adeno virus, Boca virus, Hepatitis C, Mycoplasma genitalium do not affect pregnancy outcome. The effects of Chlamydia trachomatis, Toxoplasma gondii, HPV, herpes simplex virus, parvovirus B19, hepatitis B is still controversial. Further studies are required to prove the definite relationship of certain infections with miscarriage and whether screening of these in pregnancy would confer improvement in reproductive outcome[50]

The exact pathogenic mechanism by which an infectious agent causes miscarriage is unknown. The various mechanisms proposed are:

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1. Plasmodium enters through maternal circulation and can invade trophoblast and multiply in it [58].
2. Listeria monocytogenes crosses the intestinal barrier to enter the maternal circulation and uses bacterial surface proteins internalin A and B to invade the placenta [59].
3. CMV can also replicate in trophoblasts and induce an inflammatory response which in turn can increase apoptosis leading to cell death [60]
4. For bacterial infections, studies in mice have shown that nitric oxide and prostaglandins produced along with LPS (lipopolysaccharides) can result in embryonic resorption [61].

Bacterial vaginosis is present in about 25% of reproductive age women [62]. Donders et al conducted a study in 759 belgian pregnant women. In this cohort,

8.4% of the patients presented with bacterial vaginosis and they were not treated. Two percent of the patients positive for bacterial vaginosis had an abortion before 25 weeks of gestation with an OR of 6.6 (OR 6.6;95% CI 2.1-20.9). They found an association of miscarriage with the absence of lactobacilli also with an OR of 4.9 (OR 4.9; 95% CI 1.4-16.9) [63]. ***As per Cochrane review including 7847 women and 21 trials, after antibiotic administration there was decreased risk of late miscarriage*** (RR 0.20; 95% CI 0.05-0.76; two trials, 1270 women, fixed effect, I2= 0%) and further studies are required to establish the benefit of screening programmes for prevention of adverse reproductive outcome [64].

Hong FC et al conducted a 10 year prospective study in Shenzhen, china from 2002 to 2012 to find out the reduction in mother to child transmission of syphilis after introduction of a national screening programme. They found that the adverse outcomes like abortions were reduced from 27.3 % in 2003 to 8.2% in 2011 [65].

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8. AGE

It is well known that the risk of spontaneous miscarriage increases with advancing age. The risk of 3 spontaneous miscarriages in a woman of age <25 years is 0.13%, whereas it is 100 times (13%) for a woman of age >40 years [66]

Marquard K et al performed a retrospective cohort study to find out the causes of RPL in women of age >35 years. They included 43 RPL women in their study and the investigations performed were cytogenetic analysis of products of conception, uterine cavity evaluation, TSH, APLA and parental karyotype. The chromosomal anomalies were found in 78 % of cases. Out of 43, 5 patients were positive for thrombophilia, 4 had antiphospholipid antibody syndrome, while one patient had

protein C deficiency. Forty patients out of 43 had normal uterine cavity. Serum TSH and parental karyotype were normal for all patients. Unexplained RPL constituted 18 % in this study. Had cytogenetic analysis of products of conception not included, the unexplained RPL would have been 80%. Thus concluding that chromosomal anomalies constitute the major cause of abortions in women over the age of 35 years [67]

Nybo Anderson et al did a population based register study and the age wise risk of RPL in their study is shown in the table 4. [68]

Table 4. Age: Risk of RPL

Age group (years)	Spontaneous miscarriage (%)
20-24	11
25-29	12
30-34	15
35-39	25
40-44	51

Nybo Andersen et al[68]

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9. OBESITY

The mechanism behind the increased incidence of abortion amongst obese women is not well understood. Obesity affects the metabolism of steroids and proteins like leptin, adiponectin and affects the secretion of androgens and sex hormone binding globulin which can affect pregnancy outcome. It can also lead to poor quality egg and abnormal endometrial development leading to implantation problems.

Cavalcante MB et al performed a recent meta-analysis which showed that obese women with a history of RPL have a risk of future pregnancy losses, but they found no risk between overweight women and pregnancy loss [69]. Lo W et al did a study to find out the effect of BMI on the outcome of pregnancy in women with unexplained recurrent miscarriage. They concluded that obesity significantly increases the risk of abortion in these women (OR 1.73; 95%CI 1.06-2.83). ***Asian women with BMI similar to Caucasian women had further high risk*** (OR 2.87; 95% CI. 1.52-5.39) [70].

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Bhandari HM et al did a retrospective observational study in 414 RPL women and concluded that cumulative pregnancy rates in obese women were higher 65.2% and 80% by three and six months as compared to women with normal BMI 49.2% and 65.8 % at 3 and 6 months respectively. They postulated that obese women may be superfertile but have more chance of miscarriage possibly due to the effects of obesity on endometrium [71]. Obesity and risk of RPL as per various studies is discussed in table 5.

Table 5. Obesity: Risk of RPL

S. No.	Author (Year)	Main outcome
1.	Boots et al[72] (2014)	Obese women had higher frequency of abortion as compared to non obese (58% vs 37%)
2.	Metwally et al[73] (2010)	Obese women had higher risk for subsequent miscarriage (OR, 1.71; 95% CI, 1.05-2.8)
3.	Lashen et al[74] (2004)	Risk of early pregnancy loss and RPL was higher among obese women (OR 1.2, 95%CI; 1.01-1.46) (OR 3.5, 95%CI; 1.03-12.01)

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10. UTERINE NK CELLS

Sharma et al postulated that there is a role of angiogenic properties of uterine NK cells in incorporating the angiogenic property to the trophoblast cells. Thus the unscheduled breakdown of this property might explain some of the cases of unexplained recurrent spontaneous abortion. Uterine NK cells appear to regulate placental and trophoblast growth, local immunomodulation and control trophoblast invasion. Peripheral NK cells may be used to understand the biology of NK cells in detail. More studies are required to know the relationship between uterine NK cell numbers and future pregnancy outcome in patients with RPL [75].

Yeh, Ching chang et al did a case control study in china to know the role of NK cells and cytokines (IL-2 and IL-12) in the prediction of women who have a higher risk of recurrent miscarriage. They also postulated that an increased percentage of CD56+ CD16+ (≥ 5.25%) or CD56+CD16- (≥ 3.4%) cells in the peripheral blood

is found in the women with RPL and these findings can be used prospectively to know the women at risk for RPL [76].

11. UNEXPLAINED RECURRENT PREGNANCY LOSS

Vandana Rai performed a metanalysis to know the risk of RPL in women with methylenetetrahydrofolate reductase C677T polymorphism. MTHFR is an enzyme which catalyzes the conversion of 5,10- methylenetetrahydrofolate to 5-methyltetrahydrofolate. Failure of this conversion can break the pathway of conversion of homocysteine to methionine, which may lead to abnormal DNA methylation and DNA strand breaks. There are 40 different genetic polymorphisms of MTHFR, out of which C677T variant is clinically important and well studied in literature. She concluded that there is a strong relationship between the MTHFR C677T variant and RPL in asian population and folate has an important role in its prevention [77].

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Yetunde Ibrahim et al reviewed the literature regarding the male contribution to RPL. Male gamete contributes 50% of the genetic material to the developing fetus still the role of male factors in causing abortions is not well understood. Only karyotype analysis of male partner is recommended in the evaluation of RPL. Structural chromosomal abnormalities, sperm DNA fragmentation, Y chromosome microdeletions can lead to pregnancy loss. Research is ongoing for the role of sperm aneuploidy, methylenetetrahydrofolate reductase (MTHFR) polymorphisms, Annexin A5 M2 haplotype, Ubiquitin-specific protease (USP26) gene alterations and shortened telomere length [78].

Wald KA et al did a prospective study in 264 RPL patients to know the prevalence of diminished ovarian reserve (DOR) in unexplained RPL patients. Out of 264 patients, 87 (33%) had an identifiable cause , while 177 patients were considered to have unexplained RPL. Forty eight percent of the patients with unexplained RPL had diminished ovarian reserve, while the prevalence of DOR in patients with known cause of RPL was 29%. It was more significant in patients <38 years old (22%) as compared to 12% in patients >38 years old [79].

MANAGEMENT OF KNOWN CAUSES OF RPL

A. ENDOCRINE FACTORS

1. PCOS

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Kamalanathan S et al reviewed the literature for pregnancy in polycystic ovary syndrome. They concluded that there is lack of evidence for the benefit of metformin in the management of pregnancy complications, more placebo controlled randomized trials are required to analyse it [80]

Lovvik et al performed a randomized double blind placebo controlled trial in which they used metformin to treat pregnant women with PCOS. They included 487 women in their study and randomly assigned women to metformin group (n=244) or placebo (n=243).The incidence of late miscarriage and preterm birth was 5% in the metformin group and 10% in the placebo group (OR 0.50, 95% CI 0.22-1.08; p=0.08).There was no difference in the incidence of gestational diabetes in both the groups. [81]

B. THROMBOPHILIA

1. ANTIPHOSPHOLIPID ANTIBODY SYNDROME

Maria G Tektonidou et al performed a systematic literature review for management of thrombotic and obstetric antiphospholipid syndrome. They retrieved 7534 articles and included 188 articles in the review. Their results were as follows: [82]

- **Pregnant women (with or without SLE) with high-risk aPL profile but with no history of thrombosis or pregnancy complications :** Low dose aspirin may be of benefit.
- **Pregnant women with a history of 'criteria' Obstetric APS :** Combination treatment with low dose aspirin and heparin is better than low dose aspirin alone. It also resulted in reduced rate of miscarriages. More RCTs are required to determine the differences in risk of preterm delivery, pre-eclampsia or IUGR.
- **Women with a history of 'non-criteria' Obstetric APS :** More RCTs are required to prove the efficacy of low dose aspirin and heparin in these women to improve the live birth rate. Alijotas-Reig J et al did a comparative study and they compared 71 women with non-criteria obstetric APS complications (two consecutive miscarriages < 10 weeks, delivery ≥ 34 weeks, late IUGR, abruption at term or placental hematoma) who received treatment with low dose aspirin and heparin with 20 untreated women. The live birth rate was 81.7% in the treatment group as compared to 55% in the non-treatment group[83].

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- **Women with a history of recurrent pregnancy complications despite treatment with low dose aspirin and prophylactic dose heparin:**

  Increase of heparin to therapeutic dose: No studies are done so far whether increasing heparin to therapeutic dose would be of benefit in these patients.

  Addition of HCQ: Mekinian A et al did a comparative study to find out the pregnancy losses among women with refractory obstetric APS before and after the addition of HCQ along with heparin and low dose aspirin. The pregnancy losses reduced from 81% to 19% in the group where HCQ was used [84].

  Addition of low- dose prednisolone in the first trimester: Bramham K et al performed a retrospective cohort study in which they compared pregnancy outcome in 23 women treated with prednisolone 10 mg/ day until 14 weeks in combination with low dose aspirin and heparin with 93 women treated with low dose aspirin and heparin. The live birth rate increased from 4% to 61% after supplementation with prednisolone [85].

  Addition of intravenous immunoglobulin: Vaquero E et al did a prospective study to compare the outcome of pregnancy in refractory APS patients who received intravenous immunoglobulin versus prednisone plus low-dose aspirin. They included 82 patients in their study. Twenty nine patients received prednisone and low dose aspirin , while 53 received intravenous immunoglobulin. They did not find significant difference in live birth rates in both the groups that is 78% and 76% respectively [86].

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Pregnant women with a history of thrombotic APS: Treatment with low dose aspirin and therapeutic dose of heparin.

INHERITED THROMBOPHILIA

Reccommendations by RCOG for thromboprophylaxis for women with inherited thrombophilias in pregnancy [87] (Table 6)

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Table 6: RCOG recommendations for thromboprophylaxis for women with inherited thrombophilias in pregnancy

<table>
<tr><th>History of VTE</th><th>Inherited thrombophilia</th><th>Antenatal management</th><th>Postnatal management</th></tr>
<tr><td rowspan="2">Previous VTE</td><td>Antithrombin deficiency</td><td>50-100% treatment dose LMWH
Involve hematologist
Anti-Xa monitoring</td><td>50-100% treatment dose LMWH * 6 weeks / until oral (PO) Anticoagulation started</td></tr>
<tr><td>All others</td><td>Consider prophylactic dose LMWH</td><td>Prophylactic dose LMWH* 6 weeks</td></tr>
<tr><td rowspan="6">Asymptomatic</td><td>Antithrombin deficiency</td><td colspan="2" rowspan="3">Not recommended</td></tr>
<tr><td>Protein C deficiency</td></tr>
<tr><td>Protein S deficiency</td></tr>
<tr><td>Compound heterozygotes</td><td colspan="2" rowspan="3">If more than one thrombophilic defect, consider prophylactic dose LMWH
Consider prophylactic dose LMWH in the presence of three other risk factors/ from 28 weeks if two other risk factors</td></tr>
<tr><td>Homozygous FVL</td><td rowspan="2">If more than one thrombophilic defect, for prophylactic dose LMWH * 6 weeks
Prophylactic dose LMWH* 10 days if one other risk factor</td></tr>
<tr><td>Homozygous prothrombin gene mutation</td></tr>
<tr><td>Heterozygous FVL</td><td></td></tr>
</table>

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Reccommendations by ACCP (American college of chest physicians) for thromboprophylaxis for women with inherited thrombophilias in pregnancy (Table 7) [88]

Table 7: ACCP recommendations for thromboprophylaxis for women with inherited thrombophilias in pregnancy

History of VTE/ pregnancy complication	Inherited thrombophilia	Antenatal management	Postnatal management
Previous VTE	Any inherited thrombophilia	Prophylactic or intermediate dose LMWH	Prophylactic or intermediate dose LMWH * 6 weeks
	All others	Consider prophylactic dose LMWH	Prophylactic dose LMWH* 6 weeks
Asymptomatic but has family history of VTE	Homozygous for Factor V Leiden	Prophylactic or intermediate dose LMWH	Prophylactic or intermediate dose LMWH or vitamin K antagonists (INR 2-3) * 6 weeks
	Homozygous for prothrombin gene mutation	Prophylactic or intermediate dose LMWH	Prophylactic or intermediate dose LMWH or vitamin K antagonists (INR 2-3) * 6 weeks
	Protein C or S deficiency	Thromboprophylaxis not recommended	Prophylactic or intermediate dose LMWH * 6 weeks
	All other inherited thrombophilia	Thromboprophylaxis not recommended	Prophylactic or intermediate dose LMWH or vitamin K antagonists (INR 2-3) * 6 weeks
Asymptomatic and no family history of VTE	Any inherited thrombophilia	Thromboprophylaxis not recommended	Thromboprophylaxis not recommended
Previous pregnancy complications	Any inherited thrombophilia	Thromboprophylaxis not recommended	Thromboprophylaxis not recommended
High risk of pre-eclampsia	Irrespective of thrombophilia history	Low- dose aspirin from second trimester	Thromboprophylaxis not recommended

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C. UTERINE CAUSES

Nouri K et al performed a retrospective cohort study to find out the reproductive outcome after hysteroscopic septoplasty in patients with septate uterus. Sixty four women with septate uterus underwent hysteroscopic septoplasty in their study period. They also performed a systematic review of literature and identified 18 studies investigating the reproductive outcome after septoplasty. Pooled analysis of data including their own study showed an overall pregnancy rate of 60% (892/1501) and a live birth rate of 45% (686/1501) [89].

The ASRM also recommended that 'it is reasonable to consider septum incision [90].

Casini ML et al did a prospective controlled study to find out whether the location of fibroid and removal of fibroid before pregnancy would improve pregnancy rate and reduce abortions. They included 181 women with uterine fibroids in their study. Amongst the group of patients who underwent myomectomy, the pregnancy rates were 43.3% in cases of submucosal fibroids, 56.5% in intramural, 40% in submucosal-intramural and 35.5% in cases of intramural-subserosal fibroids respectively. Among the other group of patients who did not undergo surgical treatment, the pregnancy rates were 27% in submucosal, 41% in intramural, 15% in submucosal-intramural, 21.4% in intramural-subserosal fibroids. The results were stastically significant in submucosal and submucosal-intramural group [91]

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Saravelos et al did a similar study in 25 women with fibroid who underwent myomectomy. They concluded that live birth rates were 23% prior to resection and 53% post resection [43].

Conforti et al reviewed the literature regarding the management of ashermann syndrome. They concluded that treatment of ashermann syndrome by hysteroscopic surgery or dilatation and currettage increases the pregnancy rate from 40% to 63%. There is a role of intrauterine devices, uterine stent, adhesion barriers and hormonal treatment to prevent adhesions, but comparative trials are needed to prove their efficacy [46].

D. CERVICAL INCOMPETENCE

Cervical cerclage is only recommended in women with previous history of mid trimester losses in whom cervical length shows shortening, funneling of cervix, >2 cm width of internal os on USG in 2nd trimester as it is associated with potential hazards related to surgery with risks of stimulating uterine contractions. History indicated cerclage is only indicated for ≥2 losses [92].

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Okusanya BO et al did a retrospective study to determine the outcome of pregnancy with history indicated cervical cerclage in RPL patients. The fetal salvage rate was 75% and repeat spontaneous miscarriage was 5.6% [93].

Kyong-No Lee et al did a retrospective observational study to find out the association between history indicated cerclage based on the number of previous second trimester losses and pregnancy outcome to validate the new ACOG recommendation. They concluded that the incidence of preterm delivery was less in patients with previous 1 loss than ≥2 losses (8% and

14%). The rates of PROM, PPROM and NICU admission were similar in both the groups [94]

Li-Quong Zhu et al did a retrospective study in 158 cases to find out the safety and effectiveness of emergency/rescue cervical cerclage and pregnancy outcome. The live birth rate was 82% in their study. The mean interval between cerclage and delivery was 52.1±26 days. The outcome was influenced by the degree of cervical dilatation, post operative CRP value and WBC counts [95].

E. LUTEAL PHASE DEFICIENCY

The treatment options for women with LPD include ovulation induction, supplementation with HCG and progesterone supplementation.

Cochrane review of 94 randomised controlled trials in 26,198 women comparing different regimens of luteal phase support. They concluded that hCG or progesterone given during the luteal phase may be associated with higher live birth rates than no treatment, but the evidence is inconclusive regarding the same. The addition of GnRh agonists to progesterone may improve outcome. Route of progesterone administration or the addition of estrogen appear to have no effect on pregnancy outcome. hCG may increase the risk of OHSS [96].

F. GENETIC COUNSELLING

De Braekeeler et al reported that there was one carrier of chromosomal aberrations in 4.7% cases of RPL and the most common being translocations (robertsonian or reciprocal).

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Genetic counseling is recommended in all the cases of RPL associated with parental chromosomal abnormalities. Treatment options are IVF in cases where chromosomes of couples are normal and donor gametes for homologous carriers of translocation. [24]

G. INFECTIONS

Kimura F et al reviewed the literature on the effect of chronic endometritis on reproduction. They concluded that the prevalence of chronic endometritis in RPL patients was 9.3-67.6 %. Treatment with antibiotics is reported to improve the reproductive outcome[97]. Mcqueen DB et al did a cohort study to find out the pregnancy outcome in women with chronic endometritis and RPL. They included 395 RPL women in their study. All women underwent endometrial biopsy and the prevalence of chronic endometritis was 9% in this cohort. The women with chronic endometritis received a course of antibiotics. The live birth rate improved from 7% to 56% after treatment [98].

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There is no role of TORCH screening in the evaluation of RPL. However, pregnant women infected with toxoplasmosis should be treated with spiramycin. It may reduce the neurological sequelae of congenital toxoplasmosis. Pyrimethamine and sulfadiazine can be given after 16th week of gestation. Data on safety and teratogenicity is limited about this combination.

MANAGEMENT OF UNEXPLAINED RPL

A. Progesterone supplementation

Stephenson MD et al did a prospective cohort study to know the effectiveness of luteal start vaginal micronized progesterone in RPL. They gave vaginal micronized progesterone at a dose of 100-200 mg 12th hourly 3 days after LH surge if nuclear cyclin E (>20%) in endometrial glandsor empirically despite normal nuclear cyclin E (≤ 20%). Live birth rate was higher in women who received progesterone as compared to controls (68% vs 51%) with an OR = 2.1 (95% CI , 1.0-4.4) [99].

Coomarasamy A et al did a randomized double-blind placebo controlled trial PROMISE : first trimester progesterone therapy in women with history of unexplained recurrent miscarriage involving 836 women randomized between progesterone (404) and placebo (432) groups. The patients received either 400 mg micronized progesterone or placebo vaginal capsules twice daily from the time of detection of pregnancy till 12 weeks period of gestation. They found that the live birth rate in progesterone group was 65.8% and in placebo group, it was 63.3%. They concluded that there is no role of first trimester progesterone therapy in unexplained RPL to improve the pregnancy outcome [100].

Cochrane analysis of 19 trials involving 2556 women showed that progesterone supplementation in women with unexplained RPL may reduce the rate of miscarriage in subsequent pregnancies. They found that supplementation with progesterone may reduce the rates of miscarriage in that pregnancy from 27.5% to 20.1% [101].

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B. Role of anticoagulants

ES Khan et al did a randomized controlled trial to know the preventive role of LMWH in unexplained RPL. They included 160 women in their study Eight women received inj enoxaparin 40 mg subcutaneous daily while the other group received placebo. They found no significant difference in the rates of live birth (78.8% vs 73.8%) [102].

Cochrane analysis reviewed 9 studies including data of 1228 women to evaluate the effect of either enoxaparin or aspirin or combination of both in unexplained recurrent miscarriage. Anticoagulants were found to be of no benefit in patients of unexplained RPL. The risk ratio for live birth in women who received aspirin as compared to placebo was found to 9.4 (95% CI, 0.8 TO 1.11, N=256). In women who received LMWH as compared to aspirin the risk ratio (RR) for live birth was 1.08 (95% CI 0.93-1.26, N=239) while RR was 1.01 in women who received LMWH and aspirin compared to no treatment (95% CI 0.87-1.16, N=322)
[103].

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B. TENDER LOVING CARE

Rao KA et al observed that tender loving care along with regular antenatal visit is one of the most efficient ways to achieve live term pregnancy in unexplained RPL. [104]

Tender loving care is defined as:

1. Optimal psychosocial support
2. Weekly medical examinations

3. Advice to rest as much as possible and to avoid heavy work and travelling
4. Coitus was not permitted
5. Bed rest was recommended for atleast 2 weeks gestational period in which the women had experienced their earlier miscarriages.

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MATERIALS AND METHODS

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This cross sectional analytical study was done in the Department of Obstetrics and Gynaecology, Jawaharlal Institute of Postgraduate medical education and research (JIPMER), Puducherry after the approval of research and ethics committee, (JIP/2017/IEC/0405). The study was undertaken between January 2018 and August 2019. The study comprised of two groups of patients.

Inclusion Criteria:

Group A- Pregnant Women admitted with first early pregnancy loss (Gestational age ≤14 weeks)

Non- pregnant Women attending OPD with history of one early Pregnancy loss and requesting investigations for pregnancy loss.

Group B- Women with two or more than two early pregnancy losses (RPL)

Exclusion criteria-.

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- Age <18 years and > 35 years
- Prior live birth
- Known cases of Hypertension, Diabetes mellitus, Hypothyroidism and auto immune disorders

Sampling

a. **Sampling population-**Married women in the age group (18-35 years) fulfilling the inclusion criteria who suffered pregnancy loss without any live child and were hospitalized for the same in JIPMER or attending OPD after having suffered pregnancy loss elsewhere were included for the study after Ethics committee approval.

b. **Sample size calculation**-The sample size was calculated using OpenEpi software version 3.0 using 95% confidence level and power of 80%. (As there were no prior studies)

We assumed that the difference in proportion of identifiable causes in two groups that is women with two or more than two pregnancy losses (RPL) and women with first early pregnancy loss to be 20%. The proportion of identifiable causes which is 50% among women in RPL [4], [25] the proportion of identifiable causes in group A is 30% the,sample size is 95 in each group and with 10% dropouts the final sample size is as follows. Group A-105 ; Group B-105

Sampling technique- Purposive Sampling technique

Study procedure- Women fulfilling the inclusion criteria were selected .Participants were explained about the protocol of the study and a written and informed consent was taken from each participant enrolled in the study

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Group A-Women with first early pregnancy loss

Group B-Women with two or more than two pregnancy losses(RPL)

Demographic data including age, occupation, education, socio-economic status was collected by interviewing the patient. Clinical profile including gravidity, parity, past obstetric history, family history, treatment history was documented on a proforma after interviewing the patient and from the medical records.A general physical examination was carried out and height, weight, BMI was measured. A complete systemic examination including thyroid, breast and Gynaecological examination was performed.

Parameters noted in this study are-

a. Age

b. BMI

c. Socioeconomic status

d. Number of pregnancy losses

e. Clinical assessment to find out the cause of pregnancy loss

f. All routine antenatal investigations

g. Urine culture

h. Cervical swab culture

i. Thyroid function test (TFT)

j. Oral glucose tolerance test (75g GTT)

k. **Ultrasonogram-**

Uterine anomalies

Fetus assessment

PCOS

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l. Hormonal profile for PCOS-(Non –Pregnant Status)

S.FSH, S.LH

LH/FSH ratio

Total testosterone, S.Prolactin

m. Thrombophilia profile:

APLA:

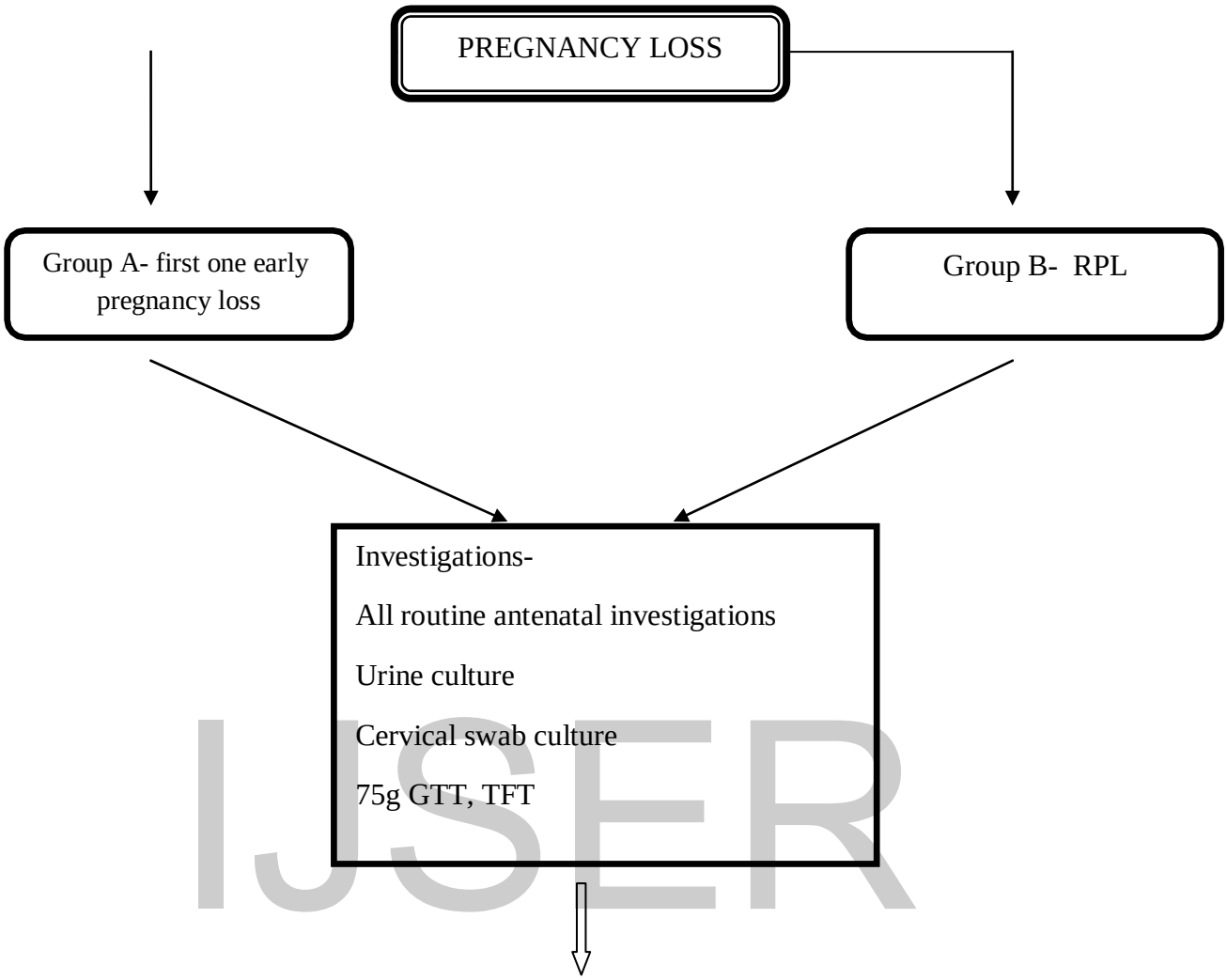
Lupus anticoagulant

beta 2 glycoprotein-1 antibody (IgM and IgG)

Anti Cardiolipin antibody (IgM and IgG)

Protein C and Protein S

RECRUITMENT



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NO ENDOCRINE, ANATOMICAL OR INFECTIOUS CAUSES (UNKNOWN CAUSE)

Thrombophilia profile (APLA; Protein c and Protein S)

STATISTICAL METHODS:

A. List of variables and their measurement methods with standardization techniques

a. **Independent variables**- Age, education, occupation, socioeconomic status, BMI.

b. **Outcome variables**- In each group

Primary outcome- Proportion of women with Identifiable causes for first early pregnancy loss

Secondary outcome –

1. Proportion of women with endocrine causes like diabetes mellitus, hypothyroidism and PCOS.
2. Proportion of women with thrombophilia.
3. Proportion of women with anatomical causes.
4. Proportion of women with infectious causes

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B. Confounding and interacting variables- nil

C. List variable wise statistical tests to be used for data analysis-

Data was collected and entered into statistical software SPSS version 15

Continuous variables like height, weight ,age, BMI, hormonal levels were expressed as mean (standard deviation) or median (Interquartile range) as per distribution of data and compared across two groups using **unpaired T-test** (normal/parametric distribution) or **Mann whitney test** (nonparametric distribution).

Catagorical variables (outcome) like proportion of women with endocrine causes and other non-endocrine causes were described as frequency and proportions and compared between groups by **chi square test.**

p value <0.05 was considered as significant

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RESULTS

The present study was undertaken in the department of Obstetrics and Gynecology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER) hospital between 2017 and 2019.One hundred and five patients were recruited in group A pregnant women admitted with first early pregnancy loss or non pregnant women attending OPD with history of one pregnancy loss and one hundred and five patients were recruited in group B with recurrent pregnancy loss. Four women in Group A (first pregnancy loss) and two women in group B (RPL) were in non pregnant state, rest of the women were pregnant. They were studied with respect to variables cited in the section on materials and methods. The various observations made are as follows.

Evaluation of women in Group A (First early pregnancy loss)

Table 8 : Socio-demographic and clinical profile of the subjects Group A

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S. No.	Parameter	Values N=105	Percentage (%)
1.	Mean age (years) ± SD	25.1 ± 4.26	-
2.	Mean BMI (kg/m^2) ± SD	22.74 ± 2.84	-
3.	BMI (kg/m^2) Underweight (<18.5) Normal weight(18.5-24.9) Pre-obesity (25-29.9) Obesity class I (30-34.9)	 4 77 23 1	 3.8 73.3 21.9 1
4.	Socio-economic status (Kuppuswamy classification) Class I Class II Class III Class IV	 - 3 29 73	 - 3.5 34.5 67
5.	Mean gestational age at pregnancy loss (weeks) ± SD	10.3 ± 1.9	-

The mean age of the women in group A was 25 years. The mean BMI was kg/m^2. Out of 105 women, 23 (21.9%) were overweight and only one was obese, while 77 (73.3%) of them were in the normal weight category. Sixty seven percent of the women belonged to class IV, while 29 (34.5%) were in class III kuppuswamy socioeconomic status classification. The mean gestational age at pregnancy loss was 10.3 weeks (Table 8).

Table 9: Age and first pregnancy loss

S.No.	Age (years)	Number of patients with pregnancy loss (percentage)
1.	<20	16 (15.23%)
2.	21-25	43 (40.95%)
3.	26-30	35 (33.33%)
4.	31-35	11 (10.4%)

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Table 9 shows the relationship of first pregnancy loss with age. About 15.23% subjects belonged to the teenage group (<20 years) and 40.95% of them were between the age group of 21-25 years. Thirty three percent were between the age group of 26-30 years. Only 10.4% of women were between 31-35 years.

Table 10: Causes of first pregnancy loss

S.No.	Cause	No. of patients (percentage)* N = 105	Percentage with respect to known causes and main etiology
1.	Unknown	44 (41.90%)	
2.	Known	61 (58.09%)	
A.	**Anatomical factors**	**5 (4.76%)**	**8.19%**
	Uterine anomaly	3 (2.9%)	60%
	Fibroid uterus	2 (1.9%)	40%
	Cervical incompetence	0	
B.	Fetal anomaly	0	
C.	**Endocrine**	**38 (36.19%)**	**62.29%**
	Hypothyroidism	9 (8.6%)	23.68%
	Type 2 DM	8 (7.6%)	21.05%
	PCOS	6 (5.7%)	15.78%
	GDM	15 (14.28%)	39.47%
D.	**Infections**	**2 (1.9%)**	**3.27%**
E.	**Combined etiology**	**16 (15.23%)**	**26.22%**
	GDM + Hypothyroidism	3 (2.9%)	18.75%
	GDM + Cervicovaginal infections	4 (3.80%)	25%
	Type 2 DM + Hypothyroidism	5 (4.8%)	31.25%
	Type 2 DM +PCOS	3 (2.9%)	18.75%
	Uterine anomalies + Hypothyroid	1 (1%)	6.25%

*percentage calculated out of total

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In 58.09% of the patients, the cause of first pregnancy loss could be identified which outnumbered patients with unknown causes (41.9%). The various causes amongst patients with known causes of first pregnancy loss were endocrine (36.19%), uterine factors (4.76%), infections (1.9%), combined etiology (15.23%).

The various endocrine causes diagnosed were GDM (14.28%), Type 2 DM (7.6%), Hypothyroidism (8.6%) and PCOS (5.7%). Out of PCOS women, only one woman was non pregnant and hormonal evaluation was normal for her. There were 3 (2.9%) cases of uterine anomalies out of which two patients had bicornuate uterus and one had uterine didelphys. Two women had submucosal fibroids which accounted for 1.9%. Two women had cervicovaginal infections with E.coli.

Combined etiologies were present among 16 (15.23%) women. Three women with GDM had hypothyroidism, while four of them with GDM had cervicovaginal infections. Two women had infection with E. Coli and two had with Klebsiella. Five women with type 2 DM also had hypothyroidism. Hypothyroidism was found in one woman with bicornuate uterus. Three of them had type 2 DM and PCOS. The causes of first pregnancy loss are shown in Table 10.

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Table 11 : Evaluation for thrombophilia in women with unknown causes in group A

S. No.	Thrombophilia evaluation	No. of cases (%)* N=44
1.	Thrombophilia negative	36 (81.81%)
2.	**Thrombophilia positive**	**8 (18.18%)**
A.	APLA positive	4 (9.09%)
	Primary	4
	Secondary	-
B.	Protein C deficiency	-
C.	Protein S deficiency	3 (6.81%)
D.	LAC positive + protein S deficiency	1 (2.27%)

*percentages calculated out of unknown causes

Thrombophilia evaluation was done in women with unknown causes in group A and it was identified as a cause in 8 (18.18%) patients. Three women had protein S deficiency, four were APLA positive while one woman was LAC positive and also had protein S deficiency. Out of the four APLA positive women, 2 were anticardiolipin antibody positive, one was lupus anticoagulant positive and one was positive for both LAC and ACLA.

In 81.81% of women with unknown causes in group A, causes could not be elicited, while 18.18% of them were positive for thrombophilias.

After thrombophilia evaluation for unknown causes, the percentage of women with known causes increased from 58.09% to 65.71%. (Table 11)

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Evaluation of women in Group B (RPL)

Table 12 : Socio-demographic and clinical profile of the subjects group B (RPL)

S. No.	Parameter	Values N=105	Percentage (%)
1.	Mean age (years) ± SD	25.9 ± 4.21	-
2.	Mean BMI (kg/m^2) ± SD	23 ± 3.16	-
3.	BMI (kg/m^2)		
	Underweight (<18.5)	4	3.8
	Normal weight(18.5-24.9)	75	71.4
	Pre-obesity (25-29.9)	22	21
	Obesity class I (30-34.9)	4	3.8
4.	Socio-economic status (Kuppuswamy classification)		
	Class I	-	-
	Class II	4	3.8
	Class III	40	38.1
	Class IV	61	58.1
5.	Mean gestational age at pregnancy loss (weeks)	10.23 ± 2.1	-
6.	Number of patients with		
	2 pregnancy losses	59	56.19%
	3 pregnancy losses	26	24.76%
	≥4 pregnancy losses	20	19.04%

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The mean age of the women in group B was 25.9 years. The mean BMI was 23 kg/m^2. Out of 105 women, 22 (21%) were overweight and 4 (3.8%) were obese, while 75 (71.4%) women were in the normal weight category. The mean gestational age at pregnancy loss was 10.2 weeks. Fifty eight percent of them belonged to class IV, while 40 (38.1%) were in class III kuppuswamy socioeconomic status classification. (Table 12)

Table 13 : Age and RPL

S.No.	Age (years)	Number of patients with pregnancy loss (percentage)
1.	<20	8 (7.62%)
2.	21-25	44 (41.9%)
3.	26-30	37 (35.23%)
4.	31-35	16 (15.23%)

Table 13 shows the relationship of recurrent pregnancy loss with age. About 7.62% of women belonged to the teenage group (<20 years) and 41.9% of them were between the age group of 21-25 years. Thirty five percent were between the age group of 26-30 years. Only 15.2% of women were between 31-35 years.

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Table 14 : Causes of RPL

S. No.	Cause	No. of women with RPL N=105 (percentage)*	Percentage with respect to known causes and main etiology
1.	Unknown	59 (56.19%)	
2.	Known	46 (43.80%)	
A.	**Anatomical factors**	**2 (1.90%)**	**4.34%**
	Uterine anomaly	1 (0.95%)	50%
	Fibroid	0	
	Cervical incompetence	1 (0.95%)	50%
B.	Fetal anomaly		
C.	**Endocrine**	**23 (21.90%)**	**50%**
	Hypothyroidism	5 (4.8%)	21.73%
	Type 2 DM	5 (4.8%)	21.73%
	PCOS	1 (0.95%)	4.34%
	GDM	12 (11.42%)	52.17%
D.	**Infections**	**1 (0.95%)**	**2.17%**
E.	**Combined etiology**	**20 (19.04%)**	**43.47%**
	GDM + PCOS	1 (0.95%)	5%
	GDM + Hypothyroidism	7 (6.7%)	35%
	GDM + Cervicovaginal infections	3 (2.9%)	15%
	GDM + PCOS + Hypothyroidism	1 (0.95%)	5%
	Type 2 DM + Hypothyroidism	3(2.9%)	15%
	Uterine anomalies + Hypothyroid	1 (0.95%)	5%
	Uterine anomalies +PCOS	1 (0.95%)	5%
	Cervical incompetence + GDM	1 (0.95%)	5%
	Cervical incompetence + hypothyroid + PCOS	1 (0.95%)	5%
	LAC + Hypothyroid	1 (0.95%)	5%

*percentage calculated out of total

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In RPL group, in 56% women causes were unknown and after evaluation (except for thrombophilia), we could identify the causes in 43% women.

The various causes amongst women with known causes of RPL were endocrine (21.9%), uterine factors (1.9%), infections (0.95%), combined etiology (19.04%). The various endocrine causes diagnosed were GDM (11.42%), Type 2 DM (4.8%), Hypothyroidism (4.8%) and PCOS (0.95%). Hormonal profile for two PCOS women in non pregnant state was done, out of which one woman had increased serum testosterone. There was one case of septate uterus and one case of cervical incompetence. Only one woman had cervicovaginal infection with Klebsiella.

Combined etiologies were present among 20 (19.04%) women. Seven women with GDM had hypothyroidism, while 3 with type 2 DM also had hypothyroidism. Hypothyroidism was found in one woman with bicornuate uterus and three of them with GDM had cervicovaginal infections. Two women had infection with E. Coli and one had with Klebsiella One woman with PCOS was found to have GDM and one had GDM, PCOS and hypothyroidism. One woman bicornuate uterus had PCOS. One woman was found to have LAC positive and hypothyroidism. Two of them with cervical incompetence also had other etiologies like GDM, PCOS and hypothyroidism. The causes of recurrent pregnancy loss are shown in Table 14.

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Table 15 : Evaluation of thrombophilia in unknown causes of group B

S. No.	Thrombophilia evaluation	No. of cases (%)* N=59
1.	Thrombophilia negative	48 (81.35%)
2.	**Thrombophilia positive**	**11 (18.64%)**
A.	APLA positive	6 (10.16%)
	Primary	5
	Secondary	1
B.	Protein C deficiency	0
C.	Protein S deficiency	4 (6.78%)
D.	ACLA positive + protein S deficiency	1 (1.69%)

*percentages calculated out of unknown causes

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Thrombophilia evaluation was done in women with unknown causes in group B and it was identified as a cause in 11 (18.64%) women. Four women had protein S deficiency, 6 were APLA positive while one woman was ACLA positive and also had protein S deficiency. Out of the six APLA positive women, two patients were anticardiolipin antibody positive, three were lupus anticoagulant positive and one woman was positive for both LAC and ACLA. Five women had primary APLA syndrome while one of them had SLE. (Table 15)

In 81.35% of women with unknown causes in group A, causes could not be elicited, while 18.64% of them were positive for thrombophilias.

After thrombophilia evaluation for unknown causes, the percentage of women with known causes increased from 43.8% to 54.28%.

Comparison of Group A and Group B

Table 16: Comparison of Socio-demographic and clinical profile of the patients between group A and B

S. No.	Parameter	Group A Values/n	Group B Values/n	p value
1.	Mean age (years) ± SD	25.1 ± 4.26	25.9 ± 4.21	0.17
2.	Mean BMI (kg/m^2) ± SD	22.74 ± 2.84	23 ± 3.16	0.68
3.	BMI (kg/m^2) Underweight (<18.5) Normal weight(18.5-24.9) Pre-obesity (25-29.9) Obesity class I (30-34.9)	 4 (3.8%) 77 (73.3%) 23 (21.9%) 1 (1%)	 4 (3.8%) 75 (71.4%) 22 (21%) 4 (3.8%)	0.584
4.	Socio-economic status (Kuppuswamy classification) Class I Class II Class III Class IV	 - 3 (3.5%) 29 (34.5%) 73 (67%)	 - 4 (3.8%) 40 (38.1%) 61 (58.1%)	0.226
5.	Mean gestational age at pregnancy loss (weeks) ± SD	10.3 ± 1.9	10.23 ± 2.1	0.49

p value was calculated using independent student t test for age, BMI and gestational age and chi-square test for BMI classification and socioeconomic status

There was no statistically significant difference of age, BMI, socioeconomic status and gestational age at pregnancy loss between women in both the groups. (Table 16)

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Table 17 : Comparison of causes of first pregnancy loss with that of recurrent pregnancy loss

S. No.	Cause	No. of patients with one pregnancy loss (percentage)	No. of patients with recurrent pregnancy loss (percentage)	p value
1.	Unknown	44 (41.90%)	59 (56.19%)	**0.038**
2.	Known	61 (58.09%)	46 (43.80%)	
A.	**Anatomical factors**	**5 (4.76%)**	**2 (1.90%)**	0.249
	Uterine anomaly	3 (2.9%)	1 (0.95%)	
	Fibroid	2 (1.9%)	0	
	Cervical incompetence	0	1 (0.95%)	
B.	Fetal anomaly	0		
C.	**Endocrine**	**38 (36.19%)**	**23 (21.90%)**	**0.023**
	Hypothyroidism	9 (8.6%)	5 (4.8%)	
	Type 2 DM	8 (7.6%)	5 (4.8%)	
	PCOS	6 (5.7%)	1 (0.95%)	
	GDM	15 (14.28%)	12 (11.42%)	
D.	**Infections**	**2 (1.9%)**	**1 (0.95%)**	0.48
E.	**Combined etiology**	**16 (15.23%)**	**20 (19.04%)**	0.464
	GDM + PCOS	0	1 (%)	
	GDM + Hypothyroidism	3 (2.9%)	7 (6.7%)	
	GDM + PCOS + Hypothyroidism	0	1 (0.95%)	
	GDM + Cervicovaginal infections	4 (3.80%)	3 (2.9%)	
	Type 2 DM + Hypothyroidism	5 (4.8%)	3(2.9%)	
	Type 2 DM +PCOS	3 (2.9%)	0	
	Uterine anomalies + Hypothyroid	1 (1%)	1 (0.95%)	
	Uterine anomalies +PCOS	0	1 (0.95%)	
	Cervical incompetence + GDM	0	1 (0.95%)	
	Cervical incompetence + hypothyroid + PCOS	0	1 (0.95%)	
	LAC + Hypothyroid	0	1 (0.95%)	

p value was calculated using chi square test for known, endocrine, infections and combined causes and fischer exact test for anatomical causes

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Table 17 shows the comparison of causes of first pregnancy loss with that of RPL. The proportion of known causes in group A women with single pregnancy loss was 58% as compared to 43% in group B RPL women and the difference was statistically significant. Endocrine causes were the commonest in both the groups and the proportion of endocrine causes in first pregnancy loss (36%) was significantly more than RPL group (21%) with p=0.023. Combined etiology was the second commonest (group A 15.23% vs group B 19% ; p=0.46). The percentage of anatomical, infectious and combined causes were similar between both the groups.

Table 18 : Comparison of thrombophilia evaluation in group A and B

S. No.	Thrombophilia evaluation	Group A n(%) N=44	Group B n(%) N= 59	p value
1.	Thrombophilia negative	36 (81.81%)	48 (81.35%)	
2.	**Thrombophilia positive**	**8 (18.18%)**	**11 (18.64%)**	0.47
A.	APLA positive	4 (50%)	6 (54.5%)	
	Primary	4	5	
	Secondary	-	1	
B.	Protein C deficiency	-	0	
C.	Protein S deficiency	3 (37.5%)	4 (36.3%)	
D.	APLA positive + protein S deficiency	1 (12.5%)	1 (9.09%)	

p value was calculated using chi-square test

Thrombophilia evaluation was done for unknown causes in both the group. Eighteen percent of women in each group were positive for thrombophilia with p value of 0.47. Thus, the proportion of thrombophilia positive women in both the groups were similar (Table 18).

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Table 19: Comparison of proportion of known and unknown causes with or without thrombophilia evaluation in Group A and B

Parameter	**Group A ; n (%) N=105**	**Group B; n (%) N=105**	**p value**
Without thrombophilia evaluation Known Unknown	61 (58.09%) 44 (41.90%)	46 (43.80%) 59 (56.19%)	0.038
With thrombophilia evaluation Known Unknown	69 (65.17%) 36 (34.28%)	57 (54.28%) 48 (45.71%)	0.09

p value was calculated using chi-square test

The proportion of known causes in women with first early pregnancy loss was 58% as compared to 43% in RPL women and the difference was statistically significant (p=0.038). Thrombophilia evaluation was done for unknown causes in both the groups. After thrombophilia evaluation, the percentage of known causes in women with first pregnancy loss increased from 58% to 65% and 43% to 54% in RPL group. The proportion of identifiable causes in both the groups were similar after thrombophilia evaluation (group A 65% vs group B 54% ; p=0.09) (Table 19)

Table 20: Age and Thrombophilia positivity

Age group	**Thrombophilia positive N=19 (%)**
<30 years	18 (94.4%)
≥30 years	1 (5.55%)

Ninety four percent of thrombophilia positive women were young (<30 years), only one woman was >30 years old. (Table 20)

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Subgroup analysis of thrombophilia

Table 21 : Thrombophilia screening in group A and B

S. No.	Investigation done for thrombophilia	Group A		Group B		p value
		Number screened	Number with abnormal results (percentage)	Number screened	Number with abnormal results (percentage)	
1.	Beta 2 glycoprotein antibody	44	0	66	1 (1.51%)	1.00
2.	Anti-cardiolipin antibody (ACLA)	44	3 (6.81%)	66	4 (6.06%)	1.00
3.	Lupus Anticoagulant	44	3 (6.81%)	66	6 (9.09%)	0.736
4.	Protein C Deficiency	23	0	27	0	-
5.	Protein S Deficiency	23	4 (17.39%)	27	5 (18.51%)	1.00

APLA was done for all the women with unknown causes in both the groups. Some women who were found to have either of the endocrine, anatomical or combined etiology in RPL group also had done APLA. Congenital thrombophilia screening being costly and because of limited funds available was done for 23 women with first pregnancy loss and 27 women with RPL. (Table 21)

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Table 22: Summary table

S. No.	Characteristic	Group A (First early pregnancy loss) N=105 (value/n and percentage)	Group B (RPL) N=105 (value/n and percentage)	p value
1.	Mean age (years) ± SD	25.1 ± 4.26	25.9 ± 4.21	0.17
2.	Mean BMI (kg/m^2) ± SD	22.74 ± 2.84	23 ± 3.16	0.68
3.	Mean gestational age at pregnancy loss (weeks) ± SD	10.3 ± 1.9	10.23 ± 2.1	0.49
4.	**Known causes**	**61 (58.09%)**	**46 (43.80%)**	**0.038**
	Endocrine factors	38 (62.29%)*	23 (50%)*	0.023
	Combined factors	16 (26.22%)	20 (43.47%)	0.464
	Anatomical factors	5 (8.19%)	2 (4.34%)	0.249
	Infections	2 (3.27%)	1 (2.17%)	0.48
5.	**Unknown**	**44 (41.90%)**	**59 (56.19%)**	**0.038**
	Thrombophilia negative	36 (81.81%)**	48 (81.35%)**	0.47
	Thrombophilia positive	**8 (18.18%)**	**11 (18.64%)**	**0.47**
	APLA positive	4	6	
	Protein S deficiency	3	4	
	APLA positive + protein S deficiency	1	1	
6.	**Without thrombophilia evaluation** Known causes	61 (58.09%)	46 (43.80%)	0.038
	Unknown causes	44 (41.90%)	59 (56.19%)	
7.	**With thrombophilia evaluation** Known causes	69 (65.17%)	57 (54.28%)	0.09
	Unknown causes	36 (34.28%)	48 (45.71%)	

*percentages calculated out of known causes

** percentages calculated out of unknown causes

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DISCUSSION

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The present study was a descriptive analytical study to know the etiology of first early pregnancy loss and to compare the proportion of identifiable causes between first early pregnancy loss and recurrent pregnancy loss. The study included 105 women in group A (first early pregnancy loss) and another 105 in group B (RPL).

We found that the proportion of identifiable causes in first early pregnancy loss was similar to that of RPL.

There are no studies in the literature for the evaluation of first early pregnancy loss. In the present study, about 40% of pregnancy loss both in first pregnancy loss and RPL group was found to be among the age group of 21-25 years (Table 23). Previous study by Nybo Anderson showed that as the age increased, the percentage of RPL increased. We did not find a similar trend in the present study. The incidence of RPL in their study in the age group of 40-44 years was 51% as compared to 11% in 21-25 years. We did not recruit women >35 years in our study. The most common age group of antenatal women in our population is 21-25 years, that might be the reason we found the maximum incidence of RPL in this age group.

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Table 23: Age and pregnancy loss

S. No.	Study	Age group (years)	Percentage of pregnancy loss in women with one pregnancy loss	Percentage of RPL
1.	Present study	<20	15.23%	7.62%
		21-25	**40.95%**	**41.9%**
		26-30	33.33%	35.23%
		31-35	10.4%	15.23%
		>35	-	-
2.	Nybo Anderson et al [68](2000)	20-24	-	11%
		25-29	-	12%
		30-34	-	15%
		35-39	-	25%
		40-44	-	51%

Table 24 : BMI and pregnancy loss

S. No.	Study	BMI (kg/m^2)	Percentage of pregnancy loss in RPL group	Percentage of pregnancy loss in first pregnancy loss group
1.	Bhandari et al [71](2016)	Underweight (<18.5)	-	-
		Normal weight (18.5-24.9)	48.6%	-
		Pre-obesity (25-29.9)	31.6%	-
		Obesity class I (>30)	19.8%	-
2.	Matjila et al[105] (2017)	Underweight (<18.5)	2.4%	-
		Normal weight (18.5-24.9)	24.3%	-
		Pre-obesity (25-29.9)	30.7%	-
		Obesity class I (>30)	42.6%	-
3.	MB Cavalcante et al[70] (2019)	Underweight (<18.5)	1.4%	-
		Normal weight (18.5-24.9)	47.1%	-
		Pre-obesity (25-29.9)	29%	-
		Obesity class I (>30)	22.5%	-
4.	Present study	Underweight (<18.5)	3.8%	3.8%
		Normal weight(18.5-24.9)	71.4%	73.3%
		Pre-obesity (25-29.9)	21%	21.9%
		Obesity class I (30-34.9)	3.8%	1%

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Bhandari et al[71] in their study on obese women with RPL found that majority of women (48.6%) had normal weight, 31 % were pre-obese and 19% were obese. Matjila et al in their study on medical conditions in RPL found in their study that majority of the women were obese (42%).

MB Cavalcante et al[70] performed a meta- analysis on obesity and recurrent miscarriage. Forty seven percent women with RPL were in normal weight category, while 29% of them were pre-obese and 22% were class I obese. In our study also, similar to Bhandari et al and meta-analysis by Cavalcante et al , majority of women had normal weight (71%) and 21% women were pre-obese, which was comparable to previous studies, but only 3.8% women were obese, which was less as compared to previous studies (Table 24). The difference in findings may be due to different population. Bhandari et al performed their study in UK and Matjila et al on south African women. The incidence of obesity as such is in India is less as compared to west. There are no studies so far in literature for first early pregnancy loss.

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Table 25 : Etiology of RPL

S. No.	Causes	Author	Percentage contribution to RPL	Percentage contribution to RPL (present study)
1.	**Endocrine**	Singh A et al[106] (2017)	**20%**	**21.9%**
		Shetty MB et al[107] (2017)	**38.8%**	
	DM	Shetty MB et al[107] (2017)	26%	16.2%
	Hypothyroidism	Lee GS et al[108] (2016)	9%	4.8%
		Shetty MB et al[107] (2017)	12.8%	
	PCOS	Li TC et al[22] (2000)	7.8%	0.95%
		Cocksedge et al[109] (2009)	4.8-81%	
2.	Anatomical factors	Lee GS et al[108] (2016)	13.5%	1.9%
	Uterine anomaly	Salim R et al[23] (2003)	5.3%	0.95%
3.	Infections	Ford HB et al[110] (2009)	0.5-5%	0.95%
4.	Combined etiology	Lee GS et al[108] (2016)	48.3%	19.04%
5.	Unknown	El Hachem et al[25] (2017)	50%	56.19%

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Based on previous studies, endocrine causes were the commonest among known causes of RPL. DM was found in 26% women, hypothyroidism in 9-12% and PCOS in 7.8% women with RPL. In the present study also, we found that endocrine causes (21.9%) were commonest among RPL women which was comparable to Singh A et al. DM, hypothyroidism and PCOS comprised 16.2%, 4.8% and 0.95% respectively in women with RPL in our study. The prevalence of hypothyroidism and diabetes was found to be higher in previous studies than the present study. The incidence of PCOS in RPL women was found to 0.95% in our study. PCOS in RPL varies widely between 4.8-80% as described in literature, so more studies are required to come to a consensus.

Salim R et al[23] found uterine anomalies in 5% of women with RPL, whereas in our study it was only 0.95%. Infections as an etiological factor was found to be less (0.95%), which was comparable to previous studies in the literature. In the present study, 19% women had combined etiology and only one study in the literature by Lee GS et al has reported combined etiology (48%) contributing for RPL, but the authors did not clarify causes included in the combined etiology.

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In the present study, 19% women had combined etiology and only one study in the literature has reported combined etiology (48%) contributing for RPL.

Similar to the previous studies, in 56% women with RPL, the cause of RPL was unknown (Table 25).

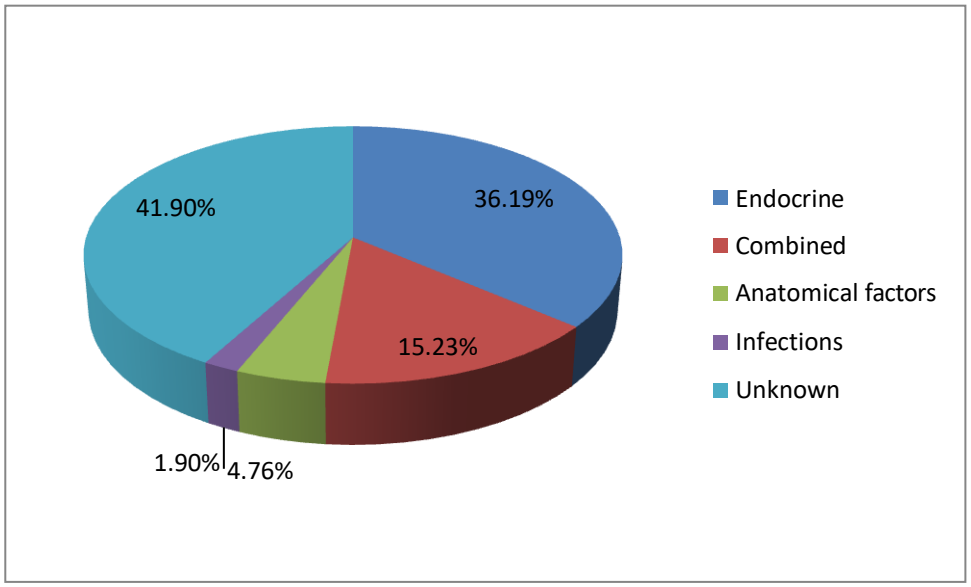

Fig 1 : Etiology of first early pregnancy loss

There are no studies to find out the etiology of first early pregnancy loss. The various etiological factors found in present study for first early pregnancy loss are shown in figure 1. Endocrine causes were significantly higher in first pregnancy loss than RPL. The proportion of other causes were similar to recurrent pregnancy loss. We found that proportion of identifiable causes in first early pregnancy loss were **more than that of RPL** which was an unanticipated finding as there are no studies / recommendations for evaluation of first pregnancy loss in the literature so far.

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Table 26 : Thrombophilia evaluation for *unknown causes in RPL*

S.No.	Study	Thrombophilia	Percentage of RPL women positive (%)
1.	Vora S et al[4] (2008)	**Acquired**	46%
		Inherited	37%
2.	Patil R et al[26] (2015)	**Acquired**	24%
		Inherited	16%
3.	Present study (2019)	**Acquired**	10.1%
		Inherited	6.7%
		Combined	1.69%

Previous study by Vora S et al (2008) showed that in women with unknown causes of RPL,75% were thrombophilia positive. Forty six percent were positive for acquired thrombophilia and 37% were positive for congenital thrombophilia. They screened for lupus anticoagulant, anticardiolipin antibodies, ß2 glycoprotein 1 antibody, annexin V, protein C, protein S, antithrombin III, factor V leiden, PT gene G20210A, MTHFR C677T, EPCR 23 bp insertion and PAI 4G/3G polymorphisms. Previous study by Patil R et al (2015) in women with unexplained RPL showed that 40% of RPL women were positive for thrombophilias.

In the present study we found that only 18% of RPL women were positive for thrombophilias. Acquired thrombophilia constituted 10% and congenital thrombophiia constituted 6.7%, while one women had both congenital and acquired thrombophilia (Table 26). The difference in the results might be because we screened only for APLA, protein C and protein S as compared to previous studies which screened for more causes of congenital thrombophilias thus explaining the incidence of thrombophilia being less in our study.

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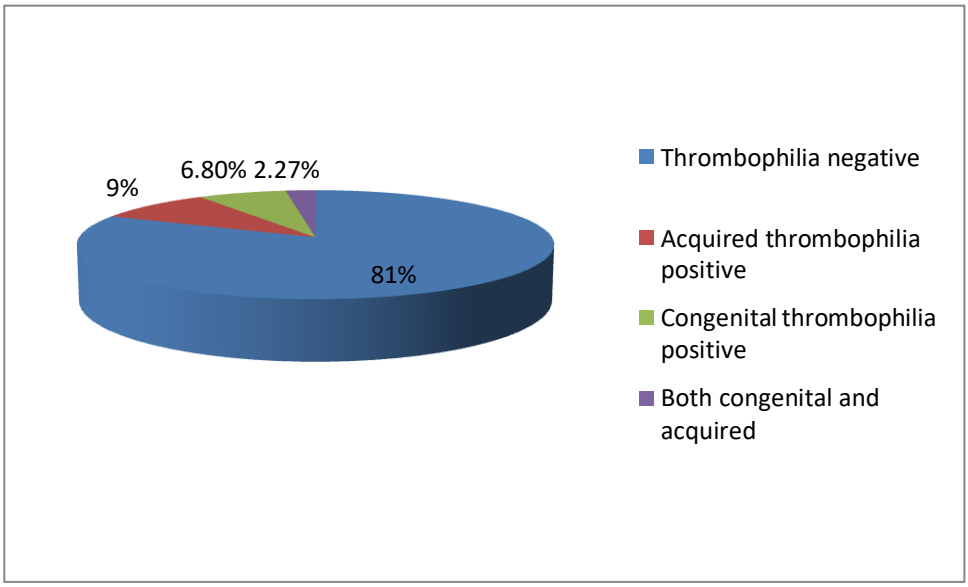

Fig 2 : Thrombophilia evaluation for **unknown causes in first early pregnancy loss**

Figure 2 shows the thrombophilia evaluation in women with unknown causes of first early pregnancy loss. The proportion of women positive for thrombophilia were comparable to RPL group. There are no previous studies in literature for thrombophilia evaluation after one miscarriage.

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Table 27 : Recommendations for thrombophilia screening in RPL

Name of the body	**Recommendation**
ESHRE[11] (2018)	Can be considered
ASRM[111] (2012)	Recommended
RCOG[12] (2011)	Recommended

Table 27 outlines the various recommendations for thrombophilia screening in RPL.

We should start investigating after first pregnancy loss, though more studies are required for evaluation of single pregnancy loss. Thrombophilia screening to be undertaken if there are no endocrine, anatomical or infectious causes.

SUMMARY

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First early pregnancy loss

1. The mean of age women with first early pregnancy loss was 25 years and mean BMI was 22 kg/m^2.
2. Seventy three percent of women had normal weight, 21% were pre-obese and only one woman was obese (class I).
3. Majority of patients belonged to class III and IV kuppuswamy socioeconomic status classification (34% and 67% respectively).
4. The mean gestational age at pregnancy loss was 10 weeks.
5. In 34%, the cause of first pregnancy loss was unknown.
6. In 65%, the various causes of first pregnancy loss was found. Endocrine causes (36%) were the commonest followed by combined etiology (15%).

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Recurrent pregnancy loss (RPL)

1. The mean of age women with recurrent early pregnancy loss was 25 years and mean BMI was 23 kg/m^2.
2. Seventy one percent of women had normal weight, 21% were pre-obese and four women were obese (class I).
3. Majority of patients belonged to class III and IV kuppuswamy socioeconomic status classification (38% and 58% respectively).
4. The mean gestational age at pregnancy loss was 10 weeks.
5. In 45%, the cause of recurrent pregnancy loss was unknown.
6. In 54%, the various causes of recurrent pregnancy loss was found. Endocrine causes (21%) were the commonest followed by combined etiology (19%) .

Comparison between first early pregnancy loss and RPL

1. The Age, BMI, socioeconomic status and gestational age at pregnancy loss were similar between women in both the groups.
2. The known causes in women with first early pregnancy loss were significantly more than women with RPL (65% vs 54%; p=0.038)
3. Out of known causes, endocrine causes were commonest in both the groups (First pregnancy loss 36% vs RPL 21%; p=0.023)
4. The proportions of anatomical factors, infections and combined etiology were similar in both the groups.

Thrombophilia

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1. Thrombophilia evaluation was done for women with unknown causes in both the groups.
2. Out of unknown causes, 18% women were positive for thrombophilia in each group.
3. In thrombophilia positive women (first early pregnancy loss), 50% were APLA positive, 37.5% had protein S deficiency, while one woman was both APLA positive and had protein S deficiency.
4. In thrombophilia positive women (recurrent pregnancy loss), 54% were APLA positive, 36.36% had protein S deficiency, while one woman was both APLA positive and had protein S deficiency.

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LIMITATIONS

LIMITATIONS

1. Thrombophilia evaluation was done only in women with unknown causes in both the groups.

2. Congenital thrombophilia screening could not be done for all women with unknown causes because of high cost and limited funds.

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CONCLUSION AND RECOMMENDATION

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CONCLUSION

- *Significant proportion of women (65%) with first early pregnancy loss had various etiological factors and endocrine factors were the most common cause.*
- *Among the identifiable causes for first early pregnancy loss anatomical factors were found in 4.76%, endocrine in 36%, thrombophilia in 18% and combined etiology in 15%.*
- *Statistically significantly more women with first pregnancy loss were found to have known etiological factors when compared to women with recurrent pregnancy loss and endocrine causes were the most common. The thrombophilia positivity was found to be similar in both the groups.*

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RECOMMENDATION

- *Based on the findings of this study, it is recommended that **evaluation is essential** for women with **first early pregnancy loss** so that pregnancy loss can be prevented during the next pregnancy and optimum pregnancy outcome can be achieved .*
- *Thrombophilia screening may be undertaken in women with unknown cause for first early pregnancy loss. Screening to be undertaken for acquired thrombophilias.*
- *More studies are required to be undertaken for evaluation of first early pregnancy loss.*

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ANNEXURES

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जवाहरलाल स्नातकोत्तर आयुर्विज्ञान शिक्षा एवं अनुसंधान संस्थान
JAWAHARLAL INSTITUTE OF POSTGRADUATE MEDICAL EDUCATION & RESEARCH
(स्वास्थ्य एवं परिवार कल्याण मंत्रालय, भारत सरकार के अधीन राष्ट्रीय महत्व का संस्थान)
भारत सरकार / GOVERNMENT OF INDIA
(An institution of National Importance under Ministry of Health & Family Welfare)
धनवंतरी नगर , पुडुचेरी / Dhanvantari Nagar, Puducherry – 605006
Website: www.jipmer.edu.in
सत्यमेव जयते
Phone: 0413 – 2296101
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DEPARTMENTAL PG DISSERTATION SCREENING COMMITTEE

CERTIFICATE

No. 6 Date: 21/10/2017

This is to certify that the PG dissertation proposal with the following details has been approved by the Departmental PG Dissertation Committee, held on 17th October, 2017 subject to clearance by the Institute Ethics Committee.

Title: Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss

Name of the postgraduate: Dr. Sonal Garg

Guide: Prof. Papa Dasari
Prof. and Head,
Department of Obstetrics and Gynaecology
JIPMER, Puducherry

Co-guides: Dr. T Chitra
Associate Professor
Department of Obstetrics and Gynaecology
JIPMER, Puducherry

Dr. Rakhee Kar
Additional Professor
Department of Pathology
JIPMER, Puducherry

HEAD OF THE DEPARTMENT
Professor & Head,
Dept. of Obst. & Gynae.,
JIPMER, Pondicherry-6

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जवाहरलाल स्नातकोत्तर आयुर्विज्ञान शिक्षा एवं अनुसंधान संस्थान
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सत्यमेव जयते
Phone: 0413 – 2296101
Fax: 0413 – 2272067, 2272735

INSTITUTIONAL ETHICS COMMITTEE (HUMAN STUDIES)

CERTIFICATE

Date: 20/03/2018

To,

Dr. Sonal Garg, Junior Resident,
Dept. of Obstetrics and Gynaecology,

Ref: Your project no. **JIP/IEC/2017/0405** entitled, **"Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss."**

Dear Dr. Sonal Garg,

The following documents of the above mentioned project were reviewed and approved through an **Expedited review** process.

1. Research Protocol
2. Patient Information Sheets
3. Consent forms
4. Data Collection Proformas
5. CV of Guide and Co-Guides

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It is understood that the study will be conducted in the direction of Dr. Dasari Paapa, Professor & Head, Dept. of Obstetrics and Gynaecology (Guide) ,Dr. Chitra T, Associate Professor, Dept. of Obstetrics and Gynaecology and Dr. Rakhee Kar, Additional Professor, Dept. of Pathology (Co-Guides), in a total of **210** research participants, as per the submitted protocol.

The IEC approves the above mentioned study.

This approval is valid for three years, the entire duration of the project or a shorter period based on the risk whichever is less.

It is the policy of IEC that, it be informed about any onsite serious adverse event or any unexpected adverse event report within 24 hours as per the formats specified in SOP 09 to IEC or by email if there is holiday. The report of SAE or death after due analysis shall be forwarded by the Investigator to the chairman of IEC and the head of the institution where the trial is been conducted within 10 calendar days of SAE or death.

In case of injury or death of participant(s) occurring during the trial, the sponsor (whether a pharmaceutical company or an institution) or his representative, whosoever had obtained permission from the Licensing Authority for conduct of the clinical trial shall make payments for medical management of the subject and also provide financial compensation for the clinical trial related injury or death.

No deviations from, or changes of the protocol and Informed Consent Document should be initiated without prior written approval by the IEC of an appropriate amendment. The IEC expects that the investigator should promptly report to the IEC any deviations from, or changes of, the protocol to eliminate immediate hazards to the research participants and about any new information that may affect adversely the safety of the research participants or the conduct of the trial.

For studies which will continue for more than a year, a continuing review report needs to be submitted (within 1 month of the due date i.e. 11 months from the date of approval) on or before 11/02/2019.

A copy of the final report should be submitted to IEC for review.

Sincerely yours

Medha R.

Dr. Medha R,
Member Secretary
Date of approval of the study: 12/03/2018

MEMBER SECRETARY
INSTITUTE ETHICS COMMITTEE
(HUMAN STUDIES) JIPMER, PUDUCHERRY

IJSER

Copy to:

Principal Investigator: Dr. Sonal Garg, Junior Resident, Obstetrics and Gynaecology
Guide: Dr. Dasari Paapa, Professor & Head, Dept. of Obstetrics and Gynaecology
Co-Guides: Dr. Chitra T, Associate Professor, Dept. of Obstetrics and Gynaecology
Dr. Rakhee Kar, Additional Professor, Dept. of Pathology

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(Institution of National Importance under Ministry of Health & Family Welfare, Government of India)
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PLAGIARISM CHECKING COMMITTEE (PCC)

CERTIFICATE

This is to certify that the manuscript submitted by Dr. Sonal Garg, Department of Obstetrics and Gynaecology, with the following details has been processed using iThenticate Software and it is acceptable for submission as a thesis / dissertation.

IJSER

Reference No: 1702

Title: Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss.

Course & Year: MS Obstetrics and Gynaecology; 2020

Guide: Dr. Dasari Paapa

Dr. Noyal Mariya Joseph
MEMBER SECRETARY
PLAGIARISM CHECKING COMMITTEE
JIPMER

Date: 16.01.2020

To
1. Candidate: Dr. Sonal Garg
2. Guide: Dr. Dasari Paapa, for information

INFORMED CONSENT DOCUMENT (ICD)

Patient / Participant information sheet

INFORMATION FOR PARTICIPANTS OF THE STUDY-GROUP A

- **Title of the project**

Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss

- **Name of the investigator/guide**

Dr Sonal Garg, JR OG; Dr. Dasari Paapa, Professor and Head, Dept. of Obstetrics and Gynaecology,JIPMER ; Dr. Chitra T, Associate Professor, Dept. of Obstetrics and Gynaecology, JIPMER : Dr Rakhee Kar, Associate Professor, Department of clinical Haematology,JIPMER,Puducherry.

- **Purpose of this project/study**

We are conducting this study to find out why women suffer from pregnancy loss for the first time. So far in clinical practice ,no investigations are recommended when a woman suffers from abortion for the first time. But we encounter many women asking explanation why they had an abortion. We expect that if we start investigating awomen after first pregnancy loss we can identify the causes in about 30%. This will help to reduce your anxiety and we can treat you earlier for the better outcome of next pregnancy.

IJSER

- **Procedure/methods of the study including withdrawal criteria-**

You will be asked questions regarding your age, socioeconomic status, present condition, nutritional history, menstrual history family history and past obstetric/medical/surgical history. You also need to tell us regarding the process of your early first pregnancy loss and the way it was managed including investigations done and treatment given either by medical methods or surgical methods. We will take your height and weight. We will perform a general Physical examination and systemic examination including internal genital examination.Your blood samples will be taken through venous puncture. This will be used to find out hemoglobin and other routine tests done during every pregnancy including HIV. Thyroid hormone level, and other hormones like FSH, LH Prolactin and serum testosterone will be estimated if

you are not Pregnant presently.Your fasting blood sample will be taken for glucose estimation and you will be asked to drink 200ml of water mixed with glucose and blood sample will be taken twice every hour to find whether you are suffering from Diabetes. Urine sample and cervical swab will be taken to identify infectious causes. Ultrasonographic examination will be done to find out any abnormalities in your uterus and ovaries and to assess your fetus. If no causes are identified by these investigations we will investigate you further by taking blood samples for rare causes like blood clotting disorders .You will be intimated about the results of the tests and explained regarding the possible cause of your pregnancy loss. And appropriate management would be offered.

- **Expected duration of the subject participation-** Once for examination and taking blood samples and next time for review of reports and follow up and management if necessary.

The benefits to be expected from the research to the participant or to others and the post trial responsibilities of the investigator-

Causes of pregnancy loss would be identified early and treated pre- conceptionally before the next pregnancy. Medical (endocrine and immunological) causes would be optimally treated and hence better pregnancy outcome would be achieved later.

IJSER

- **Any risks expected from the study to the participant**- You will experience pain associated with blood sampling,in very rare cases there might be haematoma formation. In addition to this, you might feel uncomfortalble as per vaginum examination will be performed.

- **Maintenance of confidentiality of records**- Confidentiality of your identity and details will be maintained during the study and also afterwards data will be used for research publication without revealing your identity.

- **Provision of free treatment for research related injury-** We do not anticipate any research related injury as such. If there is some foreseen injury, you will be given compensation as per JIPMER guidelines and free treatment will be provided.You are free to withdraw anytime from the study.

- **Reimbursement for participating in the study**- No compensation will be given for participation in this study.

- **Compensation to the participants for foreseeable risks and unforeseeable risks related to research study leading to disability or death**- No foreseeable risks. For unforseeable risks related to research you will be provided compensation as per JIPMER guidelines.

- **Freedom to withdraw from the study at any time during the study period without the loss of benefits that the participant would otherwise be entitled**- Participation in this study is voluntary. You have the right to withdraw from the study at any point of time without loosing your benefits.

- **Possible current and future uses of the biological material to be generated from the research and if the material is likely to be used for secondary purposes or would be shared with others, this should be mentioned**- The biological material will be used till the tests are done for this study and it will not be used for future purposes.

- **Possible current and future uses of the data to be generated from the research and if the data is likely to be used for secondary purposes or would be shared with others, this should be mentioned**- The data generated from this study may be used for scientific presentation and publication, however your personal information and identity will not be revealed.

IJSER

- **Address and mobile number of the Principal investigator (PI) and Co- PI, if any :**

Dr. Sonal Garg, Junior Resident, Department of Obstetrics and Gynaecology, 7598907264

Dr.Dasari Paapa, Professor and Head, Department of Obstetrics and Gynaecology, 9442566883

Dr.Chitra T, Associate Professor, Department of Obstetrics and Gynaecology,9488818644

Dr Rakhee Kar, Associate Professor, Department of Clinical Haematology,9487896560

Signature of the investigator:

Signature of the participant:

Place: JIPMER, Puducherry

Date :

INFORMED CONSENT DOCUMENT (ICD)

Patient / Participant information sheet

INFORMATION FOR PARTICIPANTS OF THE STUDY-GROUP B

- **Title of the project**

Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss

- **Name of the investigator/guide**

Dr Sonal Garg, JR OG; Dr. Dasari Paapa, Professor and Head, Dept. of Obstetrics and Gynaecology ,JIPMER ; Dr. Chitra T, Associate Professor, Dept. of Obstetrics and Gynaecology, JIPMER : Dr Rakhee Kar, Associate Professor, Department of clinical Haematology ,JIPMER,Puducherry.

- **Purpose of this project/study**

We are conducting this study to find out why women suffer from pregnancy loss for the first time. So far in clinical practice ,no investigations are recommended when a woman suffers from abortion for the first time. But we encounter many women asking explanation why they had an abortion. We expect that if we start investigating awomen after first pregnancy loss we can identify the causes in about 30%. This will help to reduce your anxiety and we can treat you earlier for the better outcome of next pregnancy.

IJSER

- **Procedure/methods of the study including withdrawal criteria-**

You will be asked questions regarding your age, socioeconomic status, present condition, nutritional history, menstrual history family history and past obstetric/medical/surgical history. You also need to tell us regarding the process of your early first pregnancy loss and the way it was managed including investigations done and treatment given either by medical methods or surgical methods. We will take your height and weight. We will perform a general Physical examination and systemic examination including internal genital examination. Your blood samples will be taken through venous puncture just like what you have undergone in your earlier Pregnancy. This will be used to find out hemoglobin and other routine tests done during every pregnancy including HIV. Thyroid hormone level, and other hormones like FSH, LH Prolactin and serum testosterone will be estimated if you are not

Pregnant presently. Your fasting blood sample will be taken for glucose estimation and you will be asked to drink 200ml of water mixed with glucose and blood sample will be taken twice every hour to find whether you are suffering from Diabetes. Urine sample and cervical swab will be taken to identify infectious causes. Ultrasonographic examination will be done to find out any abnormalities in your uterus and ovaries and to assess your fetus. If no causes are identified by these investigations we will investigate you further by taking blood samples for rare causes like blood clotting disorders .You will be intimated about the results of the tests and explained regarding the possible cause of your pregnancy loss. And appropriate management would be offered.

- **Expected duration of the subject participation-** Once for examination and taking blood samples and next time for review of reports and follow up and management if necessary.

The benefits to be expected from the research to the participant or to others and the post trial responsibilities of the investigator-

Causes of pregnancy loss would be identified early and treated pre- conceptionally before the next pregnancy. Medical (endocrine and immunological) causes would be optimally treated and hence better pregnancy outcome would be achieved later.

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- **Any risks expected from the study to the participant**- You will experience pain associated with blood sampling,in very rare cases there might be haematoma formation. In addition to this, you might feel uncomfortalble as per vaginum examination will be performed.
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- **Reimbursement for participating in the study**- No compensation will be given for participation in this study.
- **Compensation to the participants for foreseeable risks and unforeseeable risks related to research study leading to disability or death**- No foreseeable risks. For

unforseeable risks related to research you will be provided compensation as per JIPMER guidelines.

- **Freedom to withdraw from the study at any time during the study period without the loss of benefits that the participant would otherwise be entitled**- Participation in this study is voluntary. You have the right to withdraw from the study at any point of time without loosing your benefits.

- **Possible current and future uses of the biological material to be generated from the research and if the material is likely to be used for secondary purposes or would be shared with others, this should be mentioned**- The biological material will be used till the tests are done for this study and it will not be used for future purposes.

- **Possible current and future uses of the data to be generated from the research and if the data is likely to be used for secondary purposes or would be shared with others, this should be mentioned**- The data generated from this study may be used for scientific presentation and publication, however your personal information and identity will not be revealed.

IJSER

Address and mobile number of the Principal investigator (PI) and Co- PI, if any

Dr. Sonal Garg, Junior Resident, Department of Obstetrics and Gynaecology, 7598907264

Dr.Dasari Paapa, Professor and Head, Department of Obstetrics and Gynaecology, 9442566883

Dr.Chitra T, Associate Professor, Department of Obstetrics and Gynaecology,9488818644

Dr. Rakhee Kar, Associate Professor, Department of Clinical Haematology,9487896560

Signature of the investigator:

Signature of the participant:

Place: JIPMER,Puducherry

Date :

CONSENT FORM

Title of the project-

Comparative study of etiological factors of first one early pregnancy loss with that of recurrent pregnancy loss

Participant's name: Address:

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have been given an information sheet giving details of the study. **Risk and benefit of this project has been explained to me.** I fully consent to participate in the above study.

IJSER

I have been informed that confidentiality regarding my participation in the study will be maintained throughout the study period as well as after completion of the study.

Signature of the witness:_____Date: _____

Name and address of the witness:

Signature of the investigator:_____Date: _____

njhptpf;fg;gL;L rk;kj Mtzk

Nehahsp / gq;FngWNthh;f;fhd jfty jhs

gq;FngWgth;f;fhd jftywpf;if (FO –A)

Ma;tpd; jiyg;G :

"Muk;gfhy fh;g;g ,og;G Nehawpe;j fhuzpfspd kWgpwg;G fh;g;g ,og;GLD; xg;gPL;L Ma;T"

Ma;T nra;ghpd ngah : LHF;LH;. Nrhdy fhh;f;
,sepiy ciwtPL kUj;Jth;
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

topfhL;bapd ngah : LHF;LH;. jhrhhpghg;gh
Nguhrphpah kw;Wk Jiwj;jiyth>
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

Jiz topfHL;bfsp;d ngah : LHF;LH;. rpj;uh .T
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kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

: LHF;LH;. uhf;fp fhh>
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Nehapay Jiw>
[pg;kh;> GJr;Nrhp

Ma;tpd; Nehf;fk :

ngz;fs fh;g;g ,og;G Kjy; Kiwahf Vd ghjpf;fg;gLfpwhh;fs vd;gij mwpa ,e;j Ma;T ELJ;JfpNwhk; xU ngz Kjy; Kiwahf fuf;fiyg;G Nehapdhy ghjpf;fg;gLifapy ve;jnthU ghpNrhjidfSk nra;ag;gLtjpy;iy. Mdhy gy ngz;fs Vd fUf;fiyg;G nra;jhh;fs vd;w tpsf;fj;ij ehq;fs re;jjp;Njhk ehk

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Kjy; fh;g;g ,og;gpw;F gpd;dh ngz;fs tprhuidia njhq;fpdhy ehk 30% fhuzq;fis MILahsk fhzKbAk vd;W vjph;ghh;f;fpNwhk;. ,J cq;fs ftiyia Fiwf;f cjTfpwJ. Kd;djhfNt rpwe;j KbTf;F ehq;fs cq;fSf;F cjt KbAk;.

Ma;tpd; nra;Kiw/ nray;Kiw> jpUk;gg;ngWtjw;fhd Kiwfs cL;gL :

cq;fs taJ> r%f nghUshjhu epiy> CL;Lr;rj;J tuyhW> khjtPLha tuyhW> FLk;g tuyhW kw;Wk FLE;J fhy kfg;Ngwpay / kUj;Jtk / mWitrpfpr;ir tuyhW gw;wpa Nfs;tpfs NFL;fg;gLk;. cq;fs Muk;gfhy Kjy fh;g;g ,og;G kw;Wk kUj;Jt Kiwfs my;yJ mWit rpfpr;ir topKiwfshy toq;fg;gL;L tprhuizfs kw;Wk rpfpr;irfs CL;gL eph;tfpf;fg;gL;L topKiwiag gw;wp ePq;fs vq;fsPLk $w Ntz;Lk;. cq;fs cauj;ijAk> VILIAAk> ehq;fs vLg;Nghk;. CLw;$wpay; ghpNrhjidia cs;sLf;fpa xU nghJ CLy ghpNrhjid kw;Wk KiwNfL ghpNrhjid nra;Nthk;. cq;fs ,uj;j khjphpfs cq;fs Ke;ija fh;g;gj;jpy cs;sijg NghyNt euk;G topahf vLf;fg;gLk;. vr;It CL;gL xt;nthU fh;g;gj;jpYk `PNkhFNshgpd kw;Wk gpw tof;fkhd Nrhjidfis fz;Lgpbf;f ,J cjTfpwJ. ijuha;L `hh;Nkhd epiy kw;Wk vg;v];vr> GNuhNyf;bd kw;Wk rPuk NL];bNuhd Nghd;w gpw `hh;Nkhd vLj;J jw;NghJ ePq;fs fh;g;gkhf ,y;iy vd;why kjpg;gPLg;gLk;. rhg;gpLtjw;F Kd ,uj;j khjphpahdJ FSNfh] kjpg;gPL;bw;F vLj;Jf;nfhs;sg;gLk;. ePhpopT Nehahy; ghjpf;fg;gLfpwjh vd;gijg ghh;g;gjw;F FSf;Nfh] kw;Wk ,uj;j khjphp fye;jpUf;Fk 200kpy;yp jz;zPiu Fbf;f Ntz;Lk;. rpWePuf khjhp> fh;g;gg;ig tha khjphp njhw;W Neha;fis MILahsk fhz vLf;fg;gLk;. MY;L;uhNrhNdh fpuhgpf ghpNrhjid cq;fs fUg;ig kw;Wk fUg;igapYs;s ve;j mrhjhuzq;fisAk; fz;Lgpbj;J cq;fs fUit kjpg;gPL nra;tjwF nra;ag;gLk;. ,e;j fhuzq;fshy; ve;jnthU fhuzpfSk MILahsk fhzg;gLtpy;iy vd;why ,uj;jf;frpT rPh;FiyTfisg Nghd;w mhpa fhuzq;fSf;fhf ,uj;j khjphpfs vLj;Jf;nfhs;tjd %yk NkYk Muha;Nthk;. cq;fs fh;g;g ,og;gpw;fhd rhj;jpakhd fhuzq;fisg gw;wp Nrhjid KbTfs gw;wp tpsf;fg;gL;L mjw;fhd nghUj;jkhd Nkyhz;ik toq;fg;gLk;.

Ma;tpy; gq;Nfw;gth; vjph;ghh;f;Fk gq;Nfw;Fk fhy mtfhrk :

xU Kiw Nrhjid kw;Wk ,uj;j khjphpfs vLj;J mLj;j Kiw mwpf;iffs Ma;T kw;Wk Njitg;gL;Lhy; njhLh;e;J nfhs;sg;gLk;.

Ma;tpdhy gq;Nfw;gth; my;yJ kw;wth;fSf;F vjph;ghh;f;fg;gLk ed;ikfs kw;Wk Ma;tpw;F gpwF Ma;TNkw;nfhs;gthpd nghWg;Gfs :

fh;g;g ,og;G fhuzq;fs Muk;g fh;g;gk kw;Wk mLj;j fh;g;gk Kd fh;g;gk Vw;gLtjw;F Kd;G rpfpr;ir kUj;Jtk (vz;NLhfpiud kw;Wk Neha;jLg;G) fhuzq;fs cfe;j Kiwapy; rpfpr;iraspf;fg;gLk;. vdNt rpwe;j fh;g;g tpisT gjd;dh MILag;gLk;.

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Ma;tpy; gq;Nfw;gtUf;F vjph;ghh;f;fg;gLk Mgj;Jfs VNjDk :

,uj;j khjphpfSld njhLh;GiLa Fiwe;j mghaj;ijtPL mjpfkhd cs;sJ.

Ma;T Fwpg;NgLfspd ,ufrpak guhkhpj;jy; :

ePq;fs ,e;j Ma;tpdpy; gq;Nfw;gjidAk> Ma;T Fwpg;NgLfisAk; ,ufrpakhf itf;fg;gLk. Ma;T KbTfis Ma;T nra;Ak NghJk> gpuRhpf;fg;gLk NghJk kw;Wk kw;w Ma;T Nkw;nfhs;gtHPlk Njitg;gL;Lhy; KbTfis gfph;e;Jnfhs;Sk;NghJk cq;fSILA jdpg;gL;L MILahsj;ij njhpag;gLj;j kHL;LhJ. cq;fisg gw;wpa Ma;T Fwpg;Gfis Ma;tpw;fhfTk> njHLH ftdpg;gpw;fhfTk %d;W tUL fhyj;jpw;F itj;jpUf;fg;gLk.

Ma;T rk;ke;jkhd ,uzq;fSf;F ,ytr rpfpr;ir trjp

Ma;T rk;ke;jkhd ,uzq;fSf;Fk [pg;kh; kUj;Jtkid topFHL;Ljy;gb ,ytrkhf rpfpr;ir toq;fg;gLk.

,e;j Ma;tpy; gq;Nfw;gjw;F VjhtJ ,og;gPL toq;fg;gLkh?

,e;j Ma;tpy; gq;Nfw;gjw;fhf ve;j ,og;gPLk toq;fg;gLkHL;LhJ.

Ma;tpdhy njhpe;Njh my;yJ njhpahkNyh Vw;gLk Cdk ,y;yJ ,wg;gpw;F ,og;gPL toq;fg;gLkh?

Ma;T rk;ke;jkhf njhpe;Njh my;yJ njhpahtpjkhfNth VNjDk Mgj;Jfs epfo;tjpd %yk Cdk my;yJ ,wg;G NEHPL;Lhy; gq;Nfw;gtUf;F [pg;kh; kUj;Jtkid topfhL;Ljy;gb ,og;gPL toq;fg;gLk.

Ma;tpypUe;J Rje;jpukhf tpyfpf;nfhs;Sk chpik gw;wp :

,e;j Ma;tpy; ePq;fs gq;Nfw;gJ Kw;wpYk; cq;fs KbthFk; Ve;j Neuj;jpYk ePq;fs Ma;tpypUe;J tpyfpf;nfhs;syhk ,jdhy [pg;kh; kUj;Jtkidapy cq;fSf;F mspf;fg;gLk rpfpr;irapy; vt;tpj ghjpg;Gk Vw;gLhJ. vg;nghLJk Nghy cq;fs Neha;f;F jukhd rpfpr;ir mspf;fg;gLk.

Ma;tpypUe;j ngwg;gL;L caphpay; nghUs;fs vjph;fhy gad;ghL> kw;Wk ,uz;LhtJ Njitf;fhf gad;gLj;JtJ my;yJ kw;wth;fSld ,jid gfph;e;Jnfhs;tJ> ,jid njhpag;gLj;jTk

,e;j Ma;tpw;fhd Nrhjidfs Nkw;nfhs;sg;gLtjw;F Kd;dh caphpay; nghUs gad;gLj;jg;gLk kw;Wk mJ vjph;fhy Nehf;fq;fSf;fhf gad;gLj;jg;gLhJ.

IJSER

Ma;tpypUe;j ngwg;gL;L tpjtuq;fspd jw;fhy kw;Wk vjph;fhy gad;ghL> kw;Wk ,uz;LhtJ Njitf;fhf gad;gLj;JtJ my;yJ kw;wth;fSld ,jid gfph;e;Jnfhs;tJ> ,jid njhpag;gLj;jTk

,e;j Ma;tpypUe;J cUthf;fg;gL;L jfty;fs mwptpay; tpsf;fj;jpw;fhf kw;Wk ntspaPL;bw;fhf gad;gLj;jg;gLyhk;. ,Ug;gpDk cq;fs jdpg;gL;L jfty;fs kw;Wk MILahsq;fs ntspg;gLj;jg;gL kHL;LhJ.

Kjd;ik Ma;thshpd ngah; kw;Wk Kfthp kw;Wk njhiyNgrp vz :

Ma;T nra;ghpd ngah : **LHF;LH;. Nrhdy fhh;f;**
,sepiy ciwtPL kUj;Jth;
kfg;NgW kw;Wk khjh;Neha;j;Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 7598907264
kpd;dQ;ry : sonalllgargg@gmail.com

topfhL;bapd ngah : **LHF;LH;. jhrhpghg;gh**
Nguhrphpah kw;Wk Jiwj;jiyth>
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9442566883
kpd;dQ;ry : dasaripapa@gmail.com

Jiz topfhL;bfs;d ngah : **LHF;LH;. rpj;uh T.**
$Ljy; Nguhrphpah
kfg;NgW kw;Wk khjh;Neha;Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9488818644
kpd;dQ;ry : drchitra@yahoo.com

: **LHF;LH;. uhf;fp fhh>**
,iz Nguhrphpah
Nehapay Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9487896560
kpd;dQ;ry : drrakheekar@gmail.com

Ma;thshpd ifnahg;gk :

gq;Nfw;gthpd ifnahg;gk / ngUtuy Nuif :

,LK :

Njj; :

IJSER

xg;Gjy; gbtk

Ma;tpd; jiyg;G:

"Muk;gfhy fh;g;g ,og;G Nehawpe;j fhuzpfspd kWgpwg;G fh;g;g ,og;GLD; xg;gPL;L Ma;T"

gq;Nfw;gtupd ngau : Kftup:

,e;j Ma;T gw;wpa jfty;fs vdf;F vdJ jha;nkhopapy VSPAEILApy vOj;J %ykhfTk tha;nkhop thapyhfTk njuptpf;fg;gL;Ls;sJ. ehd mtw;iw KOikahf vt;tpj lakpd;wp Gupe;Jnfhz;Ls;Nsd; NkYk vdJ laq;fis fisa cupa tha;g;Gk mspf;fg;gL;Ls;sJ. ,e;j Ma;tpy; vdJ gq;fspg;G vdJ KOtpUg;gj;jpdhy kL;LNk vd;Wk ahnjhU fhuzKk;d;wp ve;j Neuj;jpYk ,e;j Ma;tpy; ,Ue;J tpyfpf;nfhs;s KO Rje;jpuKs;sJ vd;Wk Gupe;J nfhz;Ls;Nsd; mt;thW tpyfpf;nfhs;tjhy vdf;F mspf;fg;gLk kUj;Jt Nritfs ghjpffg;gLhJ vd;gijAk; tpuptpf Gupe;Jnfhz;NLd; ,e;j Ma;tpdhy Vw;gLk ed;ikfs kw;Wk Mgj;Jfs gw;wp vdf;F tpsf;fkhf $wg;gL;LJ. ,e;j Ma;tpy; FPILF;Fk KbTfis mwptpay; Nehf;fj;jpy gad;gLj;Jk gL;rj;jpy vdf;F ML;Nrhgid ,y;iy. ,e;j Ma;it gw;wpa KOjfty; gbtk vdf;F toq;fg;gL;Ls;sJ. ,e;j Ma;tpy; gq;Fngw ehd KO xg;Gjy nfhLf;fpd;Nwd;.

IJSER

gq;Nfw;gtupd ifnahg;gk / ifeHL;L : Njjp:

rhL;rpahsupd ifnahg;gk / ifeHL;L : Njjp:

rhL;rpahsupd ngau kw;Wk Kftup:

Ma;thsupd ifnahg;gk : Njjp:

njhptpf;fg;gL;L rk;kj Mtzk

Nehahsp / gq;FngWNthh;f;fhd jfty jhs

gq;FngWgth;f;fhd jftywpf;if (FO –B)

Ma;tpd; jiyg;G :

"Muk;gfhy fh;g;g ,og;G Nehawpe;j fhuzpfspd kWgpwg;G fh;g;g ,og;GLD; xg;gPL;L Ma;T"

Ma;T nra;ghpd ngah : LHF;LH;. Nrhdy fhh;f;
,sepiy ciwtPL kUj;Jth;
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

topfhL;bapd ngah : LHF;LH;. jhrhhpghg;gh
Nguhrphpah kw;Wk Jiwj;jiyth>
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

Jiz topfhL;bfs;d ngah : LHF;LH;. rpj;uh .T
,iz Nguhrphpah
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp

: LHF;LH;. uhf;fp fhh>
,iz Nguhrphpah
Nehapay Jiw>
[pg;kh;> GJr;Nrhp

Ma;tpd; Nehf;fk :

xU fh;g;g ,og;G kw;wk ,uz;L my;yJ mjw;F Nkw;gL;L fh;g;g ,og;Gfshy ngz;fSf;F ,J Nghdw; fhuzq;fisf fz;Lwpayhkh vd;gijf fz;Lwpa ,e;j Ma;T Nkw;nfhs;fpNwhk;. xU ngz Kjy Kiwahf fUf;fiyg;G Nehapdhy ghjpf;fg;gLifapy ve;jnthU tprhuidfSk kWf;fg;gLfpd;wd. Mdhy gyngz;fs Vd fUf;fiyg;G nra;jhh;fs vd;w tpsf;fj;ij ehq;fs re;jpj;Njhk;. ehk Kjy

IJSER

fh;g;g ,og;Gf;F gjd;dh ngz;fs tprhuid njhq;fpdhy ehk 30% fhuzq;fs MILahsk fhd KbAk vd;W vjph;ghh;f;fpNwhk;. ,J mLj;j Kiw rpwe;j fh;g;gk tpistpf;Fk Kd;G cq;fSf;F rpfpr;ir mspf;f cjtpfukhf ,Uf;Fk;.

Ma;tpd; nra;Kiw kw;Wk nray;Kiw :

cq;fs taJ> r%f nghUshjhu epiy> CL;Lr;rj;J tuyhW> khjtPLha tuyhW> FLk;g tuyhW kw;Wk FLE;J fhy kfg;Ngwpay / kUj;Jtk / mWitrpfpr;ir tuyhW gw;wpa Nfs;tpfs NFL;fg;gLk;. cq;fs Muk;gfhy Kjy fh;g;g ,og;G kw;Wk kUj;Jt Kiwfs my;yJ mWit rpfpr;ir topKiwfshy toq;fg;gL;L tprhuizfs kw;Wk rpfpr;irfs CL;gL eph;tfpf;fg;gL;L topKiwiag gw;wp ePq;fs vq;fsPLk $w Ntz;Lk;. cq;fs cauj;ijAk> VILIAAk> ehq;fs vLg;Nghk;. CLw;$wpay; ghpNrhjidia cs;sLf;fpa xU nghJ CLy ghpNrhjid kw;Wk KiwNfL ghpNrhjid nra;Nthk;. cq;fs ,uj;j khjphpfs cq;fs Ke;ija fh;g;gj;jpy cs;sijg NghyNt euk;G topahf vLf;fg;gLk;. vr;lt; CL;gL xt;nthU fh;g;gj;jpYk ̀PNkhFNshgpd kw;Wk gpw tof;fkhd Nrhjidfis fz;Lgpbf;f ,J cjTfpwJ. ijuha;L ̀hh;Nkhd epiy kw;Wk vg;v];vr> GNuhNyf;bd kw;Wk rPuk NL];bNuhd Nghd;w gpw ̀hh;Nkhd vLj;J jw;NghJ ePq;fs fh;g;gkhf ,y;iy vd;why kjpg;gPLg;gLk;. rhg;gpLtjw;F Kd ,uj;j khjphpahdJ FSNfh] kjpg;gPL;bw;F vLj;Jf;nfhs;sg;gLk;. ePhpopT Nehahy ghjpf;fg;gLfpwjh vd;gijg ghh;g;gjw;F FSf;Nfh] kw;Wk ,uj;j khjphp fye;jpUf;Fk 200kpy;yp jz;zPiu Fbf;f Ntz;Lk;. rpWePuf khjphp> fh;g;gg;ig tha khjphp njhw;W Neha;fis MILahsk fhz vLf;fg;gLk;. MY;L;uhNrhNdh fpuhgpf ghpNrhjid cq;fs fUg;ig kw;Wk fUg;igapYs;s ve;j mrhjhuzq;fisAk; fz;Lgpbj;J cq;fs fUit kjpg;gPL nra;tjw;F nra;ag;gLk;. ,e;j fhuzq;fshy; ve;jnthU fhuzpfSk MILahsk fhzg;gLtpy;iy vd;why ,uj;jf;frpT rPh;FiyTfisg Nghd;w mhpa fhuzq;fSf;fhf ,uj;j khjphpfs vLj;Jf;nfhs;tjd %yk NkYk Muha;Nthk;. cq;fs fh;g;g ,og;gpw;fhd rhj;jpakhd fhuzq;fisg gw;wp Nrhjid KbTfs gw;wp tpsf;fg;gL;L mjw;fhd nghUj;jkhd Nkyhz;ik toq;fg;gLk;.

Ma;tpy; gq;Nfw;gth; vjph;ghh;f;Fk gq;Nfw;Fk fhy mtfhrk :

xU Kiw Nrhjid kw;Wk ,uj;j khjphpfs vLj;J mLj;j Kiw mwpf;iffs Ma;T kw;Wk Njitg;gL;Lhy; njhLh;e;J nfhs;sg;gLk;.

Ma;tpdhy gq;Nfw;gth; my;yJ kw;wth;fSf;F vjph;ghh;f;fg;gLk ed;ikfs kw;Wk Ma;tpw;F gpwF Ma;TNkw;nfhs;gthpd nghWg;Gfs :

fh;g;g ,og;G fhuzq;fs Muk;g fh;g;gk kw;Wk mLj;j fh;g;gk Kd fh;g;gk Vw;gLtjw;F Kd;G rpfpr;ir kUj;Jtk (vz;NLhfpiud kw;Wk Neha;jLg;G) fhuzq;fs cfe;j Kiwapy; rpfpr;iraspf;fg;gLk;. vdNt rpwe;j fh;g;g tpisT gjd;dh MILag;gLk;.

IJSER

Ma;tpy; gq;Nfw;gtUf;F vjph;ghh;f;fg;gLk Mgj;Jfs VNjDk :

,uj;j khjphpfSld njhLh;GiLa Fiwe;j mghaj;ijtPL mjpfkhd cs;sJ.

Ma;T Fwpg;NgLfspd ,ufrpak guhkhpj;jy; :

ePq;fs ,e;j Ma;tpdpy; gq;Nfw;gjidAk> Ma;T Fwpg;NgLfisAk; ,ufrpakhf itf;fg;gLk. Ma;T KbTfis Ma;T nra;Ak NghJk> gpuRhpf;fg;gLk NghJk kw;Wk kw;w Ma;T Nkw;nfhs;gtHPLk Njitg;gL;Lhy; KbTfis gfph;e;Jnfhs;Sk;NghJk cq;fSILA jdpg;gL;L MILahsj;ij njhpag;gLj;j kHL;LhJ. cq;fisg gw;wpa Ma;T Fwpg;Gfis Ma;tpw;fhfTk> njHLH ftdpg;gpw;fhfTk %d;W tUL fhyj;jpw;F itj;jpUf;fggLk.

Ma;T rk;ke;jkhd ,uzq;fSf;F ,ytr rpfpr;ir trjp

Ma;T rk;ke;jkhd ,uzq;fSf;Fk [pg;kh; kUj;Jtkid topFHL;Ljy;gb ,ytrkhf rpfpr;ir toq;fg;gLk.

,e;j Ma;tpy; gq;Nfw;gjw;F VjhtJ ,og;gPL toq;fg;gLkh?

,e;j Ma;tpy; gq;Nfw;gjw;fhf ve;j ,og;gPLk toq;fg;gLkHL;LhJ.

Ma;tpdhy njhpe;Njh my;yJ njhpahkNyh Vw;gLk Cdk ,y;yJ ,wg;gpw;F ,og;gPL toq;fg;gLkh?

Ma;T rk;ke;jkhf njhpe;Njh my;yJ njhpahtpjkhfNth VNjDk Mgj;Jfs epfo;tjpd %yk Cdk my;yJ ,wg;G NEHPL;Lhy; gq;Nfw;gtUf;F [pg;kh; kUj;Jtkid topfhL;Ljy;gb ,og;gPL toq;fg;gLk.

Ma;tpypUe;J Rje;jpukhf tpyfpf;nfhs;Sk chpik gw;wp :

,e;j Ma;tpy; ePq;fs gq;Nfw;gJ Kw;wpYk; cq;fs KbthFk. Ve;j Neuj;jpYk ePq;fs Ma;tpypUe;J tpyfpf;nfhs;syhk ,jdhy [pg;kh; kUj;Jtkidapy cq;fSf;F mspf;fg;gLk rpfpr;irapy; vt;tpj ghjpg;Gk Vw;gLhJ. vg;nghLJk Nghy cq;fs Neha;f;F jukhd rpfpr;ir mspf;fg;gLk.

Ma;tpypUe;j ngwg;gL;L caphpay; nghUs;fs vjph;fhy gad;ghL> kw;Wk ,uz;LhtJ Njitf;fhf gad;gLj;JtJ my;yJ kw;wth;fSld ,jid gfph;e;Jnfhs;tJ> ,jid njhpag;gLj;jTk

,e;j Ma;tpw;fhd Nrhjidfs Nkw;nfhs;sg;gLtjw;F Kd;dh caphpay; nghUs gad;gLj;jg;gLk kw;Wk mJ vjph;fhy Nehf;fq;fSf;fhf gad;gLj;jg;gLhJ.

Ma;tpypUe;j ngwg;gL;L tptuq;fspd jw;fhy kw;Wk vjph;fhy gad;ghL> kw;Wk ,uz;LhtJ Njitf;fhf gad;gLj;JtJ my;yJ kw;wth;fSld ,jid gfph;e;Jnfhs;tJ> ,jid njhpag;gLj;jTk

IJSER

,e;j Ma;tpypUe;J cUthf;fg;gL;L jfty;fs mwptpay; tpsf;fj;jpw;fhf kw;Wk ntspaPL;bw;fhf gad;gLj;jg;gLyhk. ,Ug;gpDk cq;fs jdpg;gL;L jfty;fs kw;Wk MiLahsq;fs ntspg;gLj;jg;gL kHL;LhJ.

Kjd;ik Ma;thshpd ngah; kw;Wk Kfthp kw;Wk njhiyNgrp vz :

Ma;T nra;ghpd ngah : **LHF;LH;. Nrhdy fhh;f;**
,sepiy ciwtPL kUj;Jth;
kfg;NgW kw;Wk khjh;Neha;j;Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 7598907264
kpd;dQ;ry : sonalllgargg@gmail.com

topfhL;bapd ngah : **LHF;LH;. jhrhpghg;gh**
Nguhrphpah kw;Wk Jiwj;jiyth>
kfg;NgW kw;Wk khjh;Neha; Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9442566883
kpd;dQ;ry : dasaripapa@gmail.com

Jiz topfhL;bfsp;d ngah : **LHF;LH;. rpj;uh T.**
$Ljy; Nguhrphpah
kfg;NgW kw;Wk khjh;Neha;Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9488818644
kpd;dQ;ry : drchitra@yahoo.com

: **LHF;LH;. uhf;fp fhh>**
,iz Nguhrphpah
Nehapay Jiw>
[pg;kh;> GJr;Nrhp
miyiyNgrp vz : 9487896560
kpd;dQ;ry : drrakheekar@gmail.com

IJSER

Ma;thshpd ifnahg;gk :

gqNfw;gthpd ifnahg;gk / ngUtpuy Nuif :

,LK :

Njjp :

xg;Gjy gbtk

Ma;tpd; jiyg;G:

"Muk;gfhy fh;g;g ,og;G Nehawpe;j fhuzpfspd kWgpwg;G fh;g;g ,og;GLD; xg;gPL;L Ma;T"

gq;Nfw;gtupd ngau : Kftup:

,e;j Ma;T gw;wpa jfty;fs vdf;F vdJ jha;nkhopapy VSPAEILApy vOj;J %ykhfTk tha;nkhop thapyhfTk njuptpf;fg;gL;Ls;sJ. ehd mtw;iw KOikahf vt;tpj lakpd;wp Gupe;Jnfhz;Ls;Nsd;. NkYk vdJ laq;fis fisa cupa tha;g;Gk mspf;fg;gL;Ls;sJ. ,e;j Ma;tpy; vdJ gq;fspg;G vdJ KOtpUg;gj;jpdhy kL;LNk vd;Wk ahnjhU fhuzKk;d;wp ve;j Neuj;jYk ,e;j Ma;tpy; ,Ue;J tpyfpf;nfhs;s KO Rje;jpuKs;sJ vd;Wk Gupe;J nfhz;Ls;Nsd;. mt;thW tpyfpf;nfhs;tjhy vdf;F mspf;fg;gLk kUj;Jt Nritfs gh;jpf;fg;gLhJ vd;gij Ak; tpuptHf Gupe;Jnfhz;NLd;. ,e;j Ma;tpdhy Vw;gLk ed;ikfs kw;Wk Mgj;Jfs gw;wp vdf;F tpsf;fkhf $wg;gL;LJ. ,e;j Ma;tpy; FPILF;Fk KbTfis mwptpay; Nehf;fj;jpy gad;gLj;Jk gL;rj;jpy vdf;F ML;Nrhgid ,y;iy. ,e;j Ma;it gw;wpa KOjfty; gbtk vdf;F toq;fg;gL;Ls;sJ. ,e;j Ma;tpy; gq;Fngw ehd KO xg;Gjy nfhLf;fpd;Nwd;.

IJSER

gqNfw;gtupd ifnahg;gk / ifEHL;L : Njj;p:

rhL;rpahsupd ifnahg;gk / ifeHL;L : Njj;p:

rhL;rpahsupd ngau kw;Wk Kftup:

Ma;thsupd ifnahg;gk : Njj;p:

PROFORMA-GROUP A

Name: Hospital No: Serial No:

Age:

Unit:

Address:

Group A/B:

Educational status: primary/secondary/graduate/postgraduate

Occupation:

Socioeconomic status : I/II/III/IV/V (Kuppuswamy ‘s socioeconomic status scale)

Obstetric formula:

Gestational age at pregnancy loss

Menstrual History:

Age at menarche No of days

Cycles: regular/irregular Dysmenorrhoea

Flow: Scanty/Moderate/Heavy LMP

Marital History:

Husband ‘s name:

No of years married:

Consanguinous/Non-consanguinous

Contraception: OCPs/IUCD/others

Past medical and surgical history:

Hypertension / Hypothyroidism / Diabetes / Cardiovascular disease / Infertility / Surgeries / Neoplasms

IJSER

Family history:

HTN/Diabetes/Cardiovascular disease / Thyroid disorders / Pulmonary TB / Infertility / Malignancies

Drug history:

OCP

Others

Personal history: DIET

High glycemic index foods

Low glycemic index foods

Total calories- Required

Consumed

Deficiency/Excess

Fat:

Protein:

IJSER

EXAMINATION:

GENERAL PHYSICAL EXAMINATION:

HEIGHT (cm)

WEIGHT (cm)

BMI

Underweight	<18.5
Normal	18.5-24.9
Overweight	25.0-29.9
Obesity- Class I	30.0-34.9
Class II	35.0-39.9
Class III	>40.0

PALLOR

THYROID

B/L BREAST

VITALS:

Temperature

Pulse

Blood pressure

SYSTEMIC EXAMINATION:

RS/CVS:

P/A:

P/S/V:

INVESTIGATIONS:

INVESTIGATION:	**STATUS: Immediate post abortal DATE:**	**STATUS: Non pregnant state DATE:**
Complete hemogram: **Hb (g%)** **Total leucocyte count** **Differential count** **Platelet count** **Peripheral smear**		
BUSE/LFT: **RBS** **Urea/Creatinine** **Sodium/Potassium** **Bilirubin(Total/Direct)** **AST/ALT** **STP/Albumin**		
HIV		
HBsAg		
VDRL		
Urine C/S		
Cervical swab C/S		
75g GTT: **Fasting** **1 Hr** **2 Hr**		
TFT		
USG		

IJSER

Hormonal profile for PCOS: **S.FSH** **S.LH** **LH/FSH** **Total testosterone** **S. Prolactin**		
Thrombophilia profile: **APLA:** **Lupus anticoagulant** **Beta 2 glycoprotein 1 Antibody:** **IgM** **IgG** **Anticardiolipin antibody:** **IgM** **IgG**		

CAUSE OF PREGNANCY LOSS:

TREATMENT ADVICED:

IJSER

PROFORMA-GROUP B

Name: Hospital No:
Serial No:

Age:

Unit:

Address:

Group A/B:

Educational status: primary/secondary/graduate/postgraduate

Occupation:

Socioeconomic status : I/II/III/IV/V (Kuppuswamy ‘s socioeconomic status scale)

Obstetric formula:

Gestational age at pregnancy loss:

IJSER

Menstrual History:

Age at menarche No of days

Cycles: regular/irregular Dysmenorrhoea

Flow: Scanty/Moderate/Heavy LMP

Marital History:

Husband ‘s name:

No of years married:

Consanguinous/Non-consanguinous

Obstetric history: Number of abortions

Order of preg	Mode of conception	GA at pregnancy loss	Type of pregnancy loss	Management

Contraception: OCPs/IUCD/others

Past medical and surgical history:

Hypertension / Hypothyroidism / Diabetes / Cardiovascular disease / Infertility / Surgeries /

Neoplasms

Family history:

HTN/Diabetes/Cardiovascular disease / Thyroid disorders / Pulmonary TB / Infertility / Malignancies

Drug history:

OCP

Others

Personal history: DIET

High glycemic index foods

Low glycemic index foods

Total calories- Required

Consumed

Deficiency/Excess

Fat:

Protein:

EXAMINATION:

GENERAL PHYSICAL EXAMINATION:

HEIGHT (cm)

WEIGHT (cm)

BMI

Underweight	<18.5
Normal	18.5-24.9
Overweight	25.0-29.9

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Obesity- Class I	30.0-34.9
Class II	35.0-39.9
Class III	>40.0

PALLOR

THYROID

B/L BREAST

VITALS:

Temperature

Pulse

Blood pressure

SYSTEMIC EXAMINATION:

RS/CVS:

P/A:

P/S/V:

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INVESTIGATIONS:

INVESTIGATION:	**STATUS: Immediate post abortal DATE:**	**STATUS: Pregnant state DATE:**
Complete hemogram: **Hb (g%)** **Total leucocyte count** **Differential count** **Platelet count** **Peripheral smear**		
BUSE/LFT: **RBS** **Urea/Creatinine** **Sodium/Potassium** **Bilirubin(Total/Direct)** **AST/ALT** **STP/Albumin**		
HIV		
HBsAg		
VDRL		
Urine C/S		
Cervical swab C/S		
75g GTT: **Fasting** **1 Hr** **2 Hr**		
TFT		
USG		
Hormonal profile for PCOS: **S.FSH** **S.LH** **LH/FSH** **Total testosterone** **S. Prolactin**		
Thrombophilia profile: **APLA:**		

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Lupus anticoagulant **Beta 2 glycoprotein 1 Antibody:** **IgM** **IgG** **Anticardiolipin antibody:** **IgM** **IgG**		

CAUSE OF PREGNANCY LOSS:

TREATMENT ADVICED:

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MASTER CHART KEY

ACLA	- Anticardiolipin antibodies
APLA	- Antiphospholipid antibodies
BIC	- Bicornuate uterus
BMI	- Body mass index
ß2 GP	- Beta 2 glycoprotein antibodies
DIDEL	- Uterus didelphius
DIP	- Diabetes in pregnancy
DM	- Diabetes mellitus
GDM	- Gestational diabetes mellitus
LAC	- Lupus anticoagulant
ND	- Not done
PCOS	- Polycystic ovary syndrome
PSD	- Protein S deficiency
SEP	- Septate uterus

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MASTER CHART

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Sl. No.	Age	Group	Gravida	Para	Live	Abortion	Obstetriccode	Noofabortions	Gaat pregnancy loss weeks	Gaat pregnancy loss days	Typeofabortion	Weight	Height	BMI	WHOBMI	Socioeconomic status Kuppuswamy classification	Uterineanomalieson USG	Typeofanomaly	PCOSfeatures on USG
1	24	Group B	2	0	0	1	G2A1	2	10	3	Missed abortion	48	1.5	21.333333	normalweight	Class IV	Absent		Absent
2	29	Group A	0	0	0	1	A1	1	9	4	Missed abortion	47	1.55	19.562955	normalweight	Class IV	Absent		Absent
3	29	Group A	0	0	0	1	A1	1	9	2	Blighted ovum	52	1.45	24.732461	normalweight	Class IV	Absent		Absent
4	29	Group B	3	0	0	2	G3A2	3	10	0	Missed abortion	59	1.56	24.243918	normalweight	Class IV	Absent		Absent
5	27	Group B	2	0	0	1	G2A1	2	13	4	Missed abortion	63	1.49	28.3771	preobesity	Class IV	Absent		Absent
6	29	Group A	1	0	0		G1	1	10	5	Missed abortion	64	1.58	25.636917	preobesity	Class IV	Absent		Absent
7	22	Group A	1	0	0		G1	1	12	1	Complete abortion	66	1.57	26.775934	preobesity	Class IV	Absent		Absent
8	23	Group A	1	0	0		G1	1	10	5	Incomplete abortion	60	1.55	24.973985	preobesity	Class III	Absent		Absent
9	23	Group B	2	0	0	1	G2A1	2	11	0	Blighted ovum	63	1.47	29.154519	preobesity	Class IV	Absent		Absent
10	21	Group A	1	0	0		G1	1	10	5	Missed abortion	45	1.5	20	normalweight	Class IV	Absent		Absent
11	20	Group A	1	0	0		G1	1	7	3	Blighted ovum	47	1.44	22.665895	normalweight	Class IV	Absent		Absent
12	20	Group A	1	0	0		G1	1	12	6	Missed abortion	45	1.56	18.491124	underweight	Class IV	Absent		Absent
13	18	Group A	1	0	0		G1	1	11	4	Missed abortion	48	1.67	17.211087	underweight	Class IV	Absent		Absent
14	23	Group B	3	0	0	2	G3A2	3	13	6	Missed abortion	50	1.43	24.451073	normalweight	Class IV	Absent		Absent
15	35	Group A	1	0	0		G1	1	11	2	Missed abortion	55	1.51	24.121749	normalweight	Class IV	Absent		Absent
16	18	Group A	1	0	0		G1	1	12	1	Blighted ovum	57	1.44	27.488426	preobesity	Class III	Absent		Absent
17	29	Group B	2	0	0	1	G2A1	2	9	4	Complete abortion	58	1.54	24.456063	normalweight	Class III	Absent		Absent
18	19	Group A	1	0	0		G1	1	12	6	Incomplete abortion	50	1.46	23.456558	normalweight	Class IV	Absent		Absent
19	22	Group B	2	0	0	1	G2A1	2	13	2	Inevitable abortion	51	1.53	21.786492	normalweight	Class IV	Absent		Absent
20	28	Group B	2	0	0	1	G2A1	2	12	4	Missed abortion	65	1.5	28.888889	preobesity	Class IV	Absent		Present
21	33	Group A	2	1	0	0	G2P1L0	1	10	4	Missed abortion	58	1.45	27.586207	preobesity	Class IV	Absent		Absent
22	26	Group A	1	0	0	0	G1	1	12	3	Incomplete abortion	56	1.44	27.006173	preobesity	Class IV	Absent		Absent
23	27	Group A	1	0	0	0	G1	1	9	6	Missed abortion	49	1.53	20.93212	normalweight	Class IV	Absent		Absent
24	25	Group A	1	0	0	0	G1	1	12	0	Missed abortion	55	1.48	25.109569	preobesity	Class III	Absent		Absent
25	28	Group B	2	0	0	1	G2A1	2	11	6	Missed abortion	50	1.4	25.510204	preobesity	Class III	Absent		Absent
26	20	Group B	2	0	0	1	G2A1	2	13	1	Blighted ovum	49	1.55	20.395421	normalweight	Class IV	Absent		Absent
27	25	Group B	2	0	0	1	G2A1	2	13	1	Missed abortion	45	1.42	22.317001	normalweight	Class III	Absent		Absent
28	28	Group A	1	0	0		G1	1	12	2	Incomplete abortion	44	1.43	21.516945	normalweight	Class III	Absent		Absent
29	32	Group B	3	0	0	2	G3A2	3	9	6	Missed abortion	70	1.46	32.839182	obesityclassI	Class IV	Absent		Absent
30	21	Group B	2	0	0	1	G2A1	2	8	3	Missed abortion	65	1.56	26.709402	preobesity	Class III	Absent		Absent
31	24	Group A	1	0	0		G1	1	12	0	Missed abortion	60	1.55	24.973985	preobesity	Class IV	Absent		Present
32	27	Group B	11	1	0	9	G11P1L0A9	> or = 4	8	5	Blighted ovum	58	1.35	31.824417	obesityclassI	Class III	Absent		Absent
33	21	Group A	1	0	0		G1	1	11	1	Blighted ovum	40	1.4	20.408163	normalweight	Class IV	Absent		Absent
34	26	Group B	2	0	0	1	G2A1	2	13	6	Missed abortion	48	1.51	21.051708	normalweight	Class IV	Absent		Present
35	35	Group A	1	0	0		G1	1	9	1	Incomplete abortion	45	1.48	20.544193	normalweight	Class III	Absent		Absent
36	23	Group B	3	1	0	1	G3P1L0A1	2	13	2	Missed abortion	55	1.54	23.191095	normalweight	Class III	Absent		Absent
37	21	Group B	2	0	0	1	G2A1	2	12	3	Missed abortion	50	1.56	20.545694	normalweight	Class IV	Absent		Absent
38	25	Group B	2	0	0		G2A1	2	13	3	Missed abortion	47	1.47	21.750197	normalweight	Class III	Absent		Absent
39	26	Group B	2	0	0	2	G3A2	3	13	4	Inevitable abortion	50	1.49	22.521508	normalweight	Class IV	Absent		Absent
40	30	Group B		0	0	9	A10	> or = 4	12	2	Missed abortion	44	1.5	19.555556	normalweight	Class III	Absent		Present
41	19	Group A	1	0	0		G1	1	12	5	Missed abortion	48	1.53	20.504934	normalweight	Class IV	Present	BIC	Absent
42	20	Group A	3	0	0	2	G3A2	3	13	3	Missed abortion	44	1.46	20.641771	normalweight	Class IV	Absent		Absent

Sl. No.	Fetusassessmenton USGanomalies	Cervicalin competence	Hbgdl	Urinecs	Cervicals wabcs	GTT	TSH	Thyroidstatus	Hormonal profile for PCOS nonpregnant state	beta 2 glycoprotein 1 antibody	Anticardiolipin antibody	Lupusanticoagulant	ProteinC	ProteinS	Causeofpregnancyloss	Known Unknown
1	Fetal anomalies absent	No	13.7	Sterile	Sterile	Abnormal	2.8	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
2	Fetal anomalies absent	No	11.8	Sterile	Sterile	Abnormal	1.8	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
3	Fetal anomalies absent	No	9.3	Sterile	Sterile	Normal	4.8	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
4	Fetal anomalies absent	No	12.8	Sterile	Sterile	Normal	2.1	Normal	Not done	Negative	Negative	Positive	Normal	Normal	LAC positive	Known
5	Fetal anomalies absent	No	8.2	Sterile	Sterile	Abnormal	1.8	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
6	Fetal anomalies absent	No	9.7	Sterile	Sterile	Abnormal	2.5	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
7	Fetal anomalies absent	No	11.9	Sterile	Growth	Normal	2.2	Normal	Not done	ND	ND	ND	ND	ND	Infection	Known
8	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.6	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
9	Fetal anomalies absent	No	9.2	Sterile	Growth	Abnormal	3.4	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
10	Fetal anomalies absent	No	6.2	Sterile	Growth	Abnormal	2.6	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
11	Fetal anomalies absent	No	9.3	Sterile	Sterile	Normal	3.1	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
12	Fetal anomalies absent	No	9.5	Sterile	Sterile	Abnormal	2.8	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
13	Fetal anomalies absent	No	10	Sterile	Sterile	Abnormal	4.5	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
14	Fetal anomalies absent	No	10.6	Sterile	Sterile	Abnormal	1.6	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
15	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	1.8	Normal	Not done	ND	ND	ND	ND	ND	Fibroid	Known
16	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
17	Fetal anomalies absent	No	10.6	Sterile	Sterile	Normal	3.05	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
18	Fetal anomalies absent	No	9.8	Sterile	Sterile	Normal	2.9	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
19	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	5.48	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
20	Fetal anomalies absent	No	9.9	Sterile	Sterile	Abnormal	2.8	Normal	Not done	ND	ND	ND	ND	ND	GDM + PCOS	Known
21	Fetal anomalies absent	No	11	Sterile	Growth	Abnormal	3.5	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
22	Fetal anomalies absent	No	11.6	Sterile	Sterile	Abnormal	2.9	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
23	Fetal anomalies absent	No	9.5	Sterile	Sterile	Abnormal	6.92	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
24	Fetal anomalies absent	No	11	Sterile	Sterile	Abnormal	3.5	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
25	Fetal anomalies absent	No	8.9	Sterile	Sterile	Normal	2.09	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
26	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.2	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
27	Fetal anomalies absent	No	11.8	Sterile	Sterile	Normal	2.9	Normal	Not done	Negative	Positive	Positive	Normal	Normal	APLA- ACLA + LAC positive	Known
28	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	3.3	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
29	Fetal anomalies absent	No	9.9	Sterile	Sterile	Abnormal	2.1	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
30	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.7	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
31	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	2.2	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
32	Fetal anomalies absent	No	9.6	Sterile	Sterile	Normal	2.4	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
33	Fetal anomalies absent	No	10.8	Sterile	Sterile	Abnormal	1.5	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
34	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	1.9	Normal	Normal	Negative	Negative	Negative	ND	ND	PCOS	Known
35	Fetal anomalies absent	No	11	Sterile	?	Normal	1.1	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
36	Fetal anomalies absent	No	6.6	Sterile	Sterile	Abnormal	5.5	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
37	Fetal anomalies absent	No	6.5	Sterile	Sterile	Normal	2.34	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
38	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
39	Fetal anomalies absent	No	9.3	Sterile	Sterile	Normal	1.21	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
40	Fetal anomalies absent	No	8	Sterile	Sterile	Abnormal	5.9	Abnormal	Abnormal	Negative	Negative	Negative	Normal	Normal	GDM + PCOS + Hypothyroid	Known
41	Fetal anomalies absent	No	11.2	Sterile	Sterile	Normal	1.6	Normal	Not done	ND	ND	ND	ND	ND	Uterine anomalies	Known
42	Fetal anomalies absent	No	13	Sterile	Sterile	Abnormal	7.8	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known

Sl. No.	Age	Group	Gravida	Para	Live	Abortion	Obstetriccode	Noofabortions	Gaat pregnancy loss weeks	Gaat pregnancy loss days	Typeofabortion	Weight	Height	BMI	WHOBMI	Socioeconomic status Kuppuswamy classification	Uterineanomalieson USG	Typeofanomaly	PCOSfeatures on USG
43	27	Group B	4	0	0	3	G4A3	> or = 4	11	2	Missed abortion	50	1.55	20.811655	normalweight	Class III	Absent		Absent
44	23	Group A	1	0	0	1	A1	1	9	3	Missed abortion	43	1.4	21.938776	normalweight	Class IV	Absent		Absent
45	26	Group B	2	0	0	1	G2A1	2	12	4	Missed abortion	45	1.5	20	normalweight	Class III	Absent		Absent
46	27	Group B	2	0	0	1	G2A1	2	9	5	Missed abortion	53	1.55	22.060354	normalweight	Class IV	Absent		Absent
47	20	Group A	1	0	0		G1	1	13	1	Missed abortion	45	1.44	21.701389	normalweight	Class III	Absent		Absent
48	35	Group B	3	0	0	2	G3A2	3	12	5	Inevitable abortion	58	1.62	22.10029	normalweight	Class III	Absent		Absent
49	27	Group A	1	0	0		G1	1	8	3	Blighted ovum	56	1.51	24.560326	normalweight	Class IV	Absent		Absent
50	35	Group A	1	0	0		G1	1	12	2	Missed abortion	48	1.53	20.504934	normalweight	Class IV	Absent		Absent
51	22	Group B	2	0	0	1	G2A1	2	8	5	Missed abortion	47	1.55	19.562955	normalweight	Class II	Absent		Absent
52	32	Group A	1	0	0		G1	1	7	1	Incomplete abortion	52	1.7	17.99308	underweight	Class II	Absent		Absent
53	27	Group A	1	0	0		G1	1	9	3	Incomplete abortion	58	1.56	23.833005	normalweight	Class IV	Absent		Absent
54	21	Group A	1	0	0		G1	1	7	6	Missed abortion	63	1.54	26.564345	preobesity	Class III	Absent		Absent
55	21	Group A	1	0	0		G1	1	10	2	Inevitable abortion	62	1.76	20.015496	normalweight	Class III	Absent		Absent
56	25	Group B	2	0	0	1	G2A1	2	7	3	Blighted ovum	55	1.57	22.313278	normalweight	Class III	Absent		Absent
57	28	Group B	2	0	0		G2A1	2	8	2	Complete abortion	61	1.65	22.405877	normalweight	Class III	Absent		Absent
58	21	Group A	1	0	0		G1	1	11	3	Blighted ovum	60	1.44	28.935185	preobesity	Class III	Absent		Absent
59	28	Group B	2	0	0	1	G2A1	2	7	6	Missed abortion	46	1.5	20.444444	normalweight	Class II	Absent		Absent
60	25	Group B	1	0	0	1	G2A1	2	11	5	Incomplete abortion	44	1.33	24.874216	normalweight	Class III	Absent		Absent
61	24	Group A	2	1	0		G2P1L0	1	9	4	Blighted ovum	45	1.45	21.403092	normalweight	Class IV	Absent		Absent
62	25	Group B	3	0	0	2	G3A2	3	7	5	Blighted ovum	46	1.6	17.96875	underweight	Class III	Absent		Absent
63	22	Group A	1	0	0		G1	1	7	1	Missed abortion	50	1.56	20.545694	normalweight	Class IV	Present	BIC	Absent
64	34	Group A	1	0	0		G1	1	12	6	Incomplete abortion	45	1.59	17.799929	underweight	Class IV	Absent		Present
65	25	Group A	1	0	0		G1	1	9	6	Missed abortion	56	1.73	18.710949	normalweight	Class III	Absent		Absent
66	26	Group A	1	0	0		G1	1	13	4	Complete abortion	58	1.52	25.103878	preobesity	Class IV	Absent		Absent
67	26	Group A	1	0	0		G1	1	11	6	Incomplete abortion	50	1.48	22.826881	normalweight	Class IV	Absent		Absent
68	28	Group B	2	0	0	1	G2A1	2	10	0	Incomplete abortion	51	1.55	21.227888	normalweight	Class IV	Absent		Absent
69	33	Group A	1	0	0		G1	1	11	3	Complete abortion	63	1.48	28.76187	preobesity	Class III	Absent		Absent
70	29	Group B	2	0	0	1	G2A1	2	10	3	Missed abortion	56	1.45	26.634958	preobesity	Class IV	Absent		Absent
71	24	Group A	1	0	0		G1	1	12	1	Missed abortion	56	1.45	26.634958	preobesity	Class III	Absent		Absent
72	29	Group A	1	0	0		G1	1	9	6	Missed abortion	47	1.45	22.35434	normalweight	Class II	Absent		Absent
73	26	Group B	4	0	0	3	G4A3	> or = 4	12	6	Missed abortion	53	1.65	19.467401	normalweight	Class III	Absent		Absent
74	21	Group B	4	0	0	3	G4A3	> or = 4	8	6	Missed abortion	52	1.56	21.367521	normalweight	Class IV	Absent		Absent
75	21	Group B	3	0	0	2	G3A2	3	12	3	Missed abortion	49	1.65	17.998163	underweight	Class IV	Absent		Absent
76	26	Group A	1	0	0		G1	1	8	5	Missed abortion	47	1.42	23.308867	normalweight	Class IV	Absent		Absent
77	26	Group A	1	0	0		G1	1	11	1	Inevitable abortion	46	1.54	19.396188	normalweight	Class IV	Absent		Absent
78	29	Group B	2	0	0	1	G2A1	2	10	2	Incomplete abortion	68	1.6	26.5625	preobesity	Class IV	Absent		Absent
79	22	Group B	3	0	0	2	G3A2	3	12	4	Missed abortion	63	1.55	26.222685	preobesity	Class III	Absent		Absent
80	26	Group A	1	0	0	1	G1	1	11	6	Missed abortion	58	1.45	27.586207	preobesity	Class IV	Absent		Absent
81	27	Group A	1	0	0		G1	1	7	0	Incomplete abortion	55	1.56	22.600263	normalweight	Class IV	Absent		Absent
82	25	Group A	1	0	0		G1	1	7	3	Missed abortion	52	1.46	24.394821	normalweight	Class III	Absent		Present
83	27	Group B	6	0	0	5	G6A5	> or = 4	8	5	Incomplete abortion	50	1.51	21.928863	normalweight	Class IV	Absent		Absent
84	23	Group A	1	0	0		G1	1	10	3	Incomplete abortion	48	1.5	21.333333	normalweight	Class III	Absent		Absent
85	30	Group A	1	0	0		G1	1	13	1	Incomplete abortion	54	1.52	23.372576	normalweight	Class IV	Absent		Absent

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Sl. No.	Fetusassessmenton USGanomalies	Cervicalin competence	Hbgdl	Urinecs	Cervicals wabcs	GTT	TSH	Thyroidstatus	Hormonal profile for PCOS nonpregnant state	beta 2 glycoprotein 1 antibody	Anticardiolipin antibody	Lupusanticoagulant	ProteinC	ProteinS	Causeofpregnancyloss	Known Unknown
43	Fetal anomalies absent	No	11.6	Sterile	Sterile	Normal	3.5	Normal	Normal	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
44	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	1.6	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
45	Fetal anomalies absent	No	12.1	Sterile	Sterile	Normal	1.1	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Unknown
46	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	1.56	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
47	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.12	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
48	Fetal anomalies absent	No	11	Sterile	Sterile	Abnormal	6.7	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
49	Fetal anomalies absent	No	11.7	Sterile	Sterile	Normal	1.4	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
50	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.5	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
51	Fetal anomalies absent	No	10.8	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
52	Fetal anomalies absent	No	10	Sterile	Sterile	Normal	4.03	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
53	Fetal anomalies absent	No	9.8	Sterile	Sterile	Abnormal	2.4	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
54	Fetal anomalies absent	No	11.2	Sterile	Sterile	Normal	2.2	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
55	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	2.5	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
56	Fetal anomalies absent	No	10.5	Sterile	Sterile	Normal	0.98	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
57	Fetal anomalies absent	No	7.5	Sterile	Sterile	Normal	1.5	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
58	Fetal anomalies absent	No	9.5	Sterile	Sterile	Normal	4.04	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
59	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.7	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
60	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	1.1	Normal	Not done	Negative	Negative	Negative	ND	Normal	Unexplained	Unknown
61	Fetal anomalies absent	No	12.4	Sterile	Sterile	Abnormal	7.8	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
62	Fetal anomalies absent	No	10.7	Sterile	Sterile	Abnormal	12.16	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
63	Fetal anomalies absent	No	11.1	Sterile	Sterile	Normal	8.9	Abnormal	Not done	ND	ND	ND	ND	ND	Uterine anomalies + Hypothyroid	Known
64	Fetal anomalies absent	No	12.8	Sterile	Sterile	Abnormal	3.19	Normal	Not done	ND	ND	ND	ND	ND	DIP + PCOS	Known
65	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	5.57	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
66	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	2.4	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
67	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
68	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	1.89	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
69	Fetal anomalies absent	No	8.8	Sterile	Sterile	Normal	2.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
70	Fetal anomalies absent	No	10.6	Sterile	Sterile	Abnormal	1.9	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
71	Fetal anomalies absent	No	10.3	Sterile	Sterile	Normal	1.1	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
72	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	2.8	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
73	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	2.1	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
74	Fetal anomalies absent	No	11.8	Sterile	Sterile	Normal	5.58	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
75	Fetal anomalies absent	No	11.4	Sterile	Sterile	Normal	1.2	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
76	Fetal anomalies absent	No	7	Sterile	Sterile	Normal	1.27	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
77	Fetal anomalies absent	No	12.4	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Positive	Negative	ND	ND	APLA- ACLA positive	Known
78	Fetal anomalies absent	No	10.5	Sterile	Sterile	Normal	5.42	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
79	Fetal anomalies absent	No	12	Sterile	D	Abnormal	1.1	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
80	Fetal anomalies absent	No	11.2	Sterile	Growth	Abnormal	3.3	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
81	Fetal anomalies absent	No	10.5	Sterile	Sterile	Normal	6.8	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
82	Fetal anomalies absent	No	10.1	Sterile	Sterile	Abnormal	1.5	Normal	Not done	ND	ND	ND	ND	ND	DIP + PCOS	Known
83	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	2.13	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
84	Fetal anomalies absent	No	9.7	Sterile	Sterile	Abnormal	3	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
85	Fetal anomalies absent	No	9.9	Sterile	Sterile	Abnormal	4.62	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known

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Sl. No.	Age	Group	Gravida	Para	Live	Abortion	Obstetriccode	Noofabortions	Gaat pregnancy loss weeks	Gaat pregnancy loss days	Typeofabortion	Weight	Height	BMI	WHOBMI	Socioeconomic status Kuppuswamy classification	Uterineanomalieson USG	Typeofanomaly	PCOSfeatures on USG
86	28	Group B	3	0	0	2	G3A2	3	9	4	Missed abortion	51	1.56	20.956607	normalweight	Class IV	Absent		Absent
87	27	Group A	1	0	0		G1	1	11	0	Incomplete abortion	50	1.49	22.521508	normalweight	Class III	Absent		Absent
88	21	Group A	2	1	0		G2P1L0	1	10	1	Missed abortion	50	1.46	23.456558	normalweight	Class IV	Absent		Absent
89	26	Group A	1	0	0		G1	1	11	4	Missed abortion	46	1.5	20.444444	normalweight	Class IV	Absent		Absent
90	34	Group B	2	0	0	1	G2A1	2	11	2	Missed abortion	48	1.55	19.979188	normalweight	Class IV	Absent		Absent
91	25	Group A	1	0	0		G1	1	12	2	Missed abortion	46	1.46	21.580034	normalweight	Class IV	Absent		Absent
92	33	Group B	2	0	0	1	G2A1	2	13	3	Incomplete abortion	48	1.53	20.504934	normalweight	Class III	Absent		Absent
93	23	Group B	2	0	0	1	G2A1	2	13	1	Missed abortion	43	1.42	21.325134	normalweight	Class IV	Absent		Absent
94	25	Group B	3	0	0	2	G3A2	3	9	5	Blighted ovum	44	1.48	20.087655	normalweight	Class IV	Absent		Absent
95	22	Group A	1	0	0		G1	1	10	0	Missed abortion	53	1.53	22.640865	normalweight	Class IV	Absent		Absent
96	20	Group B	2	0	0	1	G2A1	2	11	1	Inevitable abortion	48	1.46	22.518296	normalweight	Class IV	Absent		Absent
97	28	Group B	2	0	0	1	G2A1	2	12	3	Missed abortion	54	1.6	21.09375	normalweight	Class IV	Absent		Absent
98	20	Group A	1	0	0		G1	1	13	0	Blighted ovum	56	1.51	24.560326	normalweight	Class III	Absent		Absent
99	20	Group A	1	0	0		G1	1	11	6	Missed abortion	56	1.57	22.718974	normalweight	Class IV	Absent		Absent
100	29	Group A	1	0	0		G1	1	11	6	Missed abortion	52	1.54	21.926126	normalweight	Class IV	Absent		Absent
101	19	Group B	4	0	0	3	G4A3	> or = 4	10	2	Missed abortion	48	1.45	22.829964	normalweight	Class IV	Absent		Absent
102	22	Group A	1	0	0		G1	1	10	5	Missed abortion	55	1.56	22.600263	normalweight	Class IV	Absent		Absent
103	23	Group A	1	0	0		G1	1	11	4	Inevitable abortion	60	1.57	24.341758	normalweight	Class IV	Absent		Absent
104	26	Group B	2	0	0		G2A1	2	8	2	Missed abortion	61	1.49	27.47624	preobesity	Class IV	Absent		Absent
105	24	Group B	3	0	0		G3A2	3	6	2	Blighted ovum	64	1.55	26.638918	preobesity	Class III	Absent		Absent
106	23	Group B	2	0	0		G2A1	2	11	0	Incomplete abortion	66	1.48	30.131483	obesityclassI	Class III	Present	BIC	Present
107	21	Group A	1	0	0		G1	1	11	5	Missed abortion	58	1.55	24.141519	normalweight	Class IV	Absent		Absent
108	23	Group B	3	0	0	2	G3A2	3	9	2	Missed abortion	60	1.47	27.766209	preobesity	Class IV	Absent		Absent
109	25	Group A	#NULL!	0	0	1	A1	1	9	5	Complete abortion	45	1.55	18.730489	normalweight	Class IV	Absent		Absent
110	31	Group B	8	0	0	7	G8A7	> or = 4	10	1	Missed abortion	48	1.45	22.829964	normalweight	Class III	Absent		Absent
111	22	Group A	1	0	0		G1	1	13	0	Missed abortion	46	1.56	18.902038	normalweight	Class IV	Present	DIDEL	Absent
112	27	Group B	6	0	0		G6A5	> or = 4	12	0	Missed abortion	52	1.65	19.100092	normalweight	Class IV	Absent		Absent
113	28	Group B	2	0	0	1	G2A1	2	12	3	Blighted ovum	50	1.45	23.781213	normalweight	Class III	Absent		Absent
114	29	Group A	1	0	0		G1	1	4	1	Incomplete abortion	53	1.51	23.244595	normalweight	Class IV	Absent		Absent
115	25	Group A	1	0	0		G1	1	11	4	Incomplete abortion	56	1.46	26.271345	preobesity	Class IV	Absent		Absent
116	22	Group A	1	0	0		G1	1	11	1	Missed abortion	55	1.54	23.191095	normalweight	Class IV	Absent		Absent
117	19	Group B	3	0	0	2	G3A2	3	11	4	Incomplete abortion	52	1.48	23.739956	normalweight	Class IV	Absent		Absent
118	19	Group A	1	0	0		G1	1	12	4	Incomplete abortion	51	1.53	21.786492	normalweight	Class III	Present	BIC	Absent
119	21	Group B	3	0	0	2	G3A2	3	13	4	Missed abortion	65	1.51	28.507522	preobesity	Class IV	Absent		Absent
120	19	Group B	2	0	0	1	G2A1	2	12	2	Missed abortion	57	1.45	27.110583	preobesity	Class IV	Absent		Absent
121	22	Group B	2	0	0	1	G2A1	2	12	1	Missed abortion	60	1.47	27.766209	preobesity	Class IV	Absent		Absent
122	25	Group B	2	0	0	1	G2A1	2	11	4	Missed abortion	48	1.53	20.504934	normalweight	Class III	Absent		Absent
123	20	Group A	1	0	0		G1	1	6	2	Missed abortion	55	1.45	26.159334	preobesity	Class IV	Absent		Absent
124	25	Group B	3	0	0	2	G3A2	3	8	4	Inevitable abortion	56	1.49	25.224089	preobesity	Class III	Absent		Absent
125	20	Group A	1	0	0		G1	1	7	2	Missed abortion	50	1.55	20.811655	normalweight	Class III	Absent		Absent
126	35	Group B	17	0	0	16	G17A16	> or = 4	13	2	Missed abortion	44	1.44	21.219136	normalweight	Class III	Absent		Absent
127	19	Group B	2	0	0	1	G2A1	2	8	5	Missed abortion	41	1.43	20.04988	normalweight	Class IV	Absent		Absent
128	28	Group A	1	0	0		G1	1	9	1	Missed abortion	68	1.5	30.222222	obesityclassI	Class IV	Absent		Present

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Sl. No.	Fetusassessmenton USGanomalies	Cervicalin competence	Hbgdl	Urinecs	Cervicals wabcs	GTT	TSH	Thyroidstatus	Hormonal profile for PCOS nonpregnant state	beta 2 glycoprotein 1 antibody	Anticardiolipin antibody	Lupusanticoagulant	ProteinC	ProteinS	Causeofpregnancyloss	Known Unknown
86	Fetal anomalies absent	No	10.6	Sterile	Sterile	Abnormal	4.45	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
87	Fetal anomalies absent	No	10.8	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
88	Fetal anomalies absent	No	13	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
89	Fetal anomalies absent	No	12	Sterile	Sterile	Abnormal	3.2	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
90	Fetal anomalies absent	No	12.8	Sterile	Sterile	Abnormal	0.55	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
91	Fetal anomalies absent	No	10.1	Sterile	Sterile	Normal	2.09	Normal	Not done	Negative	Positive	Negative	Normal	Normal	APLA- ACLA positive	Known
92	Fetal anomalies absent	No	8.4	Sterile	Sterile	Normal	1.65	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
93	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
94	Fetal anomalies absent	No	12.1	Sterile	Sterile	Abnormal	5.54	Abnormal	Not done	Negative	Negative	Negative	ND	ND	GDM + Hypothyroid	Known
95	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.67	Normal	Not done	Negative	Negative	Negative	ND	Normal	Unexplained	Unknown
96	Fetal anomalies absent	No	11.4	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
97	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	2.2	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
98	Fetal anomalies absent	No	8.9	Sterile	Sterile	Normal	2.45	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
99	Fetal anomalies absent	No	10	Sterile	Sterile	Abnormal	2.9	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
100	Fetal anomalies absent	No	9.2	Sterile	Sterile	Normal	4.25	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
101	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	2.1	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
102	Fetal anomalies absent	No	11	Sterile	Sterile	Abnormal	3.22	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
103	Fetal anomalies absent	No	7.8	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Positive	Normal	Deficient	LAC positive + protein S deficiency	Known
104	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	1.1	Abnormal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
105	Fetal anomalies absent	No	11.2	Sterile	Sterile	Abnormal	0.32	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
106	Fetal anomalies absent	No	12.9	Sterile	Sterile	Normal	0.76	Normal	Not done	ND	ND	ND	ND	ND	Uterine anomalies + PCOS	Known
107	Fetal anomalies absent	No	11	Sterile	Sterile	Abnormal	2.48	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
108	Fetal anomalies absent	No	9.5	Sterile	Sterile	Normal	0.98	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
109	Fetal anomalies absent	No	12.9	Sterile	Sterile	Normal	1.25	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
110	Fetal anomalies absent	No	11.7	Sterile	Sterile	Abnormal	4.1	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
111	Fetal anomalies absent	No	7.8	Sterile	Sterile	Normal	1.71	Normal	Not done	ND	ND	ND	ND	ND	Uterine anomalies	Known
112	Fetal anomalies absent	No	12	Sterile	Sterile	Normal	1.6	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
113	Fetal anomalies absent	No	11.5	Sterile	Sterile	Normal	0.64	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
114	Fetal anomalies absent	No	11.5	Sterile	Sterile	Normal	4.9	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
115	Fetal anomalies absent	No	10.6	Sterile	Sterile	Abnormal	2.4	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
116	Fetal anomalies absent	No	10.6	Sterile	Sterile	Abnormal	2	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
117	Fetal anomalies absent	No	10.5	Sterile	Sterile	Normal	0.76	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
118	Fetal anomalies absent	No	11.3	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	ND	ND	Uterine anomalies	Known
119	Fetal anomalies absent	No	9.2	Sterile	Sterile	Normal	3.49	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
120	Fetal anomalies absent	No	5.3	2 (E Coli)	Sterile	Normal	2.7	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
121	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	1.3	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
122	Fetal anomalies absent	No	10.8	Sterile	Growth	Abnormal	3.2	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
123	Fetal anomalies absent	No	8.6	Sterile	Sterile	Abnormal	1.8	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
124	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
125	Fetal anomalies absent	No	8.2	Sterile	Sterile	Abnormal	5.1	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
126	Fetal anomalies absent	No	8.9	Sterile	Sterile	Normal	4.8	Abnormal	Not done	ND	ND	Positive	ND	ND	LAC + Hypothyroid	Known
127	Fetal anomalies absent	No	10.8	Sterile	Sterile	Normal	1.1	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
128	Fetal anomalies absent	No	11	Sterile	Sterile	Abnormal	0.9	Normal	Not done	ND	ND	ND	ND	ND	DIP + PCOS	Known

Sl. No.	Age	Group	Gravida	Para	Live	Abortion	Obstetriccode	Noofabortions	Gaat pregnancy loss weeks	Gaat pregnancy loss days	Typeofabortion	Weight	Height	BMI	WHOBMI	Socioeconomic status Kuppuswamy classification	Uterineano malieson USG	Typeofanomaly	PCOSfeatures on USG
129	26	Group A	1	0	0		G1	1	7	6	Missed abortion	62	1.55	25.806452	preobesity	Class IV	Absent		Absent
130	30	Group A	1	0	0		G1	1	10	6	Incomplete abortion	58	1.55	24.141519	normalweight	Class III	Absent		Present
131	26	Group B	2	0	0		G2A1	2	8	0	Missed abortion	56	1.35	30.727023	obesityclassI	Class IV	Absent		Absent
132	32	Group A	1	0	0		G1	1	9	1	Inevitable abortion	43	1.45	20.451843	normalweight	Class IV	Absent		Absent
133	26	Group B	3	0	0		G3A2	3	12	6	Blighted ovum	45	1.51	19.735976	normalweight	Class IV	Absent		Absent
134	35	Group B	9	1	0	7	G9P1L0A7	> or = 4	13	5	Inevitable abortion	46	1.49	20.719787	normalweight	Class IV	Absent		Absent
135	29	Group A	1	0	0		G1	1	8	4	Missed abortion	52	1.54	21.926126	normalweight	Class III	Absent		Absent
136	23	Group B	3	0	0	2	G3A2	3	10	0	Missed abortion	52	1.55	21.644121	normalweight	Class IV	Absent		Absent
137	35	Group A	1	0	0		G1	1	10	1	Missed abortion	48	1.47	22.212967	normalweight	Class IV	Absent		Absent
138	25	Group A	1	0	0		G1	1	12	1	Missed abortion	51	1.48	23.283419	normalweight	Class II	Absent		Absent
139	28	Group A	1	0	0		G1	1	8	5	Incomplete abortion	47	1.5	20.888889	normalweight	Class IV	Absent		Absent
140	25	Group A	1	0	0		G1	1	11	1	Missed abortion	45	1.51	19.735976	normalweight	Class III	Absent	FIBROID	Absent
141	26	Group B	2	0	0	1	G2A1	2	8	1	Missed abortion	48	1.49	21.620648	normalweight	Class IV	Absent		Absent
142	27	Group A	1	0	0		G	1	10	3	Incomplete abortion	52	1.55	21.644121	normalweight	Class IV	Absent		Absent
143	18	Group A	1	0	0		G1	1	11	4	Missed abortion	45	1.46	21.110903	normalweight	Class IV	Absent		Absent
144	27	Group A	1	0	0		G1	1	12	4	Missed abortion	47	1.52	20.342798	normalweight	Class IV	Absent		Absent
145	19	Group B	2	0	0	1	G2A1	2	11	2	Missed abortion	53	1.55	22.060354	normalweight	Class IV	Absent		Absent
146	26	Group B	4	0	0	3	G4A3	> or = 4	13	4	Blighted ovum	47	1.48	21.457268	normalweight	Class III	Absent		Absent
147	29	Group B	4	0	0	3	G4A3	> or = 4	10	5	Missed abortion	58	1.62	22.10029	normalweight	Class IV	Absent		Absent
148	26	Group A	1	0	0		G1	1	13	3	Blighted ovum	55	1.52	23.805402	normalweight	Class IV	Absent		Absent
149	22	Group A	1	0	0		G1	1	8	4	Missed abortion	47	1.53	20.077748	normalweight	Class IV	Absent		Absent
150	24	Group A	1	0	0		G1	1	9	1	Missed abortion	49	1.56	20.13478	normalweight	Class IV	Absent		Absent
151	23	Group B	2	0	0	1	G2A1	2	11	0	Missed abortion	52	1.71	17.78325	underweight	Class IV	Absent		Absent
152	21	Group B	2	0	0	1	G2A1	2	12	4	Missed abortion	55	1.56	22.600263	normalweight	Class III	Absent		Absent
153	35	Group B	8	1	0	6	G8P1L0A6	> or = 4	12	6	Incomplete abortion	60	1.54	25.299376	preobesity	Class IV	Absent		Absent
154	23	Group A	#NULL!	0	0	1	A1	1	6	2	Complete abortion	61	1.69	21.357796	normalweight	Class IV	Absent		Absent
155	23	Group A	1	0	0		G1	1	12	4	Missed abortion	57	1.57	23.12467	normalweight	Class IV	Absent		Absent
156	22	Group A	1	0	0		G1	1	13	2	Missed abortion	58	1.62	22.10029	normalweight	Class III	Absent		Present
157	24	Group A	1	0	0		G1	1	9	2	Missed abortion	62	1.44	29.899691	preobesity	Class IV	Absent		Absent
158	22	Group A	1	0	0		G1	1	12	1	Missed abortion	48	1.53	20.504934	normalweight	Class III	Absent		Absent
159	23	Group B	2	0	0	1	G2A1	2	9	2	Missed abortion	46	1.45	21.878716	normalweight	Class III	Absent		Absent
160	35	Group B	4	0	0	3	G4A3	> or = 4	10	2	Missed abortion	48	1.47	22.212967	normalweight	Class IV	Absent		Absent
161	27	Group A	1	0	0		G1	1	9	6	Missed abortion	50	1.58	20.028842	normalweight	Class IV	Absent		Absent
162	22	Group A	1	0	0		G1	1	9	2	Missed abortion	53	1.57	21.501886	normalweight	Class IV	Absent		Absent
163	26	Group A	1	0	0		G1	1	8	3	Incomplete abortion	47	1.59	18.591037	normalweight	Class IV	Absent		Present
164	25	Group A	1	0	0		G1	1	15	3	Missed abortion	54	1.73	18.042701	underweight	Class IV	Absent		Absent
165	28	Group B	3	0	0	2	G3A2	3	13	4	Complete abortion	57	1.55	23.725286	normalweight	Class III	Absent		Present
166	32	Group B	2	0	0	1	G2A1	2	13	2	Inevitable abortion	55	1.48	25.109569	preobesity	Class IV	Absent		Absent
167	27	Group B	5	0	0		G5A4	> or = 4	8	2	Missed abortion	48	1.53	20.504934	normalweight	Class IV	Absent		Absent
168	25	Group A	1	0	0		G1	1	13	2	Incomplete abortion	64	1.48	29.218408	preobesity	Class III	Absent		Absent

Sl. No.	Fetusassessmenton USGanomalies	Cervicalin competence	Hbgdl	Urinecs	Cervicals wabcs	GTT	TSH	Thyroidstatus	Hormonal profile for PCOS nonpregnant state	beta 2 glycoprotein 1 antibody	Anticardiolipin antibody	Lupusanticoagulant	ProteinC	ProteinS	Causeofpregnancyloss	Known Unknown
129	Fetal anomalies absent	No	10.1	Sterile	Sterile	Normal	3	Normal	Not done	Negative	Positive	Positive	Normal	Normal	APLA- ACLA + LAC positive	Known
130	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.06	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
131	Fetal anomalies absent	No	10.3	Sterile	Sterile	Normal	0.98	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
132	Fetal anomalies absent	No	10.3	Sterile	Sterile	Abnormal	4.04	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
133	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
134	Fetal anomalies absent	No	10	Sterile	Sterile	Abnormal	0.93	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
135	Fetal anomalies absent	No	9.2	Sterile	Sterile	Normal	0.78	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
136	Fetal anomalies absent	No	13.1	Sterile	Sterile	Normal	2.44	Normal	Not done	Negative	Positive	Negative	Normal	Normal	APLA- ACLA positive	Known
137	Fetal anomalies absent	No	12	Sterile	Growth	Abnormal	1.5	Normal	Not done	ND	ND	ND	ND	ND	GDM + cervicovaginal Infections	Known
138	Fetal anomalies absent	No	9.3	Sterile	Sterile	Normal	1.7	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
139	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
140	Fetal anomalies absent	No	10.1	Sterile	Sterile	Normal	0.7	Normal	Not done	ND	ND	ND	ND	ND	Fibroid	Known
141	Fetal anomalies absent	No	9.6	Sterile	Sterile	Abnormal	2.38	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
142	Fetal anomalies absent	No	8.6	Sterile	Sterile	Normal	1.86	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
143	Fetal anomalies absent	No	10.1	Sterile	Sterile	Abnormal	2.73	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
144	Fetal anomalies absent	No	12.5	Sterile	Sterile	Normal	2.08	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
145	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	1.3	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
146	Fetal anomalies absent	No	11	Sterile	Growth	Normal	1.1	Normal	Not done	ND	ND	ND	ND	ND	Infection	Known
147	Fetal anomalies absent	No	11.2	Sterile	Sterile	Abnormal	1.14	Normal	Not done	Negative	Negative	Negative	ND	ND	DIP	Known
148	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	0.76	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
149	Fetal anomalies absent	No	11.3	Sterile	Sterile	Abnormal	1.08	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
150	Fetal anomalies absent	No	10.7	Sterile	Sterile	Abnormal	1.56	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
151	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.1	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
152	Fetal anomalies absent	No	10.9	Sterile	Sterile	Abnormal	5.3	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
153	Fetal anomalies absent	Yes	12.3	Sterile	Sterile	Normal	1.98	Normal	Not done	Negative	Negative	Negative	ND	ND	Cervical incompetence	Known
154	Fetal anomalies absent	No	9.6	Sterile	Sterile	Normal	2.3	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
155	Fetal anomalies absent	No	11.4	Sterile	Sterile	Normal	1.23	Normal	Not done	Negative	Negative	Positive	Normal	Normal	LAC positive	Known
156	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	1.14	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
157	Fetal anomalies absent	No	12.8	Sterile	Sterile	Abnormal	1.9	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
158	Fetal anomalies absent	No	11.4	Sterile	Growth	Normal	1.14	Normal	Not done	ND	ND	ND	ND	ND	Infection	Unknown
159	Fetal anomalies absent	No	12	Sterile	Sterile	Abnormal	6.2	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
160	Fetal anomalies absent	No	10.3	Sterile	Growth	Abnormal	1.64	Normal	Not done	Negative	Negative	Negative	ND	ND	GDM + cervicovaginal Infections	Known
161	Fetal anomalies absent	No	11.6	Sterile	Sterile	Normal	7.8	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
162	Fetal anomalies absent	No	13.1	Sterile	Sterile	Normal	2.61	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
163	Fetal anomalies absent	No	11.4	Sterile	Sterile	Normal	1.65	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
164	Fetal anomalies absent	No	12.4	Sterile	Sterile	Abnormal	1.79	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
165	Fetal anomalies absent	Yes	10.5	Sterile	Sterile	Normal	2.2	Normal	Not done	ND	ND	ND	ND	ND	Cervical incompetence + Hypothyroid + PCOS	Known
166	Fetal anomalies absent	No	11.9	Sterile	Sterile	Abnormal	1.16	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
167	Fetal anomalies absent	No	11.6	Sterile	Sterile	Normal	3.01	Normal	Not done	Negative	Positive	Negative	ND	ND	APLA- ACLA positive	Known
168	Fetal anomalies absent	No	10.4	Sterile	Sterile	Normal	0.89	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown

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Sl. No.	Age	Group	Gravida	Para	Live	Abortion	Obstetriccode	Noofabortions	Gaat pregnancy loss weeks	Gaat pregnancy loss days	Typeofabortion	Weight	Height	BMI	WHOBMI	Socioeconomic status Kuppuswamy classification	Uterineanomalieson USG	Typeofanomaly	PCOSfeatures on USG
169	23	Group A	1	0	0		G1	1	13	4	Missed abortion	56	1.47	25.915128	preobesity	Class IV	Absent		Absent
170	24	Group A	1	0	0		G1	1	11	2	Blighted ovum	53	1.45	25.208086	preobesity	Class IV	Absent		Absent
171	24	Group B	2	0	0	1	G2A1	2	10	1	Blighted ovum	49	1.47	22.675737	normalweight	Class III	Absent		Absent
172	20	Group A	1	0	0		G1	1	13	1	Missed abortion	51	1.65	18.732782	normalweight	Class IV	Absent		Absent
173	24	Group A	1	0	0		G1	1	10	6	Missed abortion	53	1.55	22.060354	normalweight	Class IV	Absent		Absent
174	32	Group B	13	0	0	12	G13A12	> or = 4	10	1	Missed abortion	49	1.65	17.998163	underweight	Class III	Absent		Absent
175	25	Group A	1	0	0		G1	1	12	5	Missed abortion	52	1.46	24.394821	normalweight	Class IV	Absent		Absent
176	34	Group B	2	0	0	1	G2A1	2	7	1	Incomplete abortion	48	1.54	20.239501	normalweight	Class IV	Absent		Absent
177	26	Group A	1	0	0		G1	1	8	1	Missed abortion	65	1.58	26.037494	preobesity	Class III	Absent		Absent
178	35	Group B	2	0	0	1	G2A1	2	12	3	Missed abortion	61	1.55	25.390219	preobesity	Class IV	Absent		Absent
179	26	Group B	4	0	0	3	G4A3	> or = 4	9	2	Missed abortion	55	1.46	25.802214	preobesity	Class IV	Absent		Absent
180	35	Group A	1	0	0		G1	1	13	2	Missed abortion	53	1.54	22.347782	normalweight	Class III	Absent		Absent
181	22	Group B	3	0	0	2	G3A2	3	12	2	Missed abortion	49	1.46	22.987427	normalweight	Class IV	Absent		Absent
182	32	Group B	3	0	0	2	G3A2	3	10	2	Incomplete abortion	48	1.51	21.051708	normalweight	Class IV	Absent		Absent
183	25	Group B	#NULL!	0	0	2	A2	2	9	2	Blighted ovum	52	1.52	22.506925	normalweight	Class IV	Present	SEP	Absent
184	26	Group A	1	0	0		G1	1	11	3	Complete abortion	56	1.48	25.566107	preobesity	Class IV	Absent		Present
185	30	Group A	1	0	0		G1	1	10	3	Blighted ovum	51	1.56	20.956607	normalweight	Class III	Absent		Absent
186	24	Group A	1	0	0		G1	1	10	4	Missed abortion	49	1.49	22.071078	normalweight	Class IV	Absent		Present
187	34	Group B	2	0	0		G2A1	2	9	4	Missed abortion	56	1.45	26.634958	preobesity	Class IV	Absent		Absent
188	23	Group B	3	0	0		G3A2	3	9	2	Inevitable abortion	48	1.52	20.775623	normalweight	Class IV	Absent		Absent
189	25	Group B	2	0	0	1	G2A1	2	12	4	Blighted ovum	50	1.55	20.811655	normalweight	Class III	Absent		Absent
190	26	Group B	2	0	0	1	G2A1	2	6	2	Missed abortion	48	1.48	21.913806	normalweight	Class IV	Absent		Absent
191	32	Group A	1	0	0		G1	1	13	5	Missed abortion	52	1.53	22.213678	normalweight	Class III	Absent		Absent
192	22	Group B	2	0	0	1	G2A1	2	7	4	Incomplete abortion	44	1.47	20.361886	normalweight	Class IV	Absent		Absent
193	21	Group B	2	0	0	1	G2A1	2	11	1	Missed abortion	46	1.48	21.00073	normalweight	Class III	Absent		Absent
194	27	Group A	1	0	0		G1	1	10	2	Blighted ovum	54	1.57	21.907582	normalweight	Class IV	Absent		Absent
195	27	Group A	2	1	0		G2P1L0	1	11	2	Incomplete abortion	50	1.46	23.456558	normalweight	Class IV	Absent		Absent
196	20	Group A	1	0	0		G1	1	11	5	Missed abortion	52	1.61	20.060954	normalweight	Class III	Absent		Absent
197	20	Group B	2	0	0	1	G2A1	2	9	6	Missed abortion	56	1.51	24.560326	normalweight	Class II	Absent		Absent
198	34	Group B	6	0	0	5	G6A5	> or = 4	8	4	Missed abortion	49	1.57	19.879103	normalweight	Class III	Absent		Absent
199	21	Group B	3	0	0	2	G3A2	3	6	6	Missed abortion	57	1.53	24.349609	normalweight	Class IV	Absent		Absent
200	24	Group B	3	0	0	2	G3A2	3	9	3	Missed abortion	60	1.45	28.537455	preobesity	Class IV	Present	BIC	Absent
201	25	Group B	2	0	0	1	G2A1	2	5	3	Blighted ovum	49	1.56	20.13478	normalweight	Class III	Absent		Absent
202	21	Group B	2	0	0	1	G2A1	2	9	2	Missed abortion	52	1.47	24.064047	normalweight	Class IV	Absent		Absent
203	29	Group B	2	0	0	1	G2A1	2	10	1	Missed abortion	48	1.51	21.051708	normalweight	Class III	Absent		Absent
204	24	Group B	5	0	0	4	G5A4	> or = 4	6	2	Blighted ovum	52	1.57	21.096191	normalweight	Class III	Absent		Absent
205	25	Group B	3	0	0	2	G3A2	3	9	2	Incomplete abortion	55	1.55	22.89282	normalweight	Class II	Absent		Absent
206	29	Group B	2	0	0	1	G2A1	2	10	2	Missed abortion	48	1.56	19.723866	normalweight	Class IV	Absent		Absent
207	23	Group B	2	0	0	1	G2A1	2	12	6	Inevitable abortion	52	1.49	23.422368	normalweight	Class III	Absent		Absent
208	28	Group B	2	0	0	1	G2A1	2	9	2	Missed abortion	57	1.46	26.740477	preobesity	Class IV	Absent		Absent
209	25	Group B	4	1	0	2	G4P1L0A2	3	10	4	Blighted ovum	50	1.52	21.641274	normalweight	Class III	Absent		Absent
210	27	Group B	3	0	0	2	G3A2	3	9	1	Missed abortion	53	1.49	23.872799	normalweight	Class III	Absent		Absent

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Sl. No.	Fetusassessmenton USGanomalies	Cervicalin competence	Hbgdl	Urinecs	Cervicals wabcs	GTT	TSH	Thyroidstatus	Hormonal profile for PCOS nonpregnant state	beta 2 glycoprotein 1 antibody	Anticardiolipin antibody	Lupusanticoagulant	ProteinC	ProteinS	Causeofpregnancyloss	Known Unknown
169	Fetal anomalies absent	No	11.5	Sterile	Sterile	Normal	1.54	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
170	Fetal anomalies absent	No	10.1	Sterile	Sterile	Normal	1.21	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Known
171	Fetal anomalies absent	No	10.5	Sterile	Sterile	Abnormal	4.2	Abnormal	Not done	ND	ND	ND	ND	ND	GDM + Hypothyroid	Known
172	Fetal anomalies absent	No	9.5	Sterile	Sterile	Normal	2.75	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
173	Fetal anomalies absent	No	11.2	Sterile	Sterile	Normal	1.9	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
174	Fetal anomalies absent	No	9.9	Sterile	Sterile	Normal	1.33	Normal	Not done	Positive	Positive	Positive	Normal	Deficient	ACLA positive + protein S deficiency	Known
175	Fetal anomalies absent	No	12.8	Sterile	Sterile	Abnormal	0.76	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
176	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.21	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
177	Fetal anomalies absent	No	11.1	Sterile	Sterile	Abnormal	2.69	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
178	Fetal anomalies absent	No	9.5	Sterile	Sterile	Abnormal	2.18	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
179	Fetal anomalies absent	Yes	9.6	Sterile	Sterile	Abnormal	1.2	Normal	Not done	ND	ND	ND	ND	ND	cervical incompetence + GDM	Known
180	Fetal anomalies absent	No	12.9	Sterile	Sterile	Abnormal	4.01	Abnormal	Not done	ND	ND	ND	ND	ND	Overt DM + Hypothyroid	Known
181	Fetal anomalies absent	No	10.2	Sterile	Sterile	Normal	6.1	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
182	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	4.6	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
183	Fetal anomalies absent	No	9.1	Sterile	Sterile	Normal	4.78	Abnormal	Not done	ND	ND	ND	ND	ND	Uterine anomalies + Hypothyroid	Known
184	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	2.5	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
185	Fetal anomalies absent	No	10.7	Sterile	Sterile	Normal	1.7	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
186	Fetal anomalies absent	No	11.4	Sterile	Sterile	Normal	2.3	Normal	Not done	ND	ND	ND	ND	ND	PCOS	Known
187	Fetal anomalies absent	No	10.2	Sterile	Sterile	Abnormal	2.6	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
188	Fetal anomalies absent	No	11.2	Sterile	Sterile	Normal	1.17	Normal	Not done	Negative	Negative	Negative	Normal	Deficient	Protein S deficiency	Known
189	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	3.1	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
190	Fetal anomalies absent	No	8	Sterile	Sterile	Abnormal	3.37	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
191	Fetal anomalies absent	No	10.2	Sterile	Sterile	Normal	6.87	Abnormal	Not done	ND	ND	ND	ND	ND	Hypothyroid	Known
192	Fetal anomalies absent	No	12.1	Sterile	Sterile	Normal	1.23	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
193	Fetal anomalies absent	No	10	Sterile	Sterile	Abnormal	2.08	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
194	Fetal anomalies absent	No	11.1	Sterile	Sterile	Abnormal	3.02	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
195	Fetal anomalies absent	No	9	Sterile	Sterile	Normal	3.52	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
196	Fetal anomalies absent	No	12	Sterile	Sterile	Abnormal	1.21	Normal	Not done	ND	ND	ND	ND	ND	DIP	Known
197	Fetal anomalies absent	No	10.4	Sterile	Sterile	Abnormal	2.3	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
198	Fetal anomalies absent	No	8.9	Sterile	Sterile	Normal	2.43	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
199	Fetal anomalies absent	No	10.7	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
200	Fetal anomalies absent	No	9.4	Sterile	Sterile	Normal	1.9	Normal	Not done	ND	ND	ND	ND	ND	Uterine anomalies	Known
201	Fetal anomalies absent	No	12	Sterile	Sterile	Abnormal	0.78	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
202	Fetal anomalies absent	No	8.6	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	ND	ND	Unexplained	Unknown
203	Fetal anomalies absent	No	12	Sterile	Sterile	Abnormal	0.9	Normal	Not done	ND	ND	ND	ND	ND	GDM	Known
204	Fetal anomalies absent	No	11	Sterile	Sterile	Normal	1.07	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
205	Fetal anomalies absent	No	10.6	Sterile	Sterile	Normal	2.4	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
206	Fetal anomalies absent	No	9.7	Sterile	Sterile	Normal	2.2	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
207	Fetal anomalies absent	No	10.8	Sterile	Sterile	Normal	1.8	Normal	Not done	Negative	Negative	Negative	Normal	Normal	Unexplained	Unknown
208	Fetal anomalies absent	No	13	Sterile	Sterile	Normal	6.5	Abnormal	Not done	ND	Negative	ND	ND	ND	Hypothyroid	Known
209	Fetal anomalies absent	No	9.5	Sterile	Sterile	Normal	0.65	Normal	Not done	Negative	Negative	Positive	Normal	Normal	LAC positive	Known
210	Fetal anomalies absent	No	10.1	Sterile	Sterile	Normal	1.7	Normal	Not done	Negative	Negative	Positive	Normal	Normal	LAC positive	Known