A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE CENTRE

DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

in partial fulfilment of

the requirements for the degree of

M.S., OBSTETRICS AND GYNAECOLOGY

BRANCH - VI

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI

APRIL-2015

BONAFIDE CERTIFICATE

This is to certify that this Dissertation entitled "**A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE CENTRE**" is the bonafide original work of **Dr.GOMATHI.M**, during the period of her Post graduate study from 2012 – 2015, under my guidance and supervision, in the Department of Obstetrics and Gynaecology, Tirunelveli Medical College & Hospital, Tirunelveli, in partial fulfilment of the requirement for M.S., Obstetrics and Gynaecology (Branch VI) examination of the Tamil Nadu Dr.M.G.R Medical University will be held in April 2015.

The DEAN
Tirunelveli Medical College,
Tirunelveli - 627011.

CERTIFICATE

I hereby certify that this dissertation entitled **"A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE CENTRE"** is a record of work done by **Dr.GOMATHI.M,** in the Department of Obstetrics and Gynaecology, Tirunelveli Medical College, Tirunelveli, during her postgraduate degree course period from 2012- 2015. This work has not formed the basis for previous award of any degree.

GUIDE

Dept. of Obstetrics & Gynaecology,
Tirunelveli Medical College,
Tirunelveli -627011

PROFESSOR & HOD

Dept. of Obstetrics & Gynaecology,
Tirunelveli Medical College,
Tirunelveli -627011

DECLARATION

I solemnly declare that this dissertation titled **"A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE CENTRE"** submitted by me for the degree of M.S., Obstetrics and Gynaecology is the record work carried out by me during the period of 2012-2015 under the guidance of **Dr.R. THAMIL KOTHAI, M.D., O.G.,** Asst. Professor, Department of Obstetrics and Gynaecology, Tirunelveli Medical College, Tirunelveli. The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, towards the partial fulfilment of requirements for the award of M.S., Obstetrics and Gynaecology Degree (Branch VI) examination to be held in April 2015.

Place: Tirunelveli
Date:

Dr.GOMATHI.M,
Post Graduate
M.S., Obstetrics and Gynaecology,
Dept. of Obstetrics & Gynaecology,
Tirunelveli MedicalCollege,
Tirunelveli-11.

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI,

STATE OF TAMILNADU, INDIA

PIN CODE:627011

Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944

Estd:1965

Under the Directorate of Medical Education, Government of Tamilnadu.

Institutional Ethical Committee

Certificate of Approval

This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr.M.Gomathi, Post Graduate in O&G, Department of Obstetrics & Gynaecology, Tirunelveli Medical College /Hospital, Tirunelveli titled **"A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE CENTRE"** registered by the IEC as 336/O&G/IEC/2013 dated. 13.03.2013. The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.

Issued on this Date

13.03.2013

Under Seal

TIRUNELVELI MEDICAL COLLEGE TAMILNADU INDIA TIRUNELVELI-11

Secretary

Secretary,
Ethical Committee,
Tirunelveli Medical College,
Tirunelveli-11.


A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A TERTIARY CARE
14%
A DESCRIPTIVE STUDY ON ECTOPIC PREGNANCY IN A
TERTIARY CARE CENTRE
CHENNAI
M.S., OBSTETRICS AND GYNAECOLOGY
BRANCH - II
Match Overview
1 3%
2 2%
3 1%
4 1%
5 1%
6 1%
7 <1%
8 <1%

ACKNOWLEDGEMENT

I express my sincere thanks to **Prof.LD.THULASIRAM, M.S.**, **Ortho.**, Dean, Tirunelveli Medical College Hospital, for allowing me to do this study on ectopic pregnancy .

I take this opportunity to express my heartfelt sincere gratitude to **Dr.M.MEENA, M.D.,D.G.O.,DNB.,** Professor and Head of the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli for her keen interest, encouragement, suggestions and guidance during the study.

I am extremely thankful to **Dr. RAMALAKSHMI,M.D.,D.G.O.,** Associate Professor, Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital for her advice, constant encouragement and valuable suggestions and guidance throughout the study.

I am truly thankful to **Dr.SHEBA ROSATTE VICTOR, M.D., O.G.,** Associate Professor, Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital for her constant cheer and support throughout the study.

I take this opportunity to express my heartfelt thanks to my guide **Dr.R.THAMIL KOTHAI , M.D.,O.G.,** Assistant Professor and Registrar of Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital for her expert guidance, opinions and encouragement throughout the study.

My thanks to **Dr.NELLAIAPPAN, M.D., RD.,** Professor and Head of the Department of Radiology, Tirunelveli Medical College Hospital for his support during the study.

I convey my thanks to **Dr.SITHY ATHIYA MUNAVARAH, M.D.,** Professor of Pathology, Department of Pathology, Tirunelveli Medical College Hospital for her support during the course of study.

I express my sincere thanks to all my Assistant Professors for their help and suggestions during the study.

I am thankful to all my colleagues, friends and staff of the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital for all their help and support they extended for the successful completion of this dissertation.

I would like to express my gratitude to all my patients and I pray for their longevity.

I also like to thank my parents and my sister for their love, support and encouragement.

ABBREVIATIONS

ART	-	Artificial Reproductive Techniques
PID	-	Pelvic Inflammatory Disease
β hCG	-	Beta human Chorionic Gonadotropin
USG	-	Ultrasound
YRS	-	Years
Hrs	-	Hours
PROKR 1	-	Prokineticin Receptor 1
GIFT	-	Gamete Intra Fallopian Transfer
ZIFT	-	Zygote Intra Fallopian Transfer
IVF	-	In Vitro Fertilization
IVF –ET	-	In Vitro Fertilization Embryo Transfer
IUCD	-	Intra Uterine Contraceptive Device
LNG	-	Levonorgestrel
EC	-	Emergency Contraception
LH	-	Leutinising Hormone
TSH	-	Thyroid Stimulating Hormone
mIU	-	million International Unit
UPT	-	Urine Pregnancy Test
TVS	-	Transvaginal Sonography
TAS	-	Transabdominal Sonography
PUL	-	Pregnancy of Unknown Location

CT	-	Computed Tomography
MRI	-	Magnetic Resonance Imaging
IGFBP 1	-	Insulin Like Growth Factor Basic Protein 1
POD	-	Pouch of Douglas
HSG	-	Hysterosalphingography
MTX	-	Methotrexate
ASRM	-	American Society of Reproductive Medicine
TVMCH	-	Tirunelveli Medical College Hospital
Gynaec	-	Gynaecology
LMP	-	Last Menstrual Period
NK	-	Not Known
NA	-	Not Applicable
ND	-	Not Done
ST	-	Sterilisation
TAT	-	Total Abdominal Tubectomy
PS	-	Puerperal Sterilisation
LS	-	Laparoscopic Sterilisation
OCP	-	Oral Contraceptive Pills
POP	-	Progesterone Only Pills
LAM	-	Lactational Amenorrhoea
Em. Pills	-	Emergency contraceptive pills
LSCS	-	Lower Segment Caesarean Section

H/O	-	History of
Prev	-	Previous
MTP	-	Medical Termination of Pregnancy
Hb	-	Haemoglobin
IMCU	-	Intensive Medical Care Unit
Gen.surgery	-	General Surgery
e/o	-	evidence of
B/L	-	Bilateral
Rt	-	Right
Lt	-	Left
RSO	-	Right Salphingo Oophorectomy
LSO	-	Left Salphingo Oophorectomy
D & C	-	Dilatation and Curettage
Chr. R	-	Chronic Rupture
R	-	Ruptured
UR	-	Unruptured
WB	-	Whole Blood
PCV	-	Packed Cell Volume
FFP	-	Fresh Frozen Plasma
PLTS	-	Platelets
ICU	-	Intensive Care Unit
Vent.support	-	Ventilatory support

HPE	-	Histopathological Examination
Ameno	-	Amenorrhoea
Pain	-	Abdominal pain
Bleed p/v	-	Bleeding per vaginum
Class.triad	-	Classical Triad
Abd	-	Abdomen
f.fullness	-	forniceal fullness
f.tenderness	-	forniceal tenderness
adnex. mass	-	adnexal mass
empty ut	-	empty uterus
PPV	-	Positive Predictive Value
NPV	-	Negative Predictive Value
O & G	-	Obstetrics and Gynaecology
P/V	-	Per Vaginum

CONTENTS

INTRODUCTION

Ectopic Pregnancy is originated from ‘Ektopos’, a Greek word. In ectopic pregnancy the fertilized ovum implants outside the endometrial cavity of the uterus [1]. The most common extra uterine pregnancy is the tubal pregnancy in which a fertilized ovum implants in the fallopian tubes. Tubal pregnancies account for 98% of all ectopic gestations. Other sites like ovary, cervix, horn of the uterus, caesarean scar and abdominal cavity are rare [2].

Incidence of ectopic pregnancy varies among different countries and also within the same country from place to place. The incidence is around 0.3 - 0.5% of deliveries in the U.S. and U.K, 4% of deliveries in Ghana and 2.1% of deliveries in Nigeria. [3,4,5].

In Shraddha Shetty K et al Study in India, the incidence was 5.6 / 1000 deliveries[6]. In Rashmi Gaddagi et al study the incidence was 1:399 pregnancies [7]. In a study conducted by Porwal Sanjay et al, the incidence was 2.46 / 1000 deliveries [8].

In current century, the developed countries are facing with four fold rise in the occurrence of ectopic pregnancy (from 0.3 to 1.2%) [9,10]. This rise is mainly because of advanced techniques for diagnosing the ectopic pregnancy in earlier stage and increased prevalence of ART & PID [11] [12].

Ectopic pregnancy is a gynaecological emergency. In developing countries like India, late presentations with tubal rupture and haemodynamic instability are seen in most of the cases. In developing countries, ectopic pregnancy remains an important cause of first trimester maternal morbidity and mortality [13]. Fetal wastage, recurrence and impairment of subsequent fertility are the most important concerns [14].

Though maternal mortality has decreased and almost very rare nowadays, it has been reported by Arpita N et al that ectopic pregnancies account for 7.1% of all pregnancy related deaths in rural India [15]. The main causes of these deaths are due to wrong diagnosis, delayed diagnosis and late admissions with severe hypovolemic shock. Ruptured ectopic pregnancies with severe intra abdominal bleed may present with gastro intestinal symptoms like nausea, vomiting, diarrhoea and poses a great challenge in correct diagnosis [16].

The management of ectopic pregnancies has been revolutionized in developed countries, because of the availability of laparoscopic techniques, medical therapy, uterine artery ligation and more advanced diagnostic modalities which help in early diagnosis, better conservation of future fertility [17], shorter hospital stay & reduced surgical morbidity.

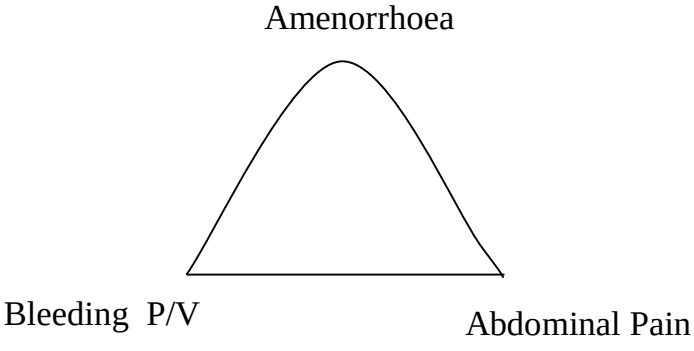
Due to several factors like delay in seeking health care, absence of investigations (like hCG, progesterone assays, transvaginal sonography) and lack of suspicion in the diagnosis of ectopic of pregnancy by most of

the inexperienced clinicians, early diagnosis is almost impossible in most developing countries [18].

In developing countries, many studies have shown that the ectopic gestation case fatality rate was 1 % - 3%. This is 10 times more than the case fatality rates reported in developed countries [19].

Although no risk factors have been identified in most of the cases with ectopic gestation, studies have highlighted that the awareness of risk factors associated with ectopic gestation helps in identifying the cases early [20] . Main risk factor is the prior tubal damage due to any cause [20]. Chlamydial infection was found to be the risk factor for 30 - 50% of all ectopic gestation [21].

Asymptomatic ectopic pregnancies are commonly seen in cases with early and unruptured ectopic gestation. When it ruptures symptoms could be acute or subacute [22]. Classical triad of ectopic pregnancy,



occurs only in 50% of the cases [23]. Because of the atypical presentations, the diagnosis remains a challenge to the obstetricians & Gynaecologists. Ectopic gestation is an important cause of maternal morbidity, presenting

with acute symptoms like pelvic pain, bleeding p/v and long term complications like infertility [24].

The incidence of rupture has declined in the last decades due to the availability of quantitative hCG assays, transvaginal sonography and minimally invasive surgeries [25].

Early diagnosis of ectopic pregnancy helps to reduce the incidence of rupture. So, the physician can provide conservative medical and surgical treatments [26].

Since, ectopic pregnancy is an important health problem among the reproductive age group women, the study was aimed to determine the clinical profile of the patients as well as the risk factors so as to make recommendations in order to reduce the incidence of this life threatening event.

JUSTIFICATION OF THE STUDY

Ectopic pregnancy is an important cause of maternal morbidity & mortality in early pregnancy on the one hand, there is a high chance of inaccurate diagnosis on the other hand, clinician might be faced with severe problems like life threatening haemorrhage. A variety of therapeutic options are available but there are no standard guidelines regarding the ideal management. Conservation of future fertility has priority while severe haemorrhage also needs to be prevented.

Ectopic pregnancy is a challenge for the clinician due to its bizarre clinical presentation. The diagnosis is complicated by the wide spectrum of presentations, from asymptomatic cases to acute abdomen and haemorrhagic shock.

Its ambiguous presentation may mimic most of the surgical and gynaecological emergencies.

The frequency of ectopic pregnancy has been increased during the last decades because of the increased incidence of sexually transmitted infections, advent of antibiotics, increased awareness and usage of various contraceptive methods and assisted reproductive technologies [27].

Prior damage to the fallopian tubes, resulting from previous ectopic pregnancy and previous tubal surgeries to relieve infertility or for sterilisation remains the highest risk factor for ectopic pregnancy [28].

A greater understanding of the etiology, an accurate history taking and relevant physical examination and its correlation with diagnostic techniques is critical for the development of preventive measures, early diagnosis and the development of novel treatments [29].

In developed countries, the management of ectopic pregnancies has dramatically improved, with less radical procedures performed with laparoscopy and medical management being increasingly utilised [30]. This is possible because majority of the patients are diagnosed early with the use of high resolution USG and improved biochemical detection of beta hCG. However, in developing countries like India salphingectomy by laparotomy still remains the main stay of treatment as majority of our patients present with tubal rupture. This is due to lack of awareness, ignorance, poverty, lack of adequate health services and difficulty in transportation [31].

Ectopic pregnancy could be considered as a public health indicator in the developing world, because ectopic pregnancy presents as an acute emergency and it is a life threatening event, providing an overall picture of a health system capacity to handle with the diagnosis and management of emergency situations.

Ectopic pregnancy is an emergency situation in which timely intervention will reduce the maternal mortality & morbidity. Clinical presentation varies from case to case and still there is a chance of missing

the diagnosis in a small proportion of patients even with the availability of advanced imaging techniques. For early diagnosis a clinician should be well aware about the various presentations of ectopic pregnancy. My study will provide a detailed clinical description on ectopic pregnancy.

AIM OF THE STUDY

A detailed study on ectopic pregnancy over a particular time period in order to determine the incidence, risk factors, clinical features, diagnostic methods, treatments, morbidity and mortality associated with ectopic pregnancy in a tertiary care hospital and analysis of various aspects of ectopic pregnancy with a view to suggest interventions to join the global trend of early diagnosis and conservative management.

OBJECTIVES

1. To know the incidence of ectopic pregnancy.
2. To know the age group, parity, sterilisation status with respect to ectopic pregnancy.
3. To analyse the various risk factors associated with ectopic pregnancy.
4. To describe the clinical presentation of ectopic pregnancy.
5. To determine the sensitivity and specificity of amenorrhoea, abdominal pain and vaginal bleeding in diagnosing ectopic pregnancy among the suspected cases.
6. To determine the positive and negative predictive values of amenorrhoea, lower abdominal pain and vaginal bleeding among the suspects.
7. To determine the sensitivity & specificity of cervical excitation test.

8. To determine the sensitivity and specificity of UPT, culdocentesis & USG among the suspects.
9. To determine the factors associated with time delay between admission & onset of treatment.
10. To determine the factors associated with radical procedures over conservative management.
11. To apply medical management criteria for all cases of unruptured ectopic pregnancy.

REVIEW OF LITERATURE

Pathophysiology of ectopic pregnancy:

Ectopic pregnancy is a significant problem for reproductive age women and their health care providers [32, 33]. An ectopic pregnancy results from disruption in the tubal transport process and ectopic pregnancy complicates 2% of all pregnancies in western countries[34]. More than 98% of ectopic gestations develop in the fallopian tube [35]. Unfortunately, there are no effective means of prevention, prediction or treatment of tubal implantation [35,36]. Despite the intense research activities, the basic pathology of tubal ectopic pregnancy remains a mystery [37, 38, 39]. This is in larger part due to our incomplete understanding of the complex molecular events behind muscle contraction, ciliary beating and tubal fluid micro environment changes during the gamete transport [39]. Disruption of any of these events results in abnormal interactions between the tubal cells & the early embryo and leads to tubal implantation [35].

The process of fertilization & earlier stages of embryonic development and implantation begins in the fallopian tube as a result of co-ordinated biochemical and physiological steps[40] . Tubal implantation may results from functional and molecular aberrations in the fallopian tubes, improper tubal fluid environment or abnormal sex steroid responsiveness [41].

Abnormal tubal transport

Abnormal ciliary function or spasm in the muscular layer of the fallopian tube may arrest the forwarding movements of the fertilised ovum along the fallopian tube and may result in tubal ectopic pregnancy. Mechanical factors like congenital obstruction of the malformed fallopian tubes, local endocrine factors, emotional & psychosomatic problems and improper progesterone secretion are associated with abnormal tubal transport. Inadequate corpus luteal secretion of progesterone may be sufficient to disrupt the normal passage of an embryo along the fallopian tube (Hunter, 1988). The diameter of the sperm head is only 5-8 micron. But the oocyte is 100 micron in diameter. In a partially obstructed fallopian tube, sometimes sperms can pass through but the oocyte could not. This explains the occurrence of an ectopic pregnancy in the distal segment of a partially obstructed tube.

Tubal infections:

Tubal infections, particularly chlamydial infection results in scarring of the fallopian tube and loss of patency (Sweet, 1982; Tuffrey et al., 1986). Tuberculosis and gonococcal infections may be involved in PID and complications at tubal level can arise following septic abortions. Inflammation arising from the bacterial infection leads to scarring of the fallopian tubes and results in partial or complete occlusion. Even if

patency has not been compromised seriously, the damage to endosalphinx results in surface alteration and favours ectopic implantation.

Ectopic pregnancy after sterilisation may result from operator errors or factors independent of operator errors [42]. Operator errors include,

1) Incorrect localisation of the sterilisation site.
2) Deviation from the recommended technique.
3) Failure to check the sterilisation method in a systematic way.
4) Transection of the fallopian tube partially or completely.
5) Application of two clips on each tube.
6) Improperly maintained mechanical occlusion device applicator.

Factors other than that of operator errors include,

1. Recanalisation of the tubes.
2. Formation of tubo peritoneal fistula, which may results from necrosis, tubal atrophy or endosalphingiosis[42].
3. Spontaneous reapproximation of the ends of tubes associated with tubal reanastomosis & recanalisation [42].
4. Congenital malformations like uterus didelphys, presence of an accessory fallopian tube and pre -existing uterotubal fistulas [43] .

Other factors like tubal healing response of the individual, extent of tubal avascularity, pre - existing proliferative tubal disease and the time

interval since operation are likely to affect the ability of tubal lumen to regenerate. [44,45,46,47,48,49].

Role of luminal fluids:

At the onset of implantation, the metabolic support to an developing embryo is fulfilled by

i) The cytoplasmic reserves of the oocyte .
ii) Various nutrients in the luminal fluid and uterine fluid, particularly pyruvate, lactate and glucose (Leese et al., 1988;Tay et al ., 1997).

These fluids are of characteristic composition regulated by the ovarian steroid hormones and developing embryo's metabolic requirements. The difference between tubal and uterine fluids are maintained by a formidable utero - tubal junction (Lee 1928; Flechon and Hunter, 1981). Glycoprotein secretions accumulating in the caudal portion of the isthmus also helps to maintain this difference. The tubal fluid also differs from region to region along and within the fallopian tubes (Leese, 1988, Nichol et al 1992). These factors help to maintain the tubal micro environment.

In reality both flexibility in substrate requirements of the embryo and overlap in the composition of uterine and tubal fluids could underlie the occurrence of an ectopic gestation (Lee 1928; Patek, 1974; Hunter, 1977, 1998). Infact, the development of an ectopic pregnancy at various

sites within the abdomen demonstrates the tolerance of the human embryo in the initial fluid environment.

Cellular and molecular mechanisms:

No biomarkers have been identified with high predictive value for tubal ectopic pregnancy [50] and the understanding of development of tubal ectopic pregnancy in humans at the cellular and molecular level is hampered by a lack of appropriate animal models [51].

17β estradiol, acting through estrogen receptors α & β may play a fundamental role in tubal cell homeostasis regulation and modulation of tubal physiological process. Fluctuations in the E_2 level, E_2 metabolism and the expression of estrogen receptor subtype in fallopian tube may play a crucial role in fallopian tube malfunction and development of ectopic pregnancy [52].

There is no animal model available for ectopic pregnancy, since it has not been reported in other organisms [53, 54]. Chlamydial infection is the main cause of ectopic pregnancy. So Chalmydial infection has been used in the mice to develop a model system for ectopic pregnancy. In this study, the muscle tone of the oviductal wall is reduced and the electrical pace maker potential which is required for the oviductal motility is lost [54, 55]. Thus tubal infection increases the incidence of ectopic pregnancy by impairing oviductal embryo transport.

Endocannabinoids, such as anandamide and 2-arachidonyl glycerol are ligands for cannabinoid receptor (CB1) and (CB2) [56]. Fatty Acid Amide hydrolase (FAAH) is an endocannabinoid degrading enzyme, which is important for maintaining the adequate levels of anandamide in the uterine environment. An increased risk of tubal pregnancy has been reported in recent studies using FAAH or CB-1 deficient mice caused by a delay in embryo transport from the oviductal tube to the uterus [53, 57].

Micro RNAs (mi RNAs), are evolutionarily conserved, small, non coding RNAs containing 21 to 24 nucleotides. They have emerged as overall gene expression regulators [58]. It has been reported that micro RNAs are expressed differentially in human fallopian tubes [59, 60]. Recently, the expression of several circulating mi RNAs have been shown as novel biomarkers for the diagnosis of tubal ectopic pregnancy.

Despite the decades of research, there is still a crucial need for the comprehensive understanding of the underlying pathophysiology of tubal ectopic pregnancy that fully integrates cellular mechanisms, gene regulation and signalling pathways in the fallopian tubes [61].

Age

Age has a role in the occurrence of ectopic pregnancy & other pregnancy complications. A study conducted by Bouyer et al proved that age has an important role and increases the exposure to other risk factors[62]. The progressive loss of myoelectrical activity along the

fallopian tube occurs due to aging. Age related changes in the tubal function and tubal diverticula predispose the older age group patients to develop ectopic pregnancy [63, 64].

In India, many studies have shown that the peak age of ectopic pregnancy incidence was between 20 - 30 years. (Samiya mufti et al, Majhi AK et al) [65, 66]. Westorm in swedon 3 in 1981 and Rubin et al in USA reported an increasing occurrence of ectopic pregnancy with age [67, 68]. Yuk JS et al showed that older age was associated with high occurrence of ectopic pregnancy in Korea [71]. This difference might be due to social factors. In India early marriage is common and most of the women finish their family before the age of 30 years. And also 20 - 30 years is the most fertile period with infrequent usage of contraception.

Parity

There is also a relationship between the parity and the ectopic pregnancy risk. A study conducted in the department of general hospital 'George Gennimatas' in Athense, Greece showed the statistically significant positive association between parity and ectopic pregnancy rupture [69]. Samiya Mufti et al.,Majhi et al., Smita Singh et al., showed the increased incidence of ectopic pregnancy in primigravida [65,66,70].

Socio economic status

Low socio economic status is associated with high incidence of ectopic pregnancy [71]. This may be due to increased prevalence of

sexually transmitted infections among the low socio economic group and their risky behaviour of delay in seeking health care facilities.

Tubal sterilisation

When tubal sterilisation fails, the ectopic pregnancy is likely. The incidence of ectopic pregnancy after tubal sterilisation failure varies between 5-90% [72]. Tubal ligation with resultant tubal damage carries odds ratio of 9.3 for ectopic pregnancy in comparison with pregnant controls [73]. Tubal sterilisation is widely regarded as a highly effective method of contraception. In a large multicentre study, the risk of ectopic gestation among women who have undergone common methods of tubal sterilisation was found to be 7.3/1000 procedures. In the same study, it was found that the rate of ectopic pregnancy for all common methods of sterilisation combined in the 4^{th} through 10^{th} years after surgery was not lower than the rate of ectopic pregnancy in the first 3 years [74].

The risk of ectopic pregnancy depends on the method used for tubal sterilisation. All methods of tubal occlusion require correct application to maximize the effectiveness. The higher rate of failures are seen with spring clip application and bipolar coagulation that highlight the requirement of proper techniques in the use of these methods as demonstrated by Hulka & Reich,Soderstrom et al.,[75,76]. The failure rate of pomeroy's technique is estimated as 0.25-2.1[77] .

The incidence of ectopic pregnancy is higher when tubal sterilisation is performed in the postpartum period because of the edematous, congested & friable fallopian tubes, which increases the chance of incomplete occlusion of the lumen [78].

The probable explanation for the occurrence of ectopic gestation after tubal sterilisation is recanalisation or formation of tuboperitoneal fistula. Sperms can pass through these fistulas but the fertilized ovum cannot. So, implantation of fertilized ovum typically occurs in the distal segment of the tube [79]. Recanalisation results in abnormal reconstruction of the tubal lumen with formation of slit like spaces & blind pouches. This may results in ectopic implantation [78]. Fluid movements within the remaining segments may also influence the implantation [78]. The risk of ectopic gestation may be higher after electro coagulation procedures, than any other methods. To avoid luteal pregnancy, sterilisation should be performed in the early follicular phase.

The ectopic pregnancy must not be disregarded in a woman who has undergone tubal sterilisation, particularly if two or more years have elapsed since the surgery [80].

Previous caesarean section and other pelvic surgery

A recent Danish register based study found a moderately increased risk of ectopic gestation in women who have undergone emergency and elective caesarean sections [81].

A meta analysis by O'neil et al showed 5% increased odds of ectopic pregnancy among the studies included, but the estimate did not reach the statistical significance [82].

A case controlled study by michalas et al found that all types of pelvic surgeries increase the risk of ectopic gestation from a 2 fold increase for appendicectomy to a 9 fold increase for ectopic pregnancy [83].

Tubal recanalisation surgery

In a large series study from various hospitals, the risk of ectopic pregnancy after sterilisation reversal was reported to be 0.3 to 3% of all pregnancies [84].

Microsurgical techniques for reversal of sterilisation provide better results than conventional surgery[85]. The lower rate of ectopic pregnancy in microsurgical technique is associated with

1. Proper alignment of lumen,
2. Gentle handling of tissues and
3. Proper excision of all pathological tissues under magnification .

Prior pelvic inflammatory disease

PID is defined as the infection of endometrium, fallopian tubes and contiguous structures by the ascent of micro organisms from the lower genito urinary tract [86]. It is most commonly associated with sexually transmitted infections, especially Chlamydia trachomatis & neisseria

gonorrhoea. These organisms often initiate the inflammatory process and then replaced by other opportunistic bacteria including anaerobes, aerobes and mycoplasma species [87]. Sexually transmitted infections represent a growing problem worldwide [88]. This concern is not only due to increasing incidence but also due to improved diagnosis. More than half of these infections are seen in young sexually active women aged between 16-25 years.

Prior infection is a risk factor for current infection. With the development of repeated PID, the risk of ectopic pregnancy and subfertility is increased. Approximately 70% of Chlamydial infections are asymptomatic. A small proportion may present with Vaginal discharge, dysuria, lower abdominal pain, arthritis or post coital bleeding.

The significance of infectious diseases in ectopic pregnancy is well documented [89, 90, 91, 92]. There might be a causal link. Declining Chlamydial infection rates attributed to preventive strategies and fall in the ectopic pregnancy risk in Sweden [93].

Multiple sexual partners and the young age at first intercourse are associated with increased risk of ectopic pregnancy (Bouyer et al).

Smoking

Many studies have reported that smoking is a major risk factor for the occurrence of ectopic pregnancy (odds ratio 1.7 - 3.9) [94]. Studies in animals and humans reported the effect of smoke exposure on oviductal /

fallopian tube function [95, 96]. Inspite of these findings, the exact mechanism by which the cigarette smoking increases the ectopic pregnancy risk remains unclear.

A recent study has reported that cotinine (an active nicotine metabolite) increases the prokineticin receptor , PROKR1 expression in the fallopian tube, which is a regulator of smooth muscle contractility and a gene important for intrauterine implantation [97]. Cigarette smoking attenuates tubal PROKR 1 expression resulting in altered tubal function and provides the explanation for the link between smoking & risk of ectopic pregnancy.

Bouyer et al demonstrated a dose-effect relationship between smoking & risk of ectopic gestation [98]. Cessation of smoking reduces the ectopic pregnancy risk a level intermediate between that of current smokers & non - smokers. However, there was no trend observed for time since smoking cessation .

Obesity and infertility

Obesity is a growing problem throughout the world and upto 25% of women in the reproductive age group are being obese (Balen and Anderson, 2007, Haslam and James, 2005). Obesity was found to be associated with reduced fecundity (Ramlau - Hansen et al 2007; vanden steeg et al, 2007). The obese women are more prone to anovulation and polycystic ovarian syndrome . (pasquali et al, 2007). In comparison to

normal weight women of reproductive age, obese women seek medical attention for their subfertility more often. (vahratian and Smith 2009). There are studies reported on the relationship between BMI and ectopic pregnancy.

1. Zhang et al, 2010
2. Sneed et al 2008
3. Mataliotakis et al, 2008
4. Fedorcsak et al, 2008
5. Wittemer et al, 2000.

None of the above mentioned studies found a significant difference between overweight and normal women. Bouyer et al ., 2003; Tanveer Shafquat et al., 2013 found that the ectopic pregnancy risk increased with the duration of subfertility [98, 99]. However, ectopic pregnancy is a risk factor for subsequent infertility [100, 101, 102]. The association between infertility and ectopic pregnancy seems to be mutual risk factors and are likely to be complex.

Ovulation induction and ART

The ovulation induction and other ARTs may be associated with increased risk of ectopic pregnancies [103]. There are many case reports of ectopic pregnancies following ovulation induction [104, 105].

ART is a known risk factor for ectopic pregnancy. The rate of ectopic gestation is higher in pregnancies resulting from ART than in

spontaneous conception with the incidence ranging from 2% to 8.6% particularly in gamete & Zygote fallopian transfer (GIFT and ZIFT)[106, 107]. Heterotopic pregnancies and bilateral tubal ectopic pregnancies, the rarest form of ectopic pregnancies have also been reported following IVF and embryo transfer (IVF - ET) and GIFT.

The presence of tubal pathology and PID could be the predisposing factors as with tubal pregnancies (Cohen et al 1999). Deep deposition of the embryos in to the uterine cavity [108] or the usage of large volume of culture fluid during the embryo transfer [109] may result in ectopic pregnancies. Ectopic pregnancies are significantly lower when single frozen thawed blastocysts are transferred compared with two blastocysts. (Yanaihara et al, 2008; Ishihara et al, 2010).

Previous ectopic pregnancies

A prior history of ectopic pregnancy is a possible indicator of the pre existing tubal damage and hence a strong risk factor for recurrence [73]. This is because tubal pathology is always bilateral and there is a strong tendency for ectopic gestation to occur first on one side and then on the other side later [110]. The patients with recurrent ectopic pregnancies are more likely to experience a tubal rupture [111]. A study reported that women with history of an ectopic pregnancy had 13 times higher the chance for the second ectopic compared to women who had a first live birth [112].

Intrauterine contraceptive device use

Although IUCD users are supposed to be protected from both intrauterine and extra uterine pregnancy, it was found that a woman who conceives with IUCD in place is seven times more likely to have an ectopic pregnancy in comparison to a woman who conceives without IUCD [113]. Ectopic pregnancy is known to occur frequently in women who use certain types of IUCDs [114]. IUCD usage was found to increase the risk of ectopic pregnancy in a study conducted by Shaista [115] in Saudia Arabia and Bouyer et al [98] in France. Bouyer et al confirmed that previous intra uterine device use has increased risk of ectopic pregnancy per se not only through an association with pelvic infections as previously suggested [116, 117].

Progesterone contraceptive pills

Levonorgestrel - only emergency contraception is a well established emergency contraception. (Gemzell - Denielsson 2010). The risk of ectopic pregnancy should be kept in mind following LNG only emergency contraception failure. There are many cases of ectopic pregnancies reported in literature after the LNG only emergency contraception failure (Ghosh et al., 2009; Kozinszky et al., 2011). The rate of ectopic pregnancies following LNG only emergency contraception failure was 4.1% which was found to be higher than the incidence of 1.1% to 1.6% in spontaneous pregnancies (Varma & Gupta 2009).

In general estrogen stimulates the tubal myo - electrical activity and progesterone inhibits that [118]. Progesterone alters the tubal motility. Inversion of tubal peristalsis and reduced fimbrial beating may contribute to the delayed arrival of egg in the endometrial cavity, a fact resulting in ectopic pregnancy [119, 120, 121, 122]. Pharmacologic level of progesterone relaxes the tubal myo-electrical activity to an extent that the transport through the isthmus does not occur [123, 124]. Higher rates of failure are seen when emergency contraception is performed during the period close to ovulation [125].

Prior history of abortions

Parazzini et al ., found that the rate of ectopic pregnancy was higher in women with history of prior induced abortions and also the risk increased with the number of induced abortions in comparison to women with history of no induced abortions [126]. Spontaneous abortions might have a causal effect, most probably mediated by pelvic infection [127]. Bouyer et al found an association between induced abortions and the risk ectopic pregnancy, with an odds ratio of 1.9 for women with history of two or more previous induced abortions [98].

Sites of ectopic pregnancy

Nearly all ectopic pregnancies are implanted in the fallopian tubes (97%) and a common factor for the occurrence of such ectopics are the presence of a tubal pathology. 70% of ectopic pregnancies occur within

the ampulla. The rate of ectopic pregnancy in different parts of the tube also varies [128] as mentioned below.

Ampullary - 70%

Fimbrial - 11%

Isthmic - 12%

Interstitial - 2.4%

Fate of the tubal pregnancy

1. Tubal mole

The gestational sac with surrounding blood clot is retained within the fallopian tube.

2. Tubal abortion

This is more common if ectopic implantation occurs in the ampullary part of the tube. Separation of the sac and its expulsion into the peritoneal cavity via the tubal ostium occurs.

Rarely reimplantation of the products of conception may result in secondary abdominal pregnancy.

The bleeding usually stops if the expulsion is complete. Sometimes bleeding may persist in incomplete separation or bleeding from the site of implantation

3. Tubal rupture

This is more common in the isthmus, which is the narrower portion of the fallopian tube. Rupture may occur early in the anti mesenteric

border and results in massive intra peritoneal haemorrhage. If rupture occurs in the mesenteric border of the fallopian tube, a broad ligament haematoma can occur.

Ectopic implantation can also occur outside the fallopian tubes, within the ovary, cervix, caesarean scars, uterine cornua and abdomen. These extra tubal implantations may not be associated with pre existing risk factor or tubal pathology.

Cervical pregnancy

Cervical pregnancy accounts for less than 1% and the rarest of ectopic pregnancies implanted in the cervical canal below the internal os[128, 129] . Induced abortions, prior uterine curettage, leimyomata, Asherman's syndrome, IUCD, IVF are the predisposing factors. Raskin suggested that the ultrasound diagnosis of cervical pregnancy required the following 4 criteria [130].

1. Diffuse enlargement of the cervix
2. Enlargement of the uterus
2. Amorphous intra uterine echoes.
3. The absence of an intra uterine gestation.

Timor - Tristch et al., refined the criteria and included that the placenta and the chorionic sac containing pregnancy must lie below the linternal os and the cervical canal must be enlarged like a barrel [131].

Anticipation of significant bleeding and a plan to prevent / control haemorrhage can avoid hysterectomy.

Ovarian Pregnancy

The incidence is 1 - 3%. The presenting signs & symptoms are like other ectopic pregnancies. It is difficult to make the pre operative diagnosis of an ovarian pregnancy. USG shows a walled cystic mass within or adjacent to the ovary. The differential diagnosis includes a corpus luteal cyst and a tubal implantation. The diagnosis is usually a pathological diagnosis made by histopathologic examination of removed adnexal mass based on the following speigelberg's criteria [132].

1. The fallopian tube should be normal and distinct from the ovary.
2. The gestational sac should occupy the normal anatomical position of the ovary.
3. There should be a connection between the gestational sac and the uterus by utero - ovarian ligament.
4. The definite ovarian tissue should be demonstrable in the wall of the gestational sac.

Doppler USG cannot always distinguish the corpus luteal cyst from the ovarian pregnancy. Speigelberg's criteria cannot be established with ultrasound[133].

Caesarean scar ectopic pregnancy

Although previously rare, the incidence of caesarean scar pregnancy is increasing worldwide [134]. This increase is presumably due to more number of caesarean sections and increased recognition. Caesarean scar ectopic pregnancy is surrounded by myometrium and the fibrous scar tissue completely and separated from the endometrial cavity and fallopian tube. Early diagnosis with ultrasound can avoid life threatening haemorrhage and uterine rupture.

Cornual ectopic pregnancy

It is one of the most dangerous types of ectopic pregnancies with high maternal mortality. The incidence is 2 - 4% of all tubal pregnancies [135]. This type of pregnancy can be discovered with advanced gestational age due to the adjacent supporting myometrial walls and good vascular supply, particularly from the uterine artery [136, 137]. In case of rupture the bleeding can be fatal. Previous pelvic surgeries, uterine anomalies, ART, previous ipsilateral salphingectomy [138] are the risk factors.

Isthmic tubal ectopic pregnancy

Upto 12% of ectopics are implanted within the isthmic portion of the fallopian tube [128]. The isthmic portion of the tube is narrow with a well defined muscularis layer. So, Isthmic ectopic pregnancies quickly invade the muscularis layer and rupture early [139].

Abdominal pregnancy

It is an extremely rare and dangerous form of extra uterine pregnancy [140]. Incidence is less than 1%. Many are due to secondary nidation within the peritoneal cavity after tubal rupture, tubal abortion or uterine rupture [141]. Primary abdominal pregnancy should satisfy the following criteria of Studdiford[142].

1. Tubes and ovaries should be normal.
2. There should not be any uteroplacental fistula.
3. The attachment to peritoneal surface early enough to exclude the possibility of secondary implantation. The placenta may be attached to bowel, mesentry, uterine wall, liver, spleen, bladder and ligaments. The placenta may separate at any time during pregnancy resulting in life threatening haemorrhage [143]. The maternal mortality rate can reach as high as 20% [144].

Bilateral tubal pregnancies

They are extremely unusual occurrence in the absence of preceding ovulation induction. More common are twin pregnancies in the same tube and heterotopic pregnancies [145]. Fishback described the criteria for diagnosing bilateral tubal pregnancies. He claimed that there should be description of the fetuses or fetal parts as well as of placental tissue [146]. Norris declared that microscopic identification of chorionic villi in each tube should suffice [147].

Clinical presentations of ectopic pregnancy

Clinical manifestations of ectopic pregnancy usually appear six to eight weeks after the last menstrual period, but it can occur later depending upon the site of ectopic implantation [148]. The classic triad of ectopic pregnancy are

1) Abdominal pain,
2) Amenorrhoea and
3) Vaginal bleeding.

These symptoms can occur in both unruptured and ruptured cases. But unfortunately the classic triad is present only in 50% of patients [149].

Abdominal pain is the cardinal feature of ectopic pregnancy present in close to 100% of cases [150]. To start with dull pain caused by tubal stretching, sharp colicky pain follows.

About 75% of the patients present with amenorrhoea of less than 6 weeks. There is no history of amenorrhoea if rupture occurs in early weeks and if vaginal bleeding and pain starts around the next expected period. Amenorrhoea lasts even upto 16 weeks in case of cornual and interstitial pregnancies [151].

Abnormal vaginal bleeding or brownish vaginal discharge occurs in 50% of the patients [149]. Early vaginal bleeding simulating uterine abortion is seen in caesarean scar pregnancy [151].

These symptoms overlap with the symptoms of spontaneous abortion. A prospective study found no statistically significant difference in the presenting features of patients with early intrauterine pregnancies versus those with unruptured ectopic pregnancies [152].

The patients may present with others common symptoms of early pregnancy including fatigue, nausea, breast fullness, heavy cramping and recent dyspareunia. Advanced abdominal pregnancy may present with painful fetal movements.

Late presentations after the rupture of the ectopic pregnancies are common in developing countries. They may present with dizziness, gastro intestinal symptoms like diarrrhoea, shoulder tip pain (due to massive haemoperitoneum), syncopal attacks and sudden cardiac arrest.

Some cases of intra peritoneal bleeding with pelvic haematocele could present with ‘toilet signs’, which include dysuria, urinary frequency and tenesmus. There are reported cases of patients with ectopic pregnancy fainting in the toilet or following sexual intercourse.

Differential diagnosis of ectopic pregnancy

The following conditions mimic the ectopic pregnancy in their clinical presentation [153].

1) Pelvic inflammatory disease
2) Dysmenorrhoea
3) Endometriosis

4) Ruptured corpus luteal cyst
5) Acute appendicitis
6) Typhoid perforation
7) Acute intestinal obstruction
8) Acute diverticulitis
9) Gastroenteritis.

Physical examination

The physical examination findings of cases with ectopic pregnancies are highly variable and mostly unhelpful. Many patients present with stable haemodynamic status and benign examination findings, especially in unruptured ectopic pregnancy.

Careful assessment of vitals may show classical picture of haemorrhagic shock including tachycardia, pallor, hypotension and oliguria [149] in case of ruptured ectopic pregnancy.

Other signs include presence of abdominal tenderness/guarding/ rigidity, abdominal distension, forniceal tenderness, positive cervical excitation test, forniceal bogginess or presence of any adnexal mass [149,154].

Tachycardia may not present in all cases of ruptured ectopic [154, 155]. Abdominal tenderness/distension is not specific for ruptured ectopic pregnancy and it is present in almost all cases of surgical emergencies.

Forniceal tenderness and cervical motion tenderness may present in PID and endometriosis other than ectopic pregnancy. So this sign is also not specific. An adnexal mass may be palpated, but it is difficult to differentiate the mass from the ipsilateral ovary. The patient's discomfort may preclude the adequate pelvic examination and overzealous assessment may result in iatrogenic tubal rupture.

In a study conducted in Mumbai abdominal tenderness was present in 59.69%, abdominal distension in 31.12%, forniceal tenderness in 71.43% and tender cervical movements in 69.89% and forniceal bogginess in 54%. Tachycardia and hypotension was seen in 52.55%. 13.7% patients had no abdominal signs [154] .

Cervical motion tenderness / cervical excitation test

Cervical motion tenderness is defined as an unpleasant sensation or response elicited on bimanual pelvic examination with movements of the cervix by the examiner's gloved hand, usually indicative of inflammatory process in the pelvic / adjacent organs that might be moved during such examination. (Stedman's part of Lippincott Williams &Wilkins).

Cervical excitation test is a controversial sign. The examiner put a finger each side of the cervix and push it from side to side which in turn stretches the tubes. The examiner has to look the patient's face for pain. Pushing the cervix forward or back may give false positive result.

Cervical excitation test may be positive in ectopic pregnancy and PID. Role of this test in ectopic pregnancy is controversial because it might burst the unruptured ectopic pregnancy. So, this test should be done gently.

Culdocentesis

Culdocentesis is an invasive procedure in which peritoneal fluid is aspirated from the cul de sac of a female patient. Spinal needle is introduced into the pouch of douglas through the vaginal wall.

Ultrasonography with its improved resolution and more availability has virtually replaced the culdocentesis as a procedure of choice. USG is an non invasive procedure and has sensitivity and specificity superior to that of culdocentesis in the diagnosis of haemoperitoneum.

Indications

1) To diagnose suspected ruptured ectopic pregnancy in the following situations [156, 157, 158, 159].
    a) Haemodynamically unstable patients.
    b) When ultrasonography is not immediately available.
2) In place of diagnostic peritoneal lavage to diagnose haemoperitoneum following blunt trauma abdomen.
3) To diagnose ruptured ovarian cyst.
4) To obtain fluid for culture in the treatment of PID.

Contra indications

- Pelvic mass including tubo ovarian abscess, ovarian tumors, pelvic kidney and appendiceal abscess.
- Fixed retroverted uterus.
- Coagulopathy.
- Prepubescence.
- Non co-operative patient.

Culdocentesis is considered positive when more than 2 ml of non clotting blood is obtained. Approximately 80-95% of ruptured ectopic pregnancies display non clotting blood on culdocentesis [157].

A study by Romero et al reported that when both a positive pregnancy test and a positive culdocentesis were present, ectopic pregnancy was found in 99% of cases [160].

About 15% of ectopic pregnancies show negative or non-diagnosis results on culdocentesis [159]. The false positive results are seen in 2.9% of cases. A ruptured corpus luteum in the presence of an intrauterine pregnancy is the common cause of false positive result [161].

Complications are rare. However the following complications may occur.

1) Rupture of an unsuspected tubo-ovarian abscess, ovarian teratoma, endometrioma or a malignant ovarian cyst.
2) Puncture of the bowel or pelvic kidney.
3) Rupture of tubal pregnancy.
4) Bleeding from the puncture site.

In the emergency situations culdocentesis can be a safe, simple and critical diagnostic procedure. In an unstable woman with a strong suspicion of a ruptured ectopic gestation, culdocentesis may facilitate the live saving surgery by conforming the diagnosis without waiting for the time consuming ultrasound exams. Many recent studies from developing countries have shown that this simple procedure helped in the diagnosis of tubal ectopic pregnancy in more than 70-80% of cases [66, 162].

Urine Pregnancy test

The currently used urine pregnancy tests detect the monoclonal antibodies to the beta subunit of hCG. So, the possibility of cross-reaction with LH and TSH is reduced. However, the antibody reacts with both intact hCG, which is the major component in serum and also with the fragments of beta - subunit (beta core) which are the major form in urine. Small amount of hCG are detected in the blood and urine of non pregnant patients [163] .

hCG is secreted from the implanting blastocyst and first appears in the blood around 6-8 days after fertilization, the levels rise progressively and reach a peak around 7 – 10 weeks. Most urine pregnancy test kits have sensitivity to detect as low as 25 mIU/ml of hCG in urine. Urine pregnancy test may become positive 3-4 days after the implantation; by 7 days (at the time of expected period), 98% will be positive [163]. A

negative result one week after the next expected menstrual period virtually says that the woman is not pregnant [163, 164].

A Urine pregnancy test for all reproductive age women experiencing vaginal bleeding or pelvic pain has been recommended [165, 166].

A systemic review conducted in 1998 reported that UPT was accurate (97 .41%) when used by health care professionals. The accuracy fell to 75% when used by consumers due to failure to follow instructions [167].

A negative urine pregnancy test does not exclude ectopic pregnancy, because the placental implantation in tubal location can be either nonviable or compromised and therefore not able to secrete enough hCG for a positive pregnancy test. (Richard M. Soderstrom M.D). Around 5^{th} to 7^{th} week of pregnancy, urine concentration of the hCG beta core fragment, an hCG variant rise dramatically, interfering with hCG detection and can cause false negative test results [168].

Serum β-hCG concentrations

The most sensitive, gold standard test to diagnose the pregnancy is the quantitative serum β-hCG estimation. The hCG secretion begins no later than the blastocyst stage. The beta hCG levels vary according to the period of gestation. In a non pregnant woman beta HCG level is less than 10mIU/ml. The estimated serum β-hCG 14 days after ovulation in a

singleton healthy pregnancy is about 100mIU/ml. The serum beta hCG level should double every 48-72 hrs in a healthy intrauterine pregnancy. The β-hCG levels are higher in multiple pregnancies [169]. A recent study reported that the minimum rate of rise in β hCG for a viable intra uterine pregnancy is 24% at first 24 hrs and 53% at 48hrs [170].

Failure of β-HCG values to double as expected, suggests that the pregnancy is unhealthy [169]. Possibilities include an ectopic pregnancy or a non-viable intrauterine pregnancy which will miscarry.

A rapid decline in serum concentrations of β-hCG usually by 21-35% or more over 2 days, is suggestive of spontaneous abortion [171] or a resolving ectopic gestation. The concentration of serum β-hCG has no definitive pattern in ectopic pregnancy [172]. However, in more than 70% cases the serial β-hCG values decrease more slowly than that of miscarriage and increase more slowly than that of a healthy intra uterine pregnancy [173]. An ectopic pregnancy is more likely when an intrauterine gestational sac is not seen by transabdominal scan when β-hCG concentration is above 6500 IU/L or by transvaginal scan at a β-hCG concentration of more than 1500 IU/L [174]. Combined transvaginal ultrasound and serial serum β-hCG estimation are approximately 96% sensitive and 97% specific for diagnosing an ectopic pregnancy [169].

Role of ultrasound in diagnosing ectopic pregnancy

Transvaginal sonography has revolutionized the approach to the diagnosis of ectopic pregnancy. TVS can confirm the diagnosis of an intrauterine pregnancy earlier than transabdominal ultrasonography (TAS). It has been reported that using transabdominal scan, an intra uterine gestational sac can be visualised when the serum β-hCG is >6500 IU/L [175]. However using TVS an intrauterine gestational sac can be visualized when the serum β-hCG is as low as 1000 IU/L [176]. Transvaginal sonography is able to identify the gestational sac at gestational age of 5.5 weeks with almost 100% accuracy [177, 178]. Intra uterine pregnancy is diagnosed definitely by sonographic visualisation of a yolk sac or an embryo in addition to the gestational sac [179, 180, 181]. An ectopic pregnancy may show 'Pseudosac', a fluid collection within the cavity of endometrium that may result from the breakdown of the decidualised endometrium. Pseudosac is centrally located inside the endometrial cavity compared to early gestational sac that is eccentrical [182]. Pseudosacs wont have a good hyper echoic decidual reaction around them and they are transient. In addition, the yolk sac and cardiac activity must be visualised clearly after 6 weeks of gestational age. So, it is important to differentiate the pseudosac from an empty intra uterine sac in early pregnancy.

The identification of an intrauterine gestation rules out the ectopic pregnancy unless a heterotopic pregnancy is strongly suspected [183]. The diagnosis of an ectopic pregnancy using the TVS is based on the positive visualisation of an extra uterine pregnancy [184]. It is classified as PUL if neither an intrauterine nor an extra uterine gestational sac is visualised on TVS. PUL should be followed until the final pregnancy outcome is known.

Tubal ectopic pregnancy

It is the most common form of ectopic pregnancy. The ectopic mass is seen in the adnexa separately from the ovary [184]. There may be

1) An extra uterine empty gestational sac with a hyper echoic ring sign.
2) Inhomogeneous adnexal mass.
3) An extra uterine gestational sac with yolk sac or/ and a fetal pole with or without cardiac activity [181 ,185].

It has been reported that an inhomogeneous adnexal mass may be seen in an early ectopic pregnancy or a resolving ectopic pregnancy [186].

Interstitial ectopic pregnancy

An interstitial or cornual ectopic pregnancy can be diagnosed with the following criteria.

1) Absence of gestational sac inside the endometrial cavity.

2) Products of conception are seen outside the endometrial cavity and with a continuous rim of myometrium surrounding it [187].

Cervical ectopic pregnancy

A cervical pregnancy is diagnosed by

1) Absent intrauterine gestational sac.
2) Enlarged and Barrel – shaped cervix.
3) The gestational sac which lie below the internal cervical os.
4) Absent 'Sliding sign' (when probe pressure is applied to cervix, the gestational sac slides against the endocervical canal in a miscarriage but this sign is absent in cervical pregnancy).
5) Blood flow surrounding the gestational sac in doppler ultrasound [188, 181].

Ovarian pregnancy

No specific criteria available for the sonological diagnosis of an ovarian ectopic pregnancy.

Caesarean scar ectopic pregnancy

The diagnosis is made by

1) Absent intra uterine gestational sac.
2) The gestational sac is located at the level of the internal os anteriorly and covers the previous caesarean section scar.
3) Blood flow on doppler ultrasound.
4) 'Sliding sign' will be absent [189].

The heterotopic pregnancy

In heterotopic pregnancy, an intra uterine pregnancy is present in conjunction with an ectopic pregnancy.

Many studies have shown that TVS has high diagnostic accuracy for ectopic pregnancy with 87 – 99% sensitivity and 94.99% specificity [190, 191, 192, 193] .

False positives can occur if other structures like corpus luteum, bowel, a hydrosalphinx, a para tubal cyst or an endometrioma are mistakenly taken as an ectopic pregnancy. If the ectopic is small or concealed by uterine anomalies or fibroids or by bowel, it can be missed.

Around 80% of the ectopic pregnancies are seen on the same side as that of the corpus luteal cyst. We have to search for any adnexal mass in the presence of corpus luteum . Visualisation of free fluid in the pouch of douglas increases the suspicion of an ectopic pregnancy [194]. However a minimal collection due to increased vascular permeability in early pregnancy is common.

Role of CT and MRI in ectopic pregnancy

Because of its lack of ionizing radiation and better soft tissue contrast, Magnetic Resonance Imaging is being frequently used in pregnant patients.

Sonography is the investigation of choice in diagnosing the early intra uterine pregnancy and ectopic pregnancy. Sometimes sonography

may fail to detect even an advanced ectopic pregnancy because of its inherent limited resolution, especially in patients with severe pain with non ideal body habitus. MRI can sometimes serve as a problem solving too l [195-196].

MRI should be reserved for haemodynamically stable patients in whom additional information is necessary for further management and that cannot be obtained using sonography [197,198].

MRI offers the following benefits as compared with sonography

1. Mutiplanar imaging,
2. Greater soft tissue contrast,
3. Lack of ionizing radiation and
4. More specific characterisation of tissues and fluids [199].

CT is frequently performed imaging modality in the evaluation of pelvic and abdominal pain of unknown cause. CT is certainly not a preferred modality to diagnose an ectopic pregnancy, particularly for a condition that can be imaged using other modalities. CT findings of ectopic pregnancy are not well recognized because CT is generally contra indicated in pregnant women due to its ionizing radiation.

Many classic sonographic features of an ectopic pregnancy have their CT and MRI correlates. So, the familiarity of various sonographic signs of ectopic pregnancy and their correlation with CT and MRI

findings facilitates prompt and accurate diagnosis and further management.

A recent study by Takahashi A et al proved that MRI is an effective modality for diagnosing the ectopic pregnancy with 91.3% sensitivity, 100% specificity and 100% positive predictive value [200].

Serum Progesterone level

The estimation of serum progesterone level is useful in the assessment of PULs [201]. The level is more than 50ng/ml in a healthy intra uterine pregnancy. However, the measurement of serum progesterone levels could not differentiate an ectopic pregnancy from a case of early IUP or a miscarriage [202]. In case of pregnancy of unknown locations, progesterone level of <5ng/ml is used to differentiate the low risk patients who can be managed conservatively and the at risk patients who require definitive management [203] .

Other serum biomarkers

Although many other serum biomarkers like 17α hydroxy progesterone, Inhibin - A, IGFBP - 1 have been reported , none of these biomarkers are used in day to day practice [204] . A recent study by Andrew Horne et al showed that the placental growth factor is a key factor in neovascularisation and it is a promising biomarker for ectopic pregnancies [201].

Diagnostic laparoscopy

When ultrasound is inconclusive but an ectopic pregnancy is suspected, a diagnostic laparoscopy may be done. Diagnostic laparoscopy is the gold standard investigation in the ectopic pregnancy. Delay in the diagnostic laparoscopy has been highlighted as one of the reason for maternal mortality [205]. However, sometimes small ectopic pregnancies may be missed during laporoscopy or laparotomy [201]. An alternative to diagnostic laparoscopy is serial USG examinations with β-hCG measurements or an endometrial biopsy or an empirical medical therapy.

Role of endometrial biopsy in ectopic pregnancy

In selected cases of pregnancy of unknown locations, an endometrial sampling can be done and analysed for the presence or absence of chorionic villi. It is useful when performed after a negative diagnostic laparoscopy for a suspected case of an ectopic pregnancy (Vanitha N Sivalingam et al) [94] .

MANAGEMENT OF THE ECTOPIC PREGNANCY

An ectopic pregnancy can be managed by any of the following method

1) Expectant management.
2) Medical management.
3) Surgical management.
4) Surgically administered medical management.

Expectant management

Sometimes, without any intervention ectopic pregnancies may regress to abortion with only minimal bleeding or they may be reabsorbed. The greater challenge lies in the identification of these cases in which intervention is not necessary.

Expectant management was first described by Lund (1955) in women with ectopic pregnancies [206]. He compared the expectant management versus the open surgery in a retrospective study. Surprisingly, the expectant management was successful in 57% of women.

Inclusion Criteria (Elito et al, 2008)[207]

1) Patients with stable haemodynamic status.
2) Falling beta-hCG levels of more than 15% in a time interval of 24 to 48 hrs.
3) Initial level of serum beta-hCG less than 1,500 mIU/ml.
4) The size of the adnexal mass should be less than 3.5 cm and it should be extra ovarian .
5) There should not be any demonstrable embryonic cardiac activity.
6) The patients who want to retain their future fertility.
7) The patients willing to sign the consent form.

Follow-up

Weekly serum Beta-hCG levels should be done until the level becomes undetectable. Usually it will take around 3 weeks for the complete resolution of an ectopic pregnancy (4 to 67 days).

Routine transvaginal scan is not necessary during the course of falling beta-hCG . Transvaginal scan and haematocrit should be done in all patients with the complaint of severe and prolonged abdominal pain to rule out the rupture . Small amount of fluid collection in POD is a common finding in most of the patients. The amount of free fluid increases with the tubal abortion. But, the surgical intervention is recommended only in haemodynamically unstable patients and in patients with tubal rupture .The complete disappearance of the adnexal mass in scan may take as long as 3 months. During this time period , we should advise the patients to avoid pregnancy. Hysterosalpingography should be done after the disappearance of tubal mass in ultrasound.

Predictive factors of success [208,209].

1) Declining level of beta-hCG over first 24 to 48hrs interval.
2) The level of initial serum beta hCG should be low.
3) No identifiable gestational sac like structure during ultrasound examination .
4) Longer the period of amenorrhoea.

Reproductive future

The fertility of women after the expectant management of an ectopic pregnancy could be determined by hysterosalpingogram or a spontaneous conception following the treatment [210,211].

Hysterosalphingography is a good method to evaluate the patency of the tubes [212] . HSG is considered as an important diagnostic procedure after the conservative management of an ectopic gestation (Mol et al., 1997)[213] Routine hysterosalpingography is usually advised 3 months after the completion of expectant management .

Elito et al. (2005a) reported the tubal patency rate of around 78% in expectantly managed women , and it was similar to the results of other studies conducted by Stovall and debby [214,215,211] .

Medical treatment

Systemic treatment

Methotrexate has been used for treating the gestational trophoblastic disease since 1956[216]. MTX inhibits the dihydrofolate reductase enzyme which is important in the synthesis of active form of folate used in RNA and DNA precursor synthesis. Side effects are commonly seen with multiple doses of MTX. Side effects are usually minimal including gastritis, nausea, vomiting, giddiness and ulcerative stomatitis. But, severe complications like neutropenia, pneumonitis and reversible alopecia can occur rarely. Folinic acid, a MTX antagonist can

be used to reduce the side effects, especially when multiple doses of methotrexate are used [217].

The medical treatment of an ectopic pregnancy with multiple doses of intramuscular MTX started in 1982 by Tanaka et al., [218]. In 1989,Stovall et al., conducted an important study and they individualised the dosage of methotrexate to improve the patient compliance, to reduce the overall costs and to minimise the side effects like pneumonitis, which ultimately led to single-dose MTX regimen of 50 mg/m^2 body surface area intramuscular use without folinic acid [219].

Selection criteria

Complete blood count, liver function tests, serum creatinine and blood grouping and Rh typing should be done in all patients before starting the treatment. Chest x-ray should be taken in patients with prior history of lung diseases.

Inclusion Criteria for Systemic Methotrexate treatment (ASRM Practice Committee, 2008; Elito et al, 2008).

1) Patients with stable haemodynamic status.
2) The size of the adnexal mass should be less than 3.5 cm and it should be extra ovarian .
3) The patients who want to retain their future fertility.
4) The patients should sign the consent form.
5) The patients should be willing for follow up.

6) There should not be any severe or persistent abdominal pain.

Absolute Contraindications for Systemic Methotrexate treatment (ASRM Practice Committee, 2008 ; Elito et al ,2008).

1) The presence of an intrauterine pregnancy.
2) Moderate to severe anaemia.
3) Thrombocytopenia (Platelets < 100000).
4) Leukocyte count less than 2000 Cells/ Cumm .
5) Sensitivity to Methotrexate .
6) Immuno compromised patients.
7) Active lung disease.
8) Active gastric ulcer.
9) Lactating women.
10) Renal and hepatic failure.

Relative Contraindications for Systemic Methotrexate treatment (ASRM Practice Committee, 2008; Elito et al, 2008)

1) Detectable embryonic cardiac activity by ultrasound.
2) Initial beta hCG concentration of more than 5,000 mIU/ml.
3) Falling beta-hCG levels of more than 15% in a time interval of 24 to 48 hrs.
4) Patients not willing for blood transfusion.
5) Patients not willing for follow-up.

Methotrexate protocols (single dose regime and multiple dose regime)

Single dose methotrexate regime and multiple dose methotrexate regime are two commonly used medical management protocols (Barnhart, 2009) [26].

In single dose methotrexate treatment 50mg/m^2 intramuscular dose of MTX is used without folinic acid [220,221]. Follow-up serum beta-hCG levels are done on days 1, 4 and 7 after MTX injection. Second dose of MTX (50mg/m2) should be given in patients with less than 15% fall in their initial beta hCG level between day 4 and day 7. Patients with more than 15% fall in their initial beta hCG level should be followed weekly until the level falls below 5 mIU/ ml. The term ‘‘single dose’’ actually describes the number of MTX injections planned, but the treatment may include additional doses of MTX injections of maximum 3 doses when the response is not adequate [220,26].

In multiple-dose protocol 4 intramuscular doses of MTX (1mg/kg) are given on alternate days with 4 intramuscular doses of leucovorin (0.1 mg/kg). Serum beta-hCG levels are done on the day of MTX injection. MTX injections are continued until the level of beta-hCG falls by 15% from its peak concentration. Approximately 50% of cases won’t require the full 8-day regimen course (Pisarka et al.,)[222].

A meta-analysis by Bankart et al., showed 93% success rate for multiple-dose treatment and 88% for single-dose treatment [223].One study

by Lipscomb et al., reported that medical management was successful in 78%–96% of selected patients [224].

Follow-up

Serum beta-hCG levels are monitored weekly until the level of beta-hCG becomes negative. It usually takes 2 to 3 weeks for the complete resolution of an ectopic pregnancy after MTX treatment in majority of the cases, but can take as long as 6 to 8 weeks which depends on the pre-treatment beta-hCG levels [221.223]. When falling beta-hCG levels rise once again, there is a possibility of persistent trophoblastic tissue.

The tubal mass may take 3 to 6 months for its complete resolution in the ultra sound [225]. Therefore, serial ultrasound evaluation after MTX treatment cannot predict the treatment failure, unless the tubal rupture is observed [226].

The following instructions should be followed after medical management

1) Patients are advised to avoid the sexual intercourse until beta-hCG level becomes negative,
2) Avoid alcohol use, non steroidal anti inflammatory drugs, foods and multi vitamins containing folic acid,
3) Avoid sun exposure to minimize the risk of dermatitis,

4) Patients should not get pregnant for at least 3 months because of the risk of teratogenicity.

Life-threatening complications are rare. Many patients complain of pain between 3 to 7 days after the onset of treatment, but it usually resolves within 4 to 12 hours [227]. In acute onset of pain, tubal rupture should be ruled out .An emergency surgery should be performed if tubal rupture is suspected.

Signs suggesting treatment failure or possible rupture include

1) Increasing abdominal pain, regardless of the beta-hCG levels.
2) Haemodynamic instability and
3) Rapidly rising beta-hCG concentrations after methotrexate treatment.

Some commonly encountered treatment effects of methotrexate are

1) Increase in beta-hCG level during the initial few days of therapy,
2) Increase in abdominal girth,
3) Bleeding or spotting per vaginum and
4) Abdominal pain between day 3 to day 7 after the onset of treatment.

Predictive factors of success

Initial beta-hCG level before the onset of treatment is the single most important factor in the prediction of treatment success. The invasion of the trophoblastic tissue in ectopic pregnancy can be predicted from the

serum beta-hCG levels. The higher the serum beta-hCG level, the greater is the invasion of the trophoblastic tissue and the lesser is the treatment success with MTX [63]. If the serum beta-hCG level is low, then the chance of treatment success is high.

The increase in beta-hCG concentrations before the onset of treatment and following MTX treatment are the more accurate predictors of tubal rupture than the level of beta-hCG on the day of MTX therapy.

Ultrasound imaging is another important consideration. Better results are observed with haematosalpinx and the worse results are observed with a live embryo.

Other predictors of treatment success are

1) Size of the gestational mass less than 3.5 cm,
2) Endometrial thickness less than 7 mm, and
3) Absence of intra peritoneal bleeding [221,228].

Therefore, predictors of treatment failure include

1) The presence of moderate or large amount of free fluid in the peritoneal cavity.
2) Initial beta-hCG levels higher than 5000 mIU/ml,
3) The pretreatment rise in the serum beta hCG level of more than 50% over a 48 hours period,
4) The presence of cardiac activity in the embryo (ASRM Practice Committee, 2008).

Reproductive future

The fertility outcome of conservatively treated patients with MTX for an unruptured ectopic pregnancy can be evaluated by hysterosalpingography or by spontaneous conception [211,229] .The radiologically normal tubes after medical treatment are shown to be associated with less risk of ectopic gestation in future pregnancy. However, the normal radiological findings show nothing about the tubal function, where tubal dysfunction can also be a cause of ectopic pregnancy.

Lipscomb et al. (2000) reported that 65% of patients succeeded with subsequent intra uterine pregnancies after medical management. The incidence of recurrent ectopic pregnancy in the study population was around 13% [230].

Surgically administered medical management

In a surgically administered medical management methotrexate is administered locally into the gestational sac either transvaginally under the ultrasound guidance or under the laparoscopic guidance. Local treatment is aassociated with less adverse effects of systemic methotrexate .

Drugs that can be used for local treatment are

1) Methotrexate 1mg/kg [231,232,233],
2) KCl

3) Prostaglandins, and

4) Hyperosmolar glucose.

Feichtinger and Kemeter first demonstrated the local treatment of an ectopic pregnancy with MTX in 1987 .They injected 10 mg of MTX into the gestational sac of tubal ectopic pregnancy transvaginally under the ultrasound guidance. That case was successfully treated without adverse effects [231].

Selection criteria

Surgically administered medical management is preferred in ectopic pregnancies with embryonic cardiac activity, particularly in cases of extra tubal ectopic pregnancies [207]. Systemic MTX treatment is preferred in ectopic pregnancies without embryonic cardiac activity. Local injection of MTX is comparatively safe and equally effective as systemic methotrexate, this local approach requires an additional invasive procedure which needs expertise and additional cost.

An important consideration is that the correct dose of MTX used for local treatment is yet to be determined [234,235,236]. Most commonly used dosage is 1mg/kg body weight.

Compared to local treatment, systemic methotrexate can be used easily. Therefore, local administration of MTX is restricted to non-tubal ectopic pregnancies with embryonic cardiac activity [237].

Follow-up

Serum beta-hCG levels are done on day 4 and day 7 after the MTX injection. Any patient with less than 15% fall in beta-hCG levels between day 4 and day 7, should be given an additional 50 mg/m^2 intramuscular dose of MTX. Local treatment is less effective in patients with high initial levels of serum beta-hCG and an additional injection of systemic MTX will improve the results. Hysterosalphingography should be performed after 3 months.

Surgical Treatment

Until the last 2 decades, ectopic pregnancies were usually treated by total salpingectomy (removal of the entire tube) via laparotomy. Today, laparoscopic surgery is preferred for treatment of ectopic pregnancies.

Laparotomy is reserved for

1) Ruptured ectopic pregnancies with severe intra abdominal bleeding, or
2) In the presence of extensive adhesions.

If the ectopic gestation is diagnosed early, before the tubal rupture, a laparoscopic salpingostomy can be performed. In this technique, the fallopian tube is opened and the products of conception are removed while leaving the tube in place. The tube then heals on its own.

A partial salpingectomy, also known as segmental resection can be performed in small ectopic gestation with the healthy fimbrial ends. In case of partial salphingectomy the remaining segments of the tubes can be reunited later using micro surgery.

If the tubes are extensively damaged, or if the ectopic pregnancy is large and bleeding profusely, a total salpingectomy should be performed.

In some cases when the ectopic gestation involves the ovary, a portion of the ovary or the entire ovary needs to be removed along with the fallopian tubes. This is called salphingo oophorectomy.

Surgical management should be done in all cases of ruptured ectopic pregnancy.

The laparoscopic surgery is preferred to an open surgery in haemodynamically stable patients. Laparoscopic surgery has many advantages as mentioned below.

1) Lower analgesia requirements,
2) Shorter operative times,
3) Less intra operative blood loss and
4) Shorter duration of hospital stay [238,239,240].

Laparotomy should be reserved for ruptured ectopic pregnancies with haemodynamic instability.

If the opposite tube is healthy, total or partial salpingectomy is preferred depending upon the condition of the tube.

Studies have reported that the subsequent rate of intra uterine pregnancy is not increased after salpingostomy compared to salpingectomy in patients with healthy contralateral tube. In addition, salphingostomy exposes the women to the risk of bleeding and persistent trophoblastic tissue [173]. According to current guidelines, the procedure of choice in the presence of a healthy contralateral tube is laparoscopic salpingectomy.[241]

In the presence of diseased contralateral tube, a laparoscopic salpingostomy should be considered if the patient desires future fertility**.** **Persistent trophoblast** is the problem of concern after salpingostomy. Persistent trophoblast should be considered in the following situations.

1) Where the size of the ectopic pregnancy is more than 2 cm,
2) Presence of active tubal bleeding and
3) If serum β-hCG concentrations are more than 3000 IU/L or rising prior to the surgery [242]**.**

Women with persistent trophoblast should be monitored with serial β-hCG estimation and sometimes systemic methotrexate therapy may be needed if there is inadequate fall [243].

Many clinicians prefer a salpingectomy because this procedure is easier to perform and less time consuming than a salpingostomy.

Management of an Interstitial ectopic pregnancy

The surgical management ranges from exploratory laparotomy and cornual wedge resection to hysterectomy in case of uncontrolled bleeding.

The ultrasound criteria for diagnosing an interstitial ectopic pregnancy are

1) Absent intrauterine gestational sac.
2) Eccentrically placed gestational sac,
3) The presence of a separate chorionic sac at least 1 cm from the lateral edge of the uterine cavity, and
4) The gestational sac with the surrounding myometrium with a thickness of atleast 5mm [244,245].

Haemodynamically stable patients with unruptured early interstitial pregnancies could be managed conservatively with intramuscular MTX injection or surgically administered medical treatment.

Management of cervical ectopic pregnancy

The classical treatment for a case of cervical ectopic pregnancy is hysterectomy. But, hysterectomy is the cause of significant morbidity and infertility. There is high chance of massive bleeding and urinary tract injuries.

The vaginal bleeding can be managed conservatively by

1) Local haemostatic stitches,
2) Curettage,
3) Uterine artery embolization and
4) Application of an intracervical balloon tamponade [246,247].

However, these conservative procedures are often ineffective and result in hysterectomy in majority of the patients.

The early asymptomatic cases of cervical ectopic pregnancies can be managed medically using local or systemic methotrexate [248,249].

Factors associated with the failure of systemic MTX treatment in cervical ectopic pregnancy are

1) The beta hCG levels more than 10,000 mIU/ml,
2) More than 9 weeks of gestation,
3) The presence of detectable embryonic cardiac activity and
4) The crown-rump length of more than 10 mm [250].

A combination therapy with intra-amniotic methotrexate injection increases the success rate.

Management of caesarean scar pregnancy

Period of gestation determines the management. A caesarean scar pregnancy can be managed by any of the following methods.

1) Expectant management,
2) Medical treatment and

3) Surgical management.

Laparotomy and hysterectomy is the management of choice for haemodynamically unstable patients presenting with shock. In haemodynamically stable patients the scar thickness should be assessed before the administration of medical management. Non-surgical procedure is appropriate when the trophoblast reaches the vesico-uterine space. Expectant management has high risk of subsequent rupture. In medical management, the methotrexate injection can be given locally, systemically or in combination.

The risk of scar rupture and heavy bleeding are seen in medical management. So it should be combined with either

1) Intracervical vasopressin injection in combination with 18 Foley catheter balloon tamponade or
2) Bilateral uterine artery embolization [251].

Management of an Ovarian pregnancy

When diagnosed early, ovarian pregnancies can be managed by laparoscopic surgeries. Medical management with MTX can be used in carefully selected patients with ovarian pregnancy, but the selection criteria are not defined. The systemic MTX may also be used to treat the persistent trophoblastic tissue after laparoscopy [252].

Management of abdominal pregnancy

Treatment is easy in earlier stage of pregnancy. The following ultrasound criteria are suggested as diagnostic

1) Absence of an intrauterine pregnancy.
2) Absence of both tubal mass and a complex adnexal mass,
3) A wide mobility similar to fluctuation of the sac, evident with pressure of the transvaginal probe towards the POD.
4) A gestational cavity surrounded by bowel loops and separated by peritoneum,

MRI is more reliable in the diagnosis of this condition.

Before surgery be ensure that

1) Multidisciplinary surgery team is ready,
2) Pre-operative arterial embolization done,
3) Blood and blood products are adequately available and
4) Bowel is prepared.

The traditional management was laparotomy and removal of the fetus with or without placenta [253]. Risk of torrential bleeding from the placental bed is an important consideration in removing the abdominal pregnancies after first trimester. Adjuvant methotrexate therapy along with the pre operative arterial embolization is suggested to control this bleeding [254].

Management of heterotopic pregnancy

More than 50% of the heterotopic pregnancies present late after rupture. Surgery is usually required and MTX usage is contraindicated in heterotopic pregnancies [255,256]. In case of heterotopic pregnancies with a viable intrauterine pregnancy and a non-tubal pregnancy with embryonic cardiac activity, KCL can be injected locally under ultrasound guidance. But, the follow-up after medical management is difficult because serum beta-hCG cannot be used as a reliable marker and ultrasound is not a good method to follow up the patient.

MATERIALS AND METHODS

Study area

The study was conducted at Tirunelveli medical college hospital. TVMCH is a tertiary care centre and a teaching hospital for undergraduate and post graduate students. It is a main referral centre for all primary health centres and government hospitals, situated in Tirunelveli ,Tuticorin and Kanyakumari districts.

Study design

The study was a cross-sectional study.

Sample size

The study consists of ‘n’ no of ectopic pregnancy cases attended Tirunelveli Medical College Hospital Obstetrics and Gynaecology department for a particular time period.

Duration of the study

From July 2012 to June 2014 for a period of 2 years.

Inclusion criteria

All cases of ectopic pregnancy / suspected ectopic pregnancy by clinical or sonological method.

Exclusion criteria

The study involved all cases of ectopic pregnancy. There was no exclusion criteria.

Methods and materials

Data collection tool was used to collect the different information. Face to face interviews were conducted using data collection tool by the investigator including detailed history taking and relevant physical examination.

A detailed history was taken from the patient (if the patient was in shock the history was taken retrospectively). After taking history physical examination was done especially for vital signs, abdominal examination, per vaginal examination, cervical excitation test and culdocentesis when needed.

The basic investigations including haemoglobin, renal function test, blood grouping and Rh typing, urine pregnancy test and ultrasound examination were done in all patients. Additional investigations like serum beta hCG, doppler study, CT, MRI were ordered in case of doubtful diagnosis.

All data were collected on a structural data form (sample enclosed) and analysed for descriptive statistics. Information regarding patient profile , risk factors , sterilisation status , use of other contraceptive methods , presenting symptoms and signs , physical examination , ultrasound findings , types of treatment , per operative findings , no of transfusions , post operative morbidity and length of hospital stay were analysed.

Post operatively HPE reports were collected from the pathology department and final diagnosis was made.

Data were summarised in tables and figures. For calculation of sensitivity, specificity, positive and negative predictive values for the diagnosis of ectopic pregnancy, 2- by-2 contingency tables were used.

Ethical clearance

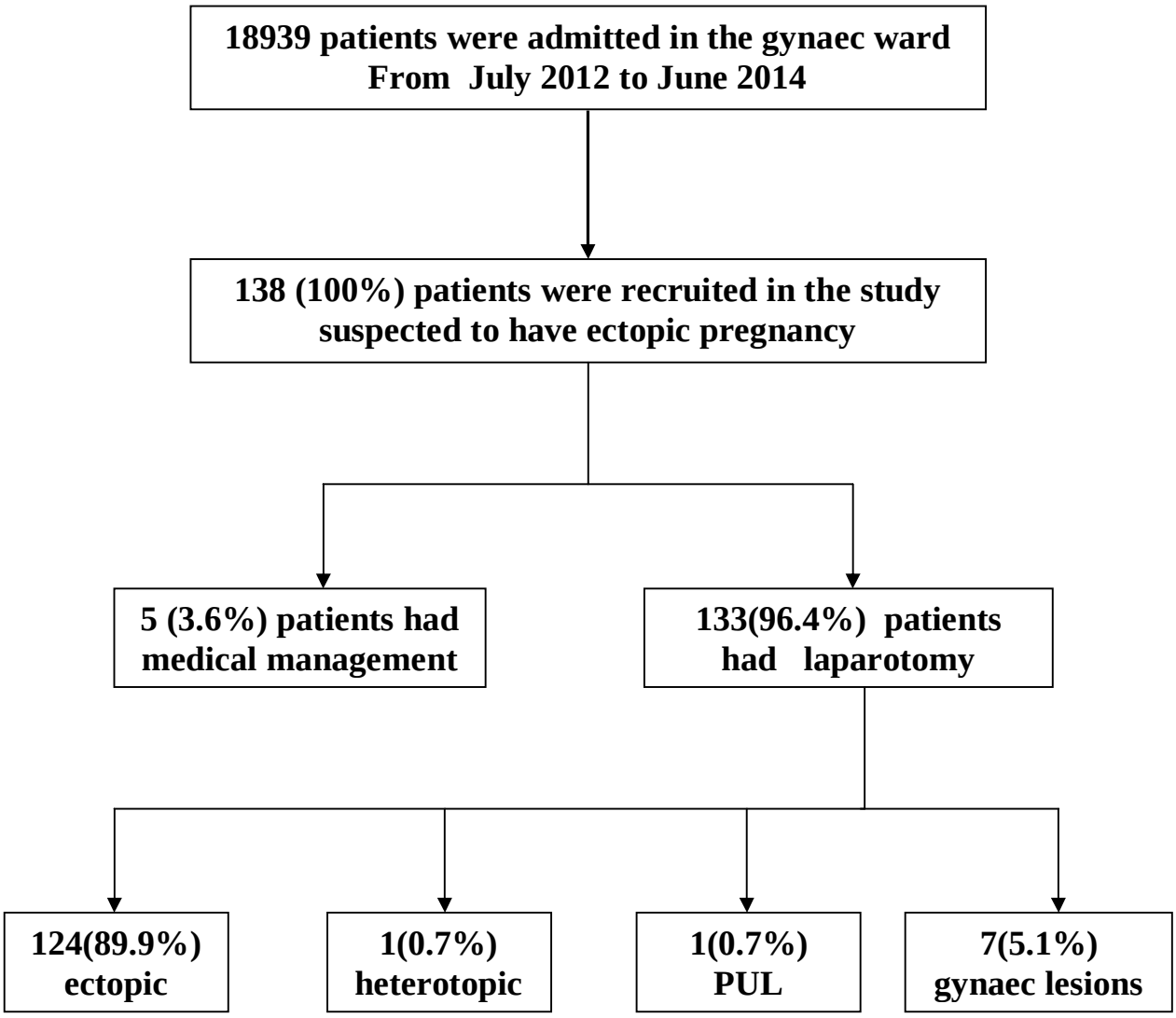
Ethical clearance was obtained from the TVMCH ethical committee. Permissions from the head of the departments of Obstetrics and Gynaecology, Radiology and Pathology were obtained in written form prior to the ethical clearance. All details of the patients remained confidential. All patients were treated according to the hospital protocol.

RESULTS

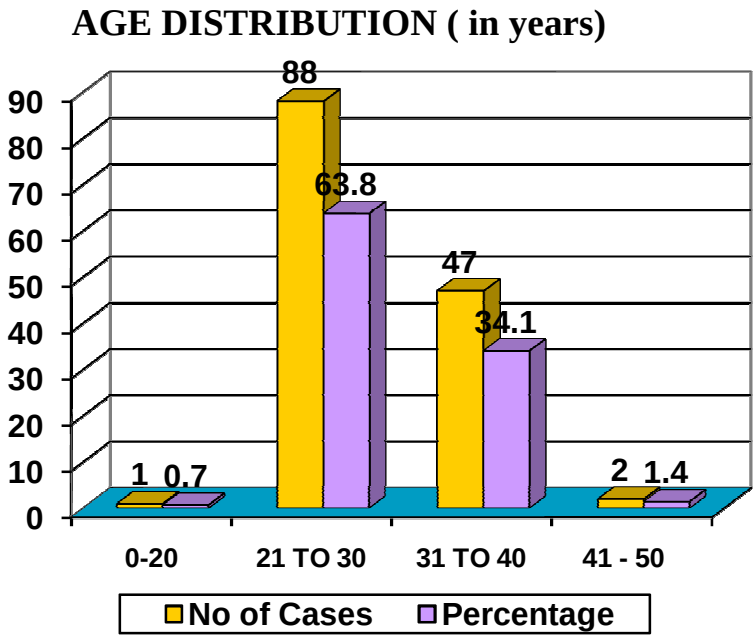
A total of 138 patients were recruited in the study. The study population was estimated to be 0.94% of total deliveries (14,688) and 0.73% of total gynaec ward admissions (18,939) during the study period.

5 patients in the study had undergone medical management. Remaining 133 patients had undergone laparotomy. During laparotomy 124 patients were found to be having ectopic pregnancy which was confirmed by histopathology. One patient had heterotopic pregnancy. One patient had negative laparotomy. Remaining 7 patients had other gynaecological lesions.

ENROLLMENT CHART

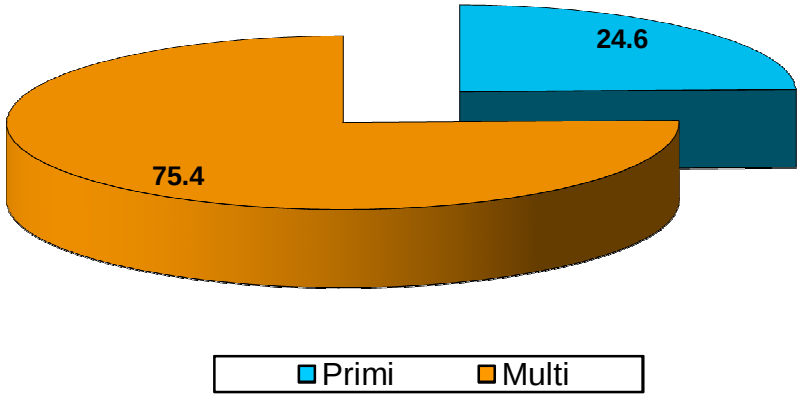


AGE OF THE STUDY PARTICIPANTS



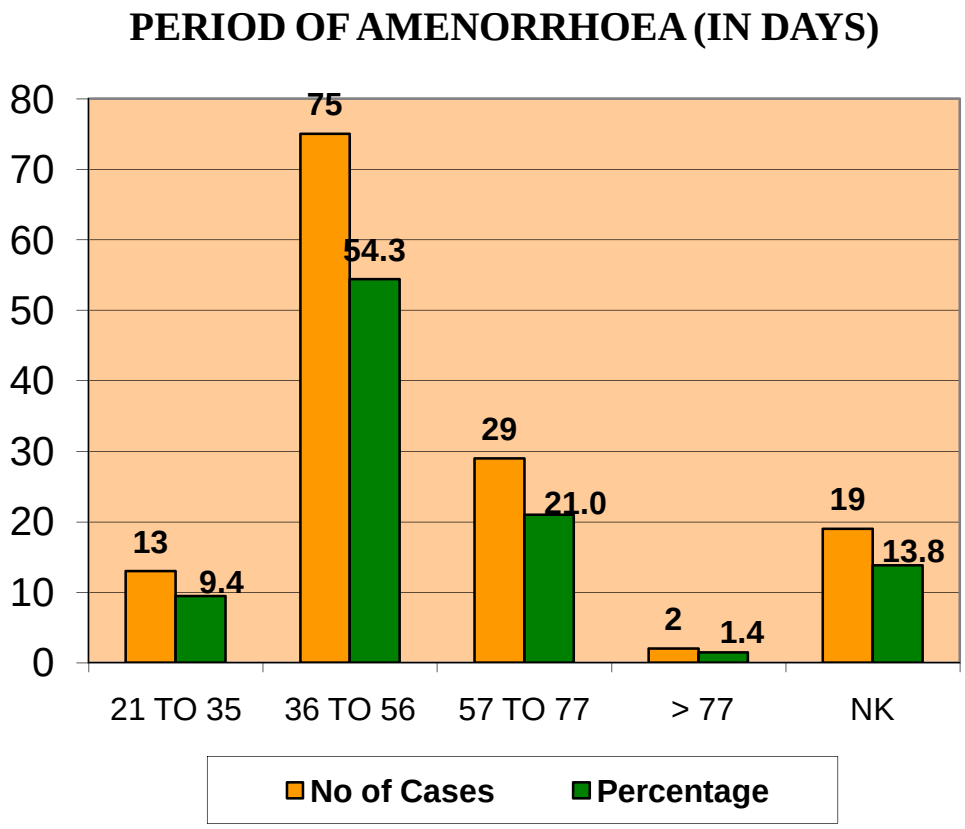
63.8% of the study population belongs to 21 to 30 years of age. 49 (35.55%) patients were more than 30 years of age.

OBSTETRIC CODE



Among the study population 74.5% were multigravida and 24.6% were primigravida.

PERIOD OF AMENORRHOEA



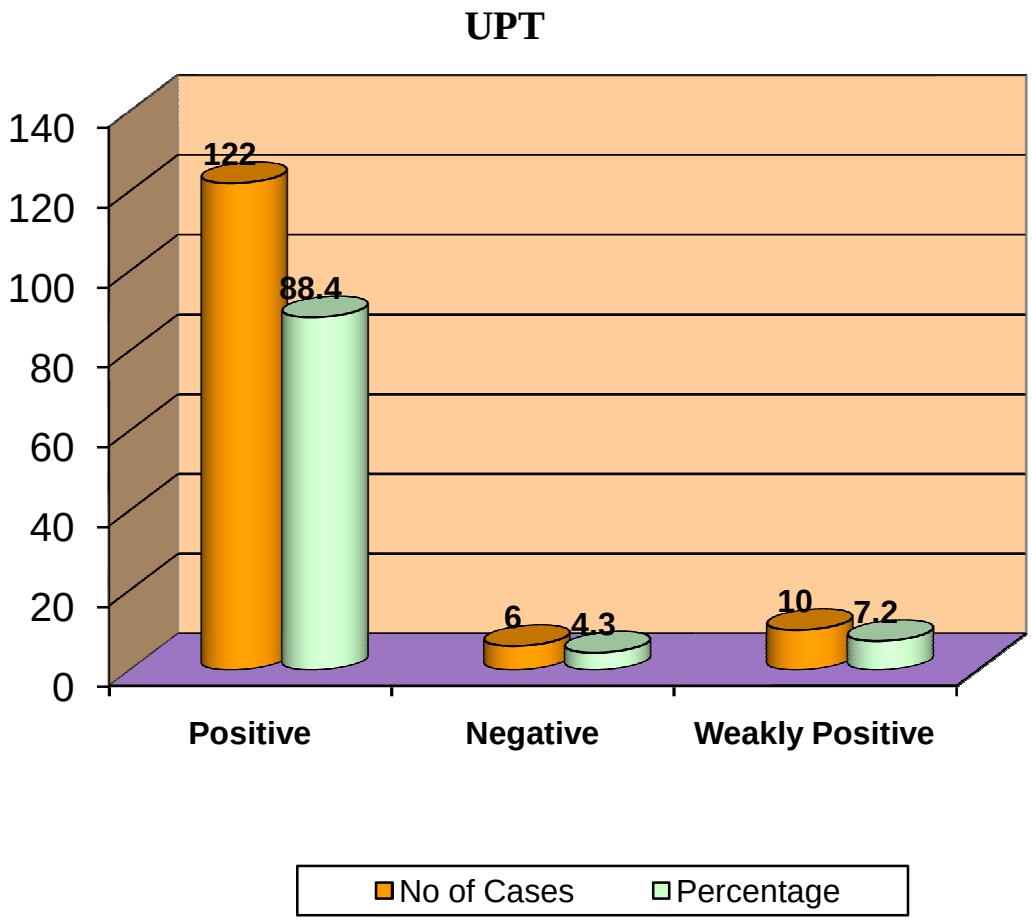
LMP was not known in 19(13.8%) patients .

75 (54.3%) patients were presented with >5 weeks to 8 weeks of amenorrhoea.

29 (21.0%) patients were presented with >8 weeks to 11 weeks of amenorrhoea.

2 (1.4%) patients had more than 11 weeks of amenorrhoea.

URINE PREGNANCY TEST

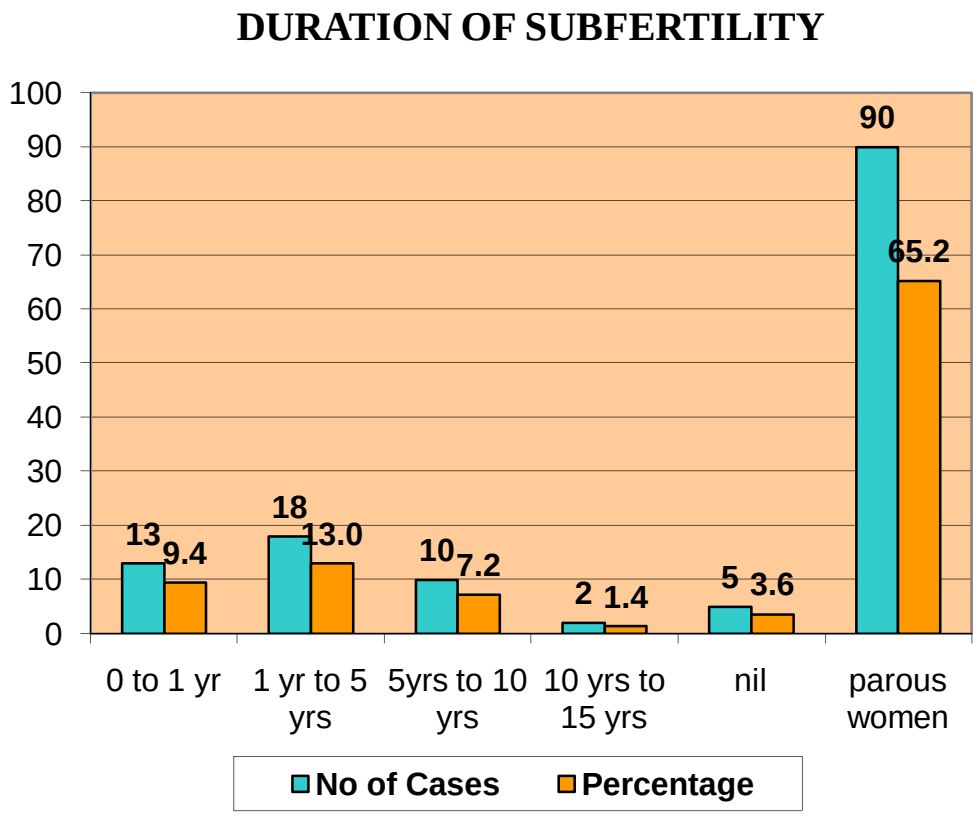


Urine pregnancy test was positive in 122 patients (88.4%).

Negative in 6 patients (4.3%).

Weakly positive in 10 patients (7.2%).

DURATION OF SUBFERTILITY



13% of women had 1 to 5 yrs of subfertility.

7.2% of women had 5 to 10 yrs of subfertility.

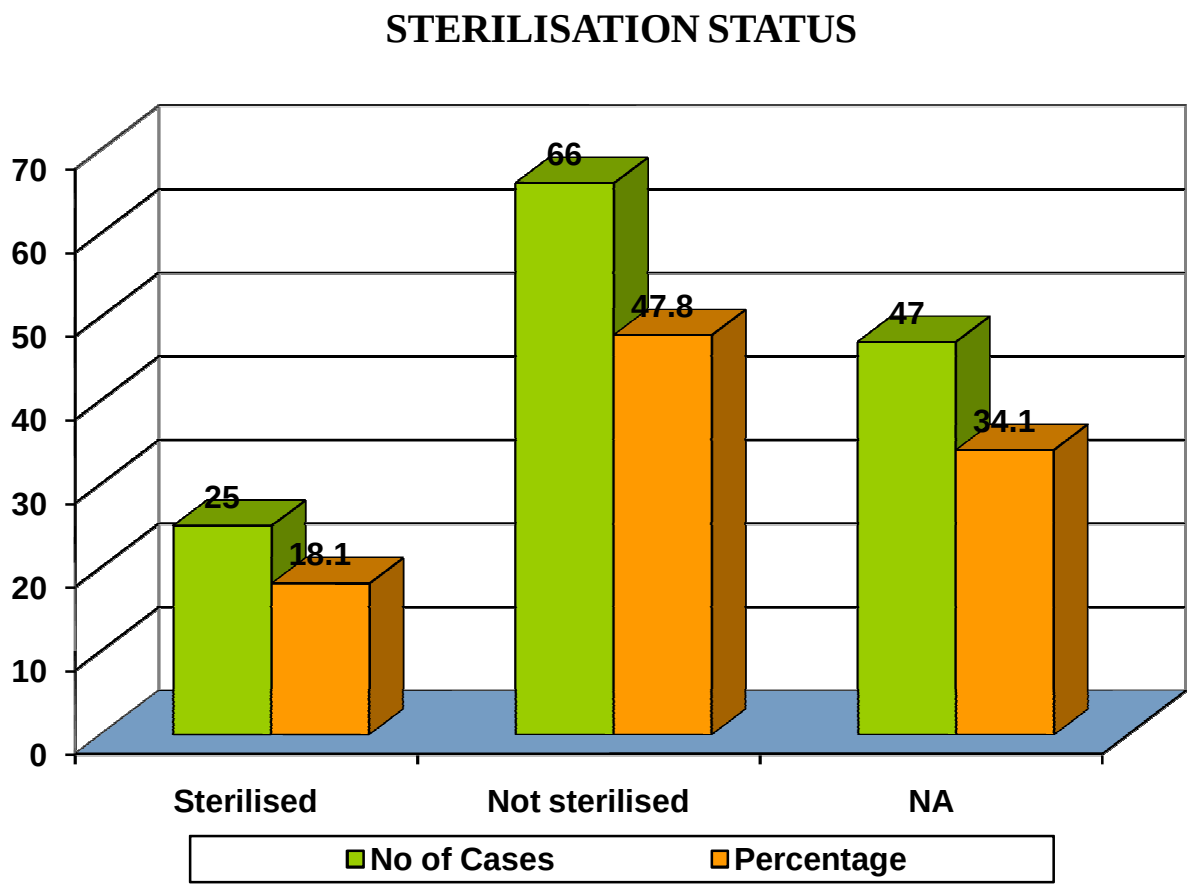
1.4% of women had 10 to 15 yrs of subfertility.

3.6% of primi gravida conceived immediately after marriage.

9.4 % of primi gravida conceived within one year.

65.2% of patients were parous women with atleast one live child.

STERILISATION STATUS

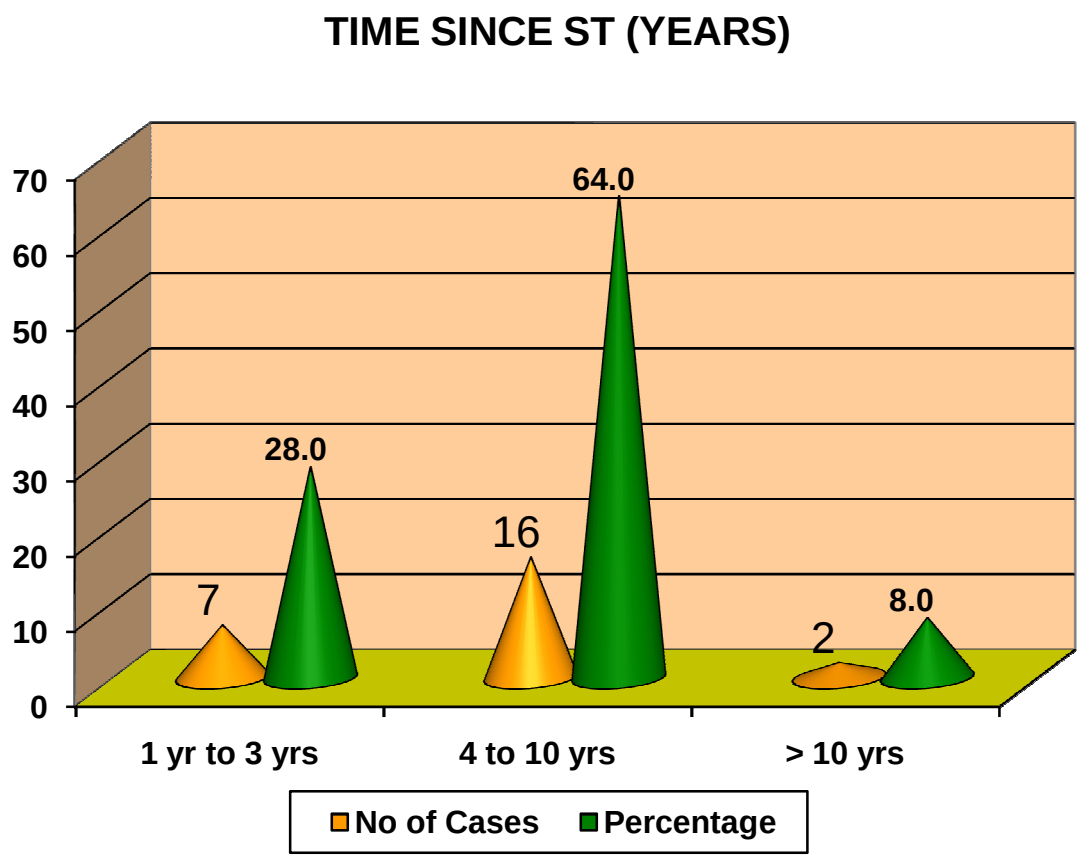


Among the parous women with atleast one live child 25(18.1%) were sterilised and 66 (47.8%) were not sterilised.

NA- not applicable.

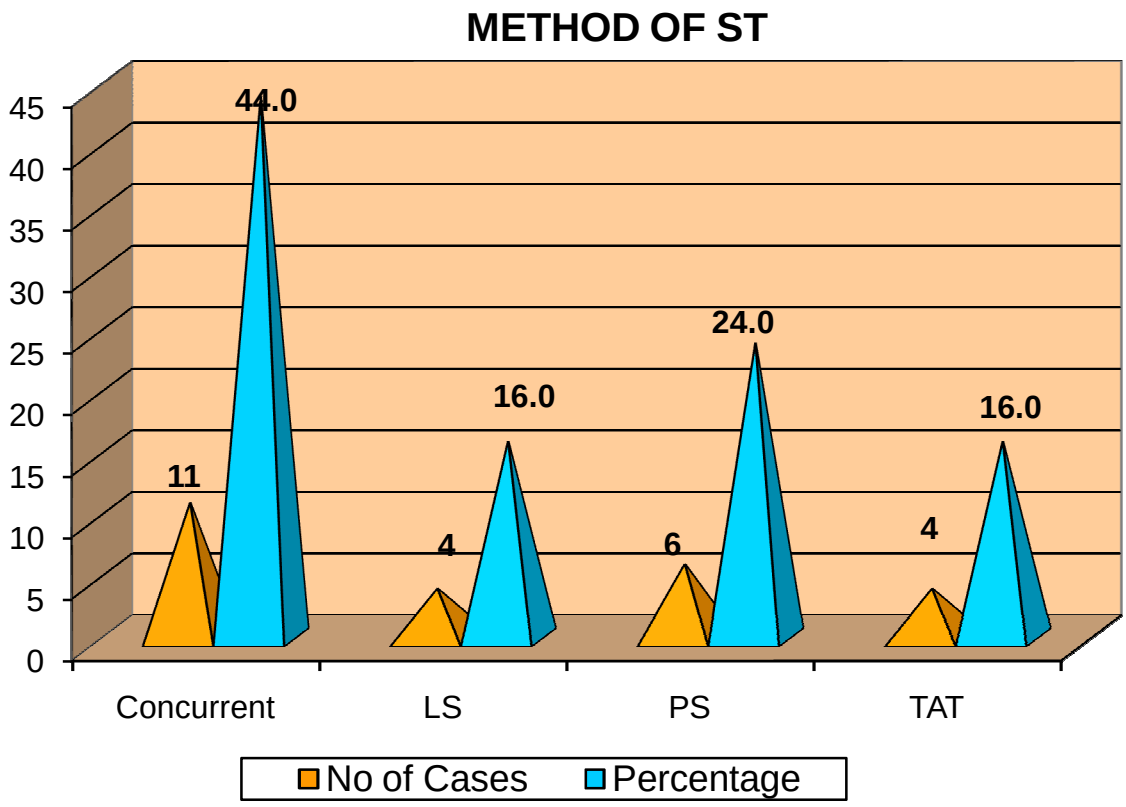
47 (34.1%) patients are primi gravida and multi gravida with no live child.

TIME SINCE STERILISATION



The duration between the sterilisation and the occurrence of ectopic pregnancy was 1-3 yrs in 7 (28%) patients, 4 to 10 yrs in 16(64%) patients , > 10 yrs in 2(8%) patients.

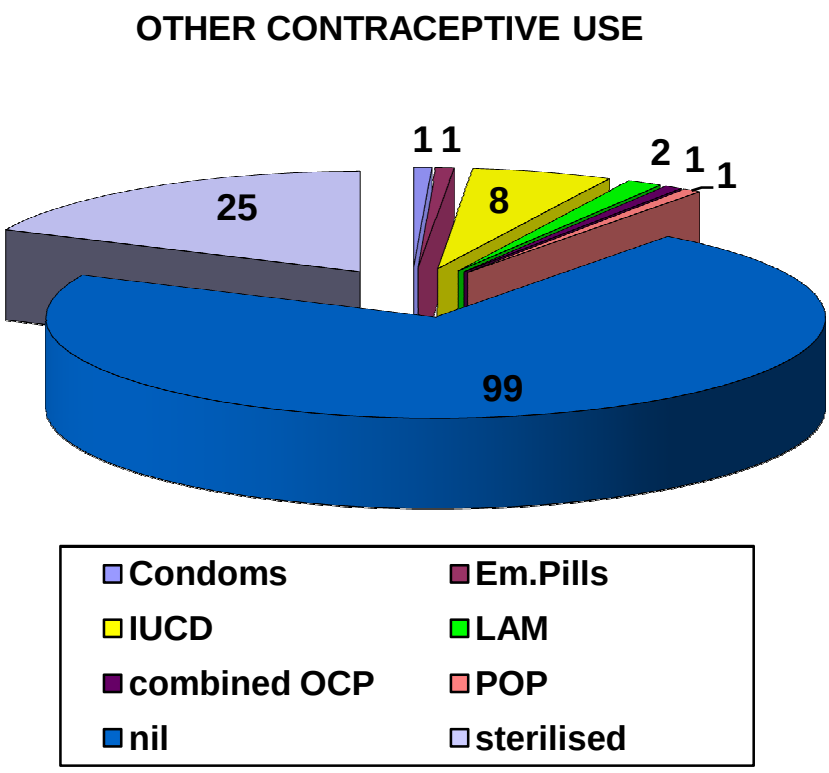
METHOD OF STERILISATION



Among the sterilised women 11(44%) patients had concurrent sterilisation,

4 (16%) patients had laparoscopic sterilisation, 6 (24%) patients had puerperal sterilisation and 4(16%) patients had total abdominal tubectomy.

OTHER CONTRACEPTIVE USE



99 patients did not use any contraceptive method .

8 patients gave history of IUCD use, but only one patient conceived with IUCD in situ.

1or 2 patients gave history of usage of condom , emergency pills , oral contraceptive pills .

2 patients were conceived during lactational amenorrhoea.

RISK FACTORS

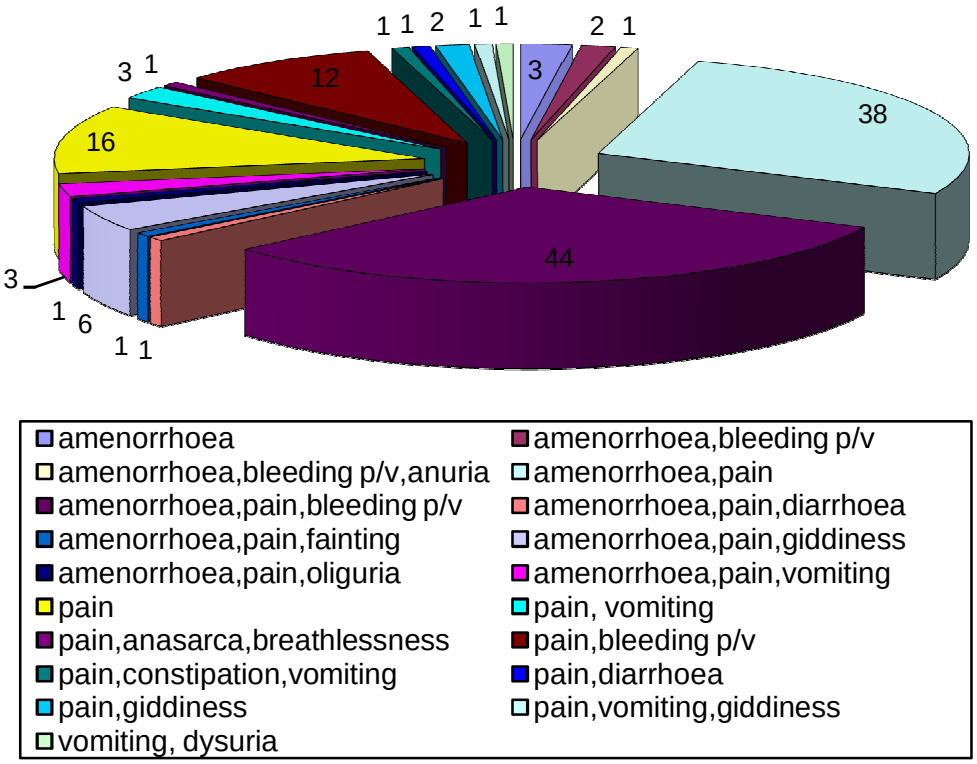
s. no	Risk factors	No of patients
1	Prior H/O abortions	25
2	Prev LSCS	44
3	Sterilisation	25
4	Tubal recanalisation	1
5	Other pelvic surgeries	1
6	Prev ectopic	6
7	PID	19
8	Ovulation induction	7
9	ART	3
10	IUCD	8
11	Emergency pills	1
12	MTP pills	6
13	Combined OCP	1
14	POP	1
15	No risk factors	37

Previous LSCS was the most common risk factor followed by tubal surgeries, abortions and PID. In some cases more than one risk factor was seen.

No risk factors were identified in 37 (26.8%)cases.

One or more risk factors were identified in 101 (73.2%) cases.

PRESENTING SYMPTOMS.



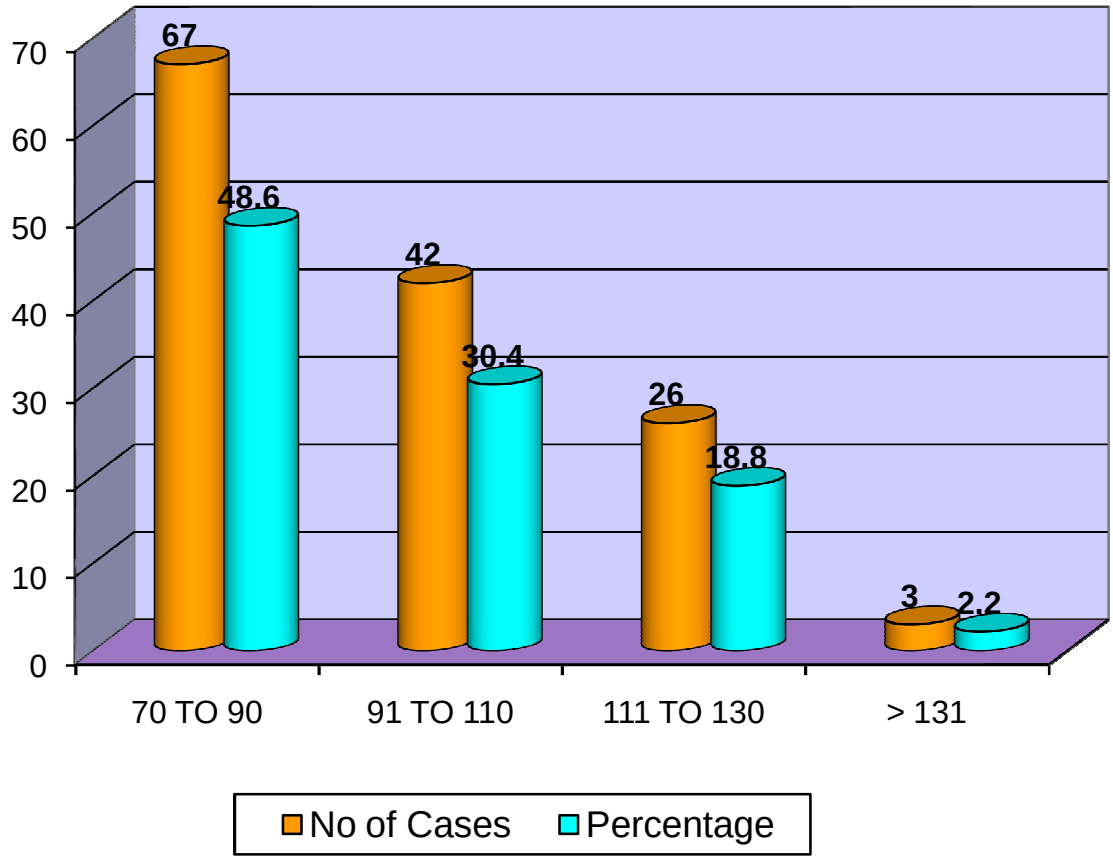
Classic triad of ectopic pregnancy was seen only in 44 cases.

Gastrointestinal symptoms , urinary symptoms and vaso vagal symptoms were reported in some cases.

CLINICAL SYMPTOMS

S.NO	CLINICAL SYMPTOMS	NO OF PATIENTS	Percentage out of 138 pts
1	Amenorrhoea	100	72.5
2	Abdominal pain	131	94.9
3	Bleeding p/v	59	42.8
4.	Amenorrhoea , abdominal pain	94	68.1
5	Amenorrhoea , bleeding p/v	47	34.1
6	Abdominal pain , bleeding p/v	56	40.1
7	Amenorrhoea , abdominal pain , bleeding p/v	44	31.9
8	Vaso vagal symptoms	10	7.2
9	Gastro intestinal symptoms	11	8.0
10	Urinary symptoms	3	2.2
11	Anasarca , breathlessness	1	0.7

PULSE RATE



Tachycardia was present in 71(51.4%) patients.

PULSE VOLUME

Pulse volume	No of patients	percentage
Normal	129	93.5
feeble	9	6.5

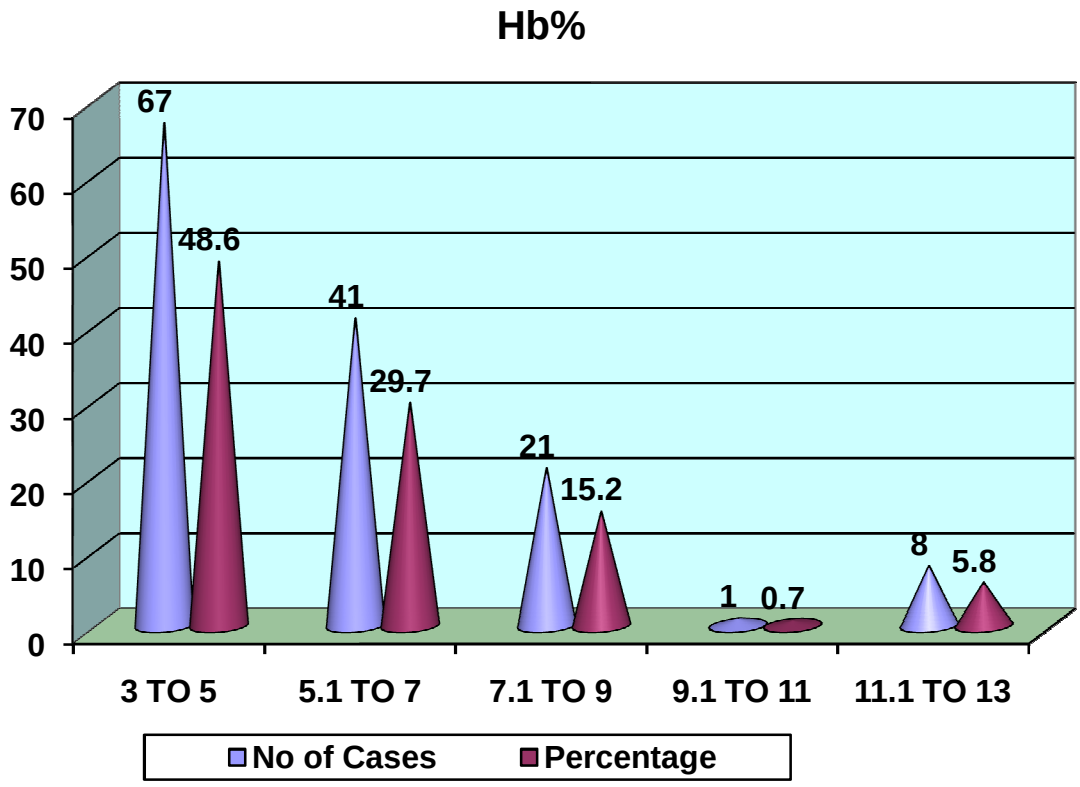
BLOOD PRESSURE

Blood pressure in mmHg	No of Patients	Percentage
< 90/60	17	12.3
90/60 or more	121	87.7

Hypotension was present in 17 (12.3%) patients.

Among them 9 (6.5%) patients were presented with shock.

HAEMOGLOBIN LEVEL

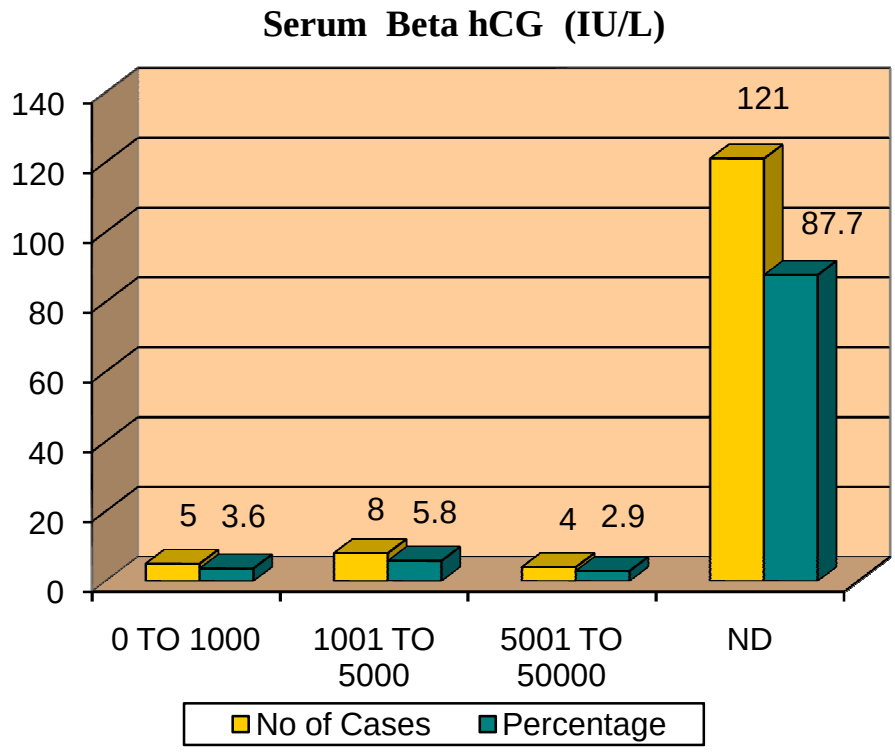


67 (48.6%) patients had 3 to 5 gm% of Hb.

41 (29.7%) patients had Hb concentration between 5.1 to 7 gm%.

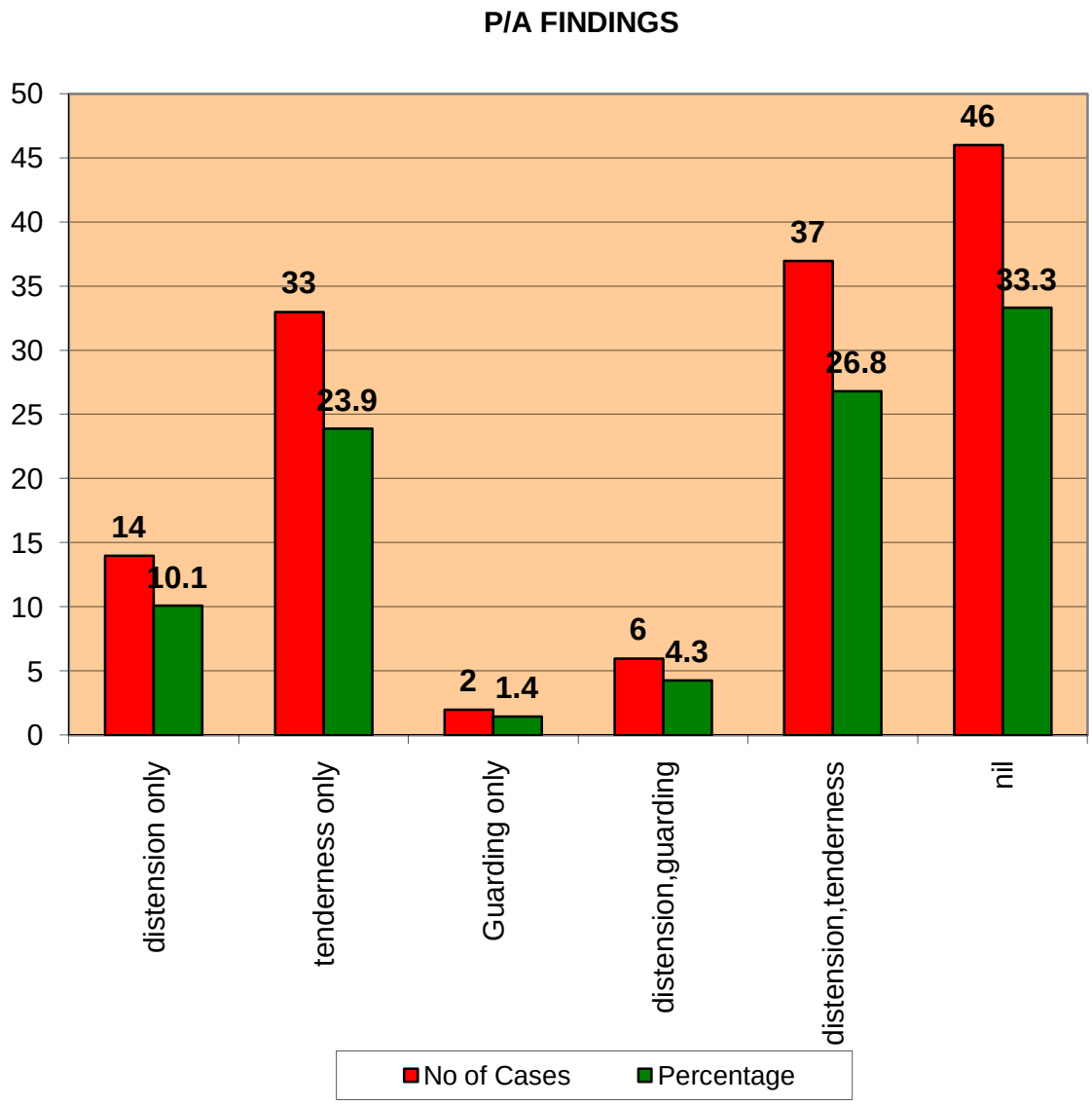
In total , 108 (78.3%) patients were severely anaemic. 21 (15.2%) patients were moderately anaemic. 1 (0.7%) patient was mild anaemic. 8 (5.8%) patients were not anaemic.

SERUM BETA hCG



Sr. beta hCG was not done in 121 (87.7%) patients . Sr. beta hCG was done in 17 (12.3%) patients with doubtful diagnosis. Among them 13 patients(9.4%) had value of < 5,000 IU/ L and 4 (2.9%) patients had >5,000 IU / L.

PER ABDOMINAL EXAMINATION FINDINGS



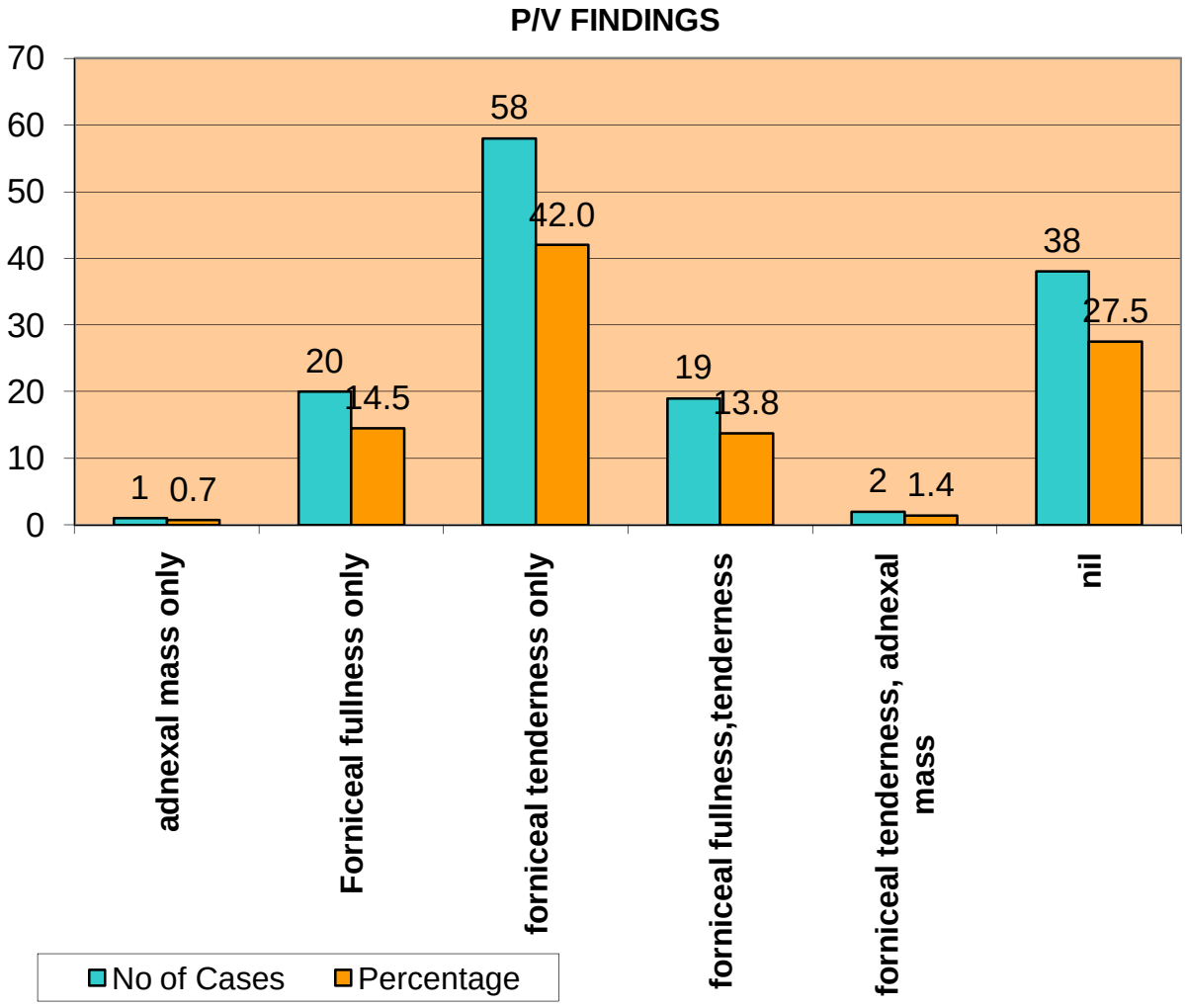
46 (33.3%) patients had no abdominal findings . Others had one or more abdominal findings as mentioned above.

Abdominal distension was present in 57 (41.3%) patients .

Abdominal tenderness / guarding was present in 78 (56.5%) patients .

Abdominal distension with tenderness / guarding was present in 43 (31.2%) patients .

PER VAGINAL EXAMINATION FINDINGS



On per vaginal examination 38 (27.5%) patients had no positive findings . Others had one or more positive findings as mentioned above .

Forniceal fullness was present in 39 (28.3%) patients.

Forniceal tenderness was present in 79 (57.2%) patients .

Adnexal mass was present in 3 (2.8%) patients.

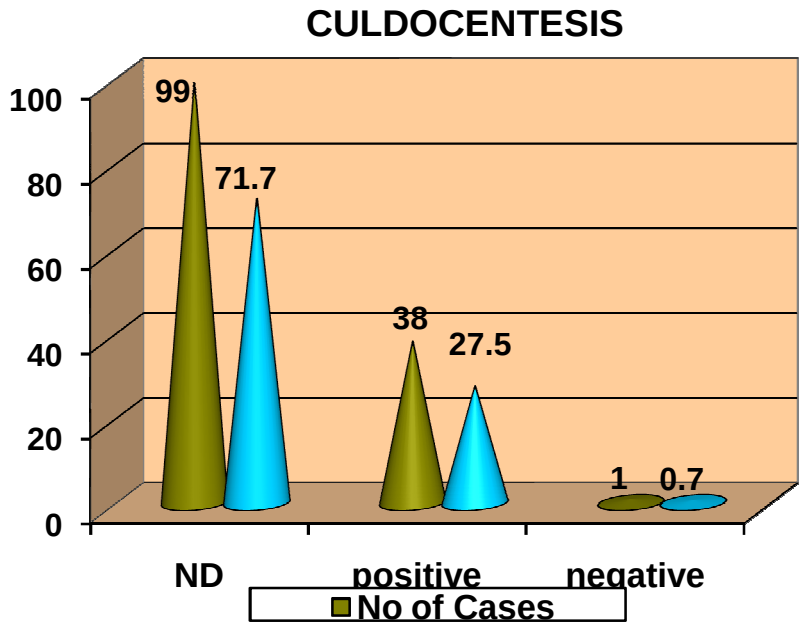
Forniceal fullness and tenderness was present in 19 (13.8%) patients .

Forniceal tenderness and adnexal mass was present in 2 (1.4%) patients .

CERVICAL EXCITATION TEST

Cervical excitation test was done in all 138 patients.

Cervical excitation test	No of patients	Percentage
Positive	100	72.5
negative	38	27.5
total	138	100



Culdocentesis was not done in 99 (71.7%) patients .

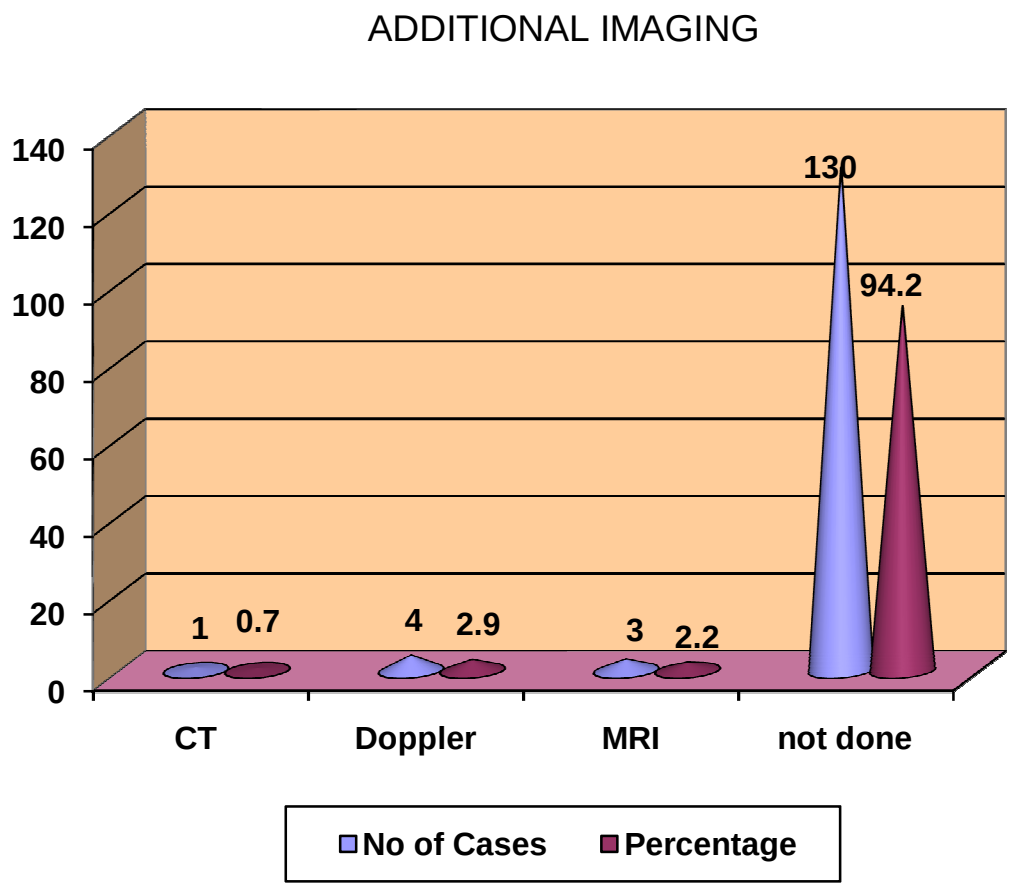
Culdocentesis was done in 39 (28.3%) patients . Among them 38 (27.5%) patients were positive and 1 (0.7%) patient was negative .

ULTRASOUND FINDINGS

Routine ultrasound examination including both transabdominal and transvaginal scans were done in all patients on admission .

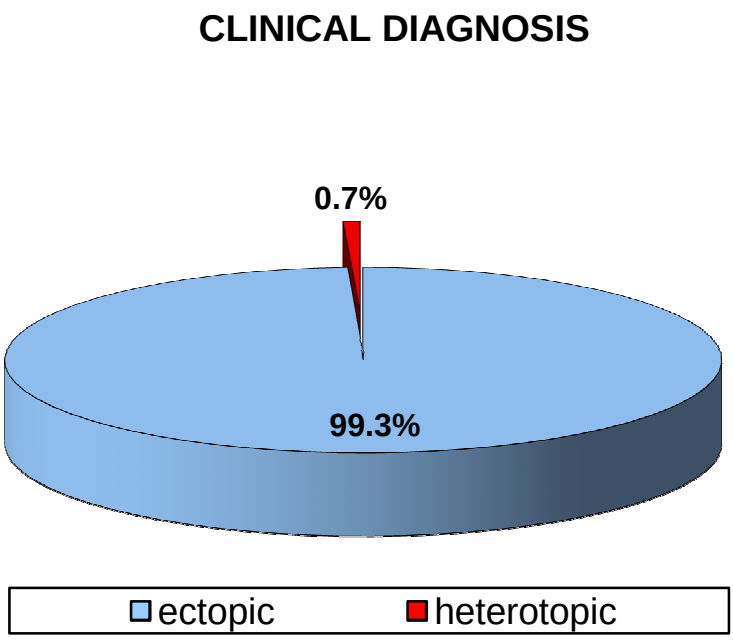
Ultrasound findings	No of patients	Percentage
Empty uterus , adnexal mass	15	10.9
Empty uterus , free fluid	34	24.6
Empty uterus , adnexal mass , free fluid	68	49.2
Normal study	2	1.4
Bicornuate uterus with free fluid	1	0.7
Bicornuate uterus with no G.sac	1	0.7
Bicornuate uterus with G.sac in both horns	1	0.7
Intra uterine G.sac with free fluid	1	0.7
Empty uterus,Extra uterine G.sac with FP and FHR	7	5.1
Empty uterus,extra uterine G.sac with FP without FHR	1	0.7
Empty uterus, extra uterine G.sac without FP	3	2.2
Empty uterus with ovarian follicle	2	1.4
IUCD in situ , adnexal mass, free fluid	1	0.7
Empty uterus , adnexal mass ,localised fluid collection	1	0.7
Total	138	

ADDITIONAL IMAGINGS



In addition to routine ultrasound done in all patients , CT was taken in one patient , Doppler was done in 4 patients . MRI was taken in 3 patients .

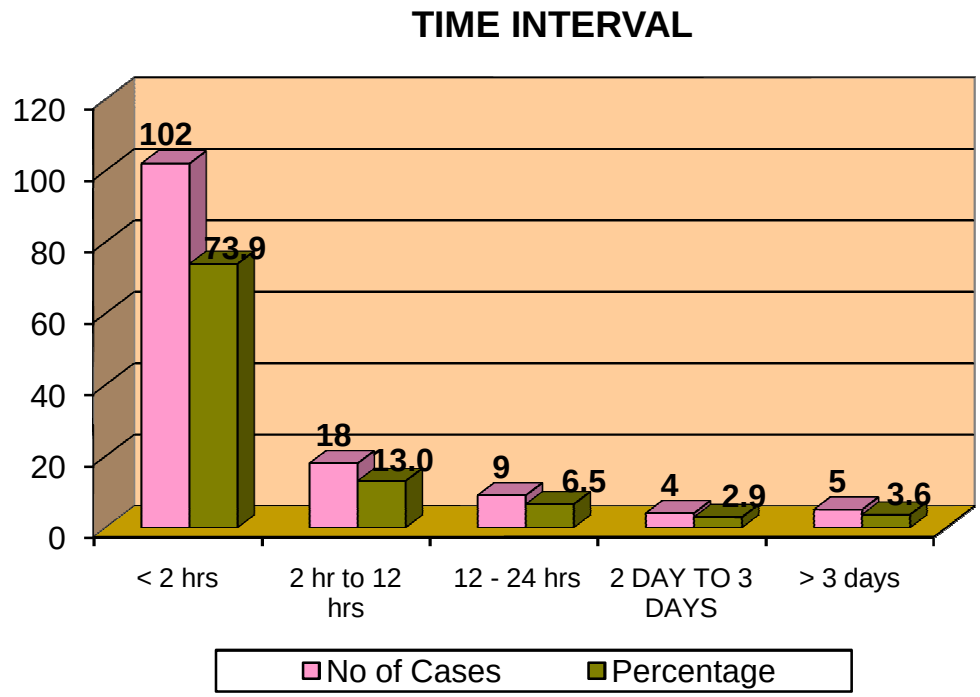
CLINICAL DIAGNOSIS



Clinical diagnosis was made after history taking , physical examination , urine pregnancy test , routine ultrasound examination and ,if necessary additional imagings and culdocentesis in some cases.

Clinical diagnosis was ectopic pregnancy in 137 (99.3%) patients and heterotopic pregnancy in 1 (0.7%) patient .

TIME INTERVAL BETWEEN ADMISSION AND ONSET OF DEFINITIVE TREATMENT

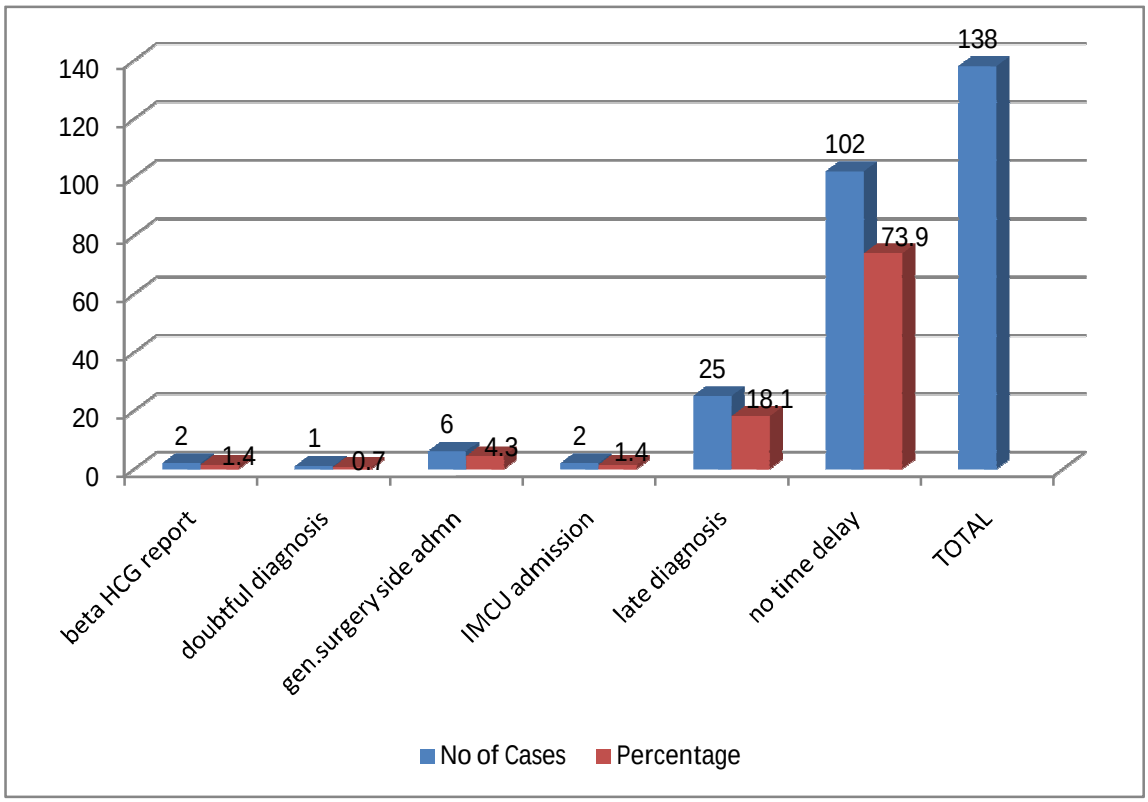


The definitive treatment by either laparotomy or medical management of ectopic pregnancy started within 2 hrs in 102 (73.9%) patients .

Time delay for more than 2 hrs was present in 36(26 %) patients .

Definitive treatment was delayed for more than 3 days in 5 (3.6%) patients .

REASONS FOR TIME DELAY

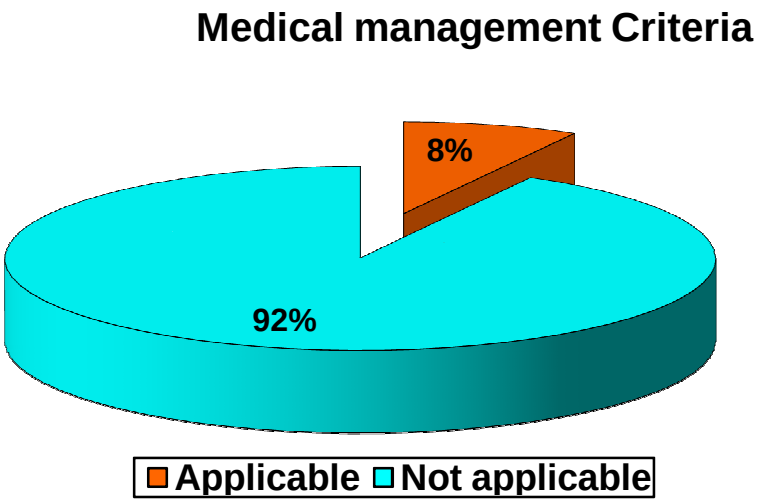


Most common cause of time delay was late diagnosis, which was present in 25 (18.1%) patients .

Other causes for time delay are waiting for beta hCG report , admission in other wards and doubtful diagnosis .

There was no time delay in 102 (73.9%) patients in whom definitive treatment started within two hours .

MEDICAL MANAGEMENT CRITERIA



Medical management criteria was applicable in only 11 (8%) patients .

Medical management criteria was not applicable in 127 (92%) patients .

MANAGEMENT

Management	No of patients	Percentage
Rt partial salphingectomy	21	15.2
Rt total salphingectomy	19	13.8
Lt partial salphingectomy	21	15.2
Lt total salphingectomy	10	7.2
RSO	10	7.2
LSO	10	7.2
RSO + LSO	1	0.7
RSO + Lt salphingectomy	13	9.4
LSO + Rt salphingectomy	3	2.2
B/L total salphingectomy	16	11.6
Rt cornual resection	2	1.4
Removal of Rt horn ectopic	1	0.7
Hysterectomy	1	0.7
Rt total salphingectomy + Rt ovarian cystectomy	1	0.7
Lt total salphingectomy + MVA	1	0.7
Lt ovariotomy + D&C	1	0.7
B/L ovarian cystectomy	1	0.7
Negative Laparotomy + D&C	1	0.7
Medical management , MTX - multidose	5	3.6
Total	138	

Medical management criteria was applicable in 11 patients . Medical management was done in 5 patients .

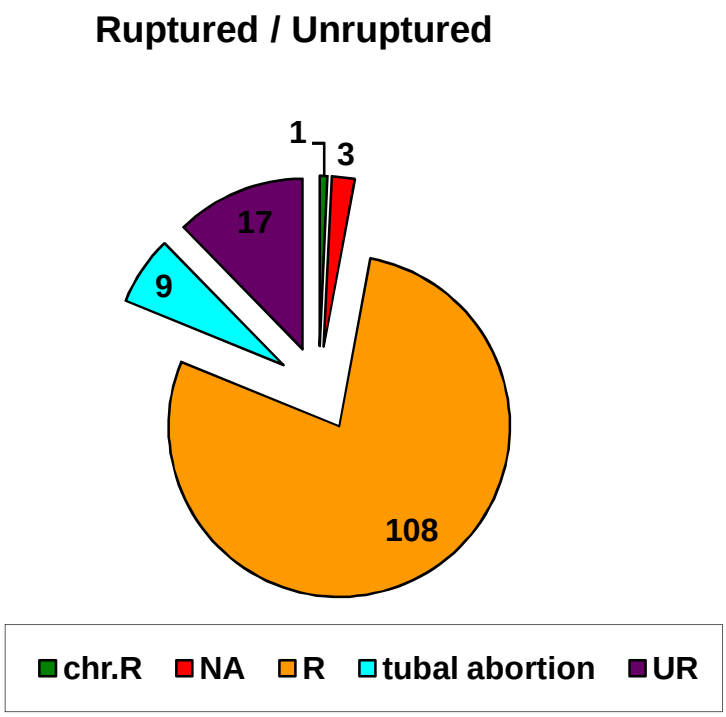
Unilateral partial salphingectomy was done in 42 patients .unilateral total salphingectomy was done in 31 patients . B/L total salphingectomy was done in 16 patients.

Unilateral salphingo oophorectomy was done in 36 patients. B/L salphingo oophorectomy was done in 1 patient.

One patient had hysterectomy .

One patient had negative laparotomy .

TYPES OF ECTOPIC PREGNANCY



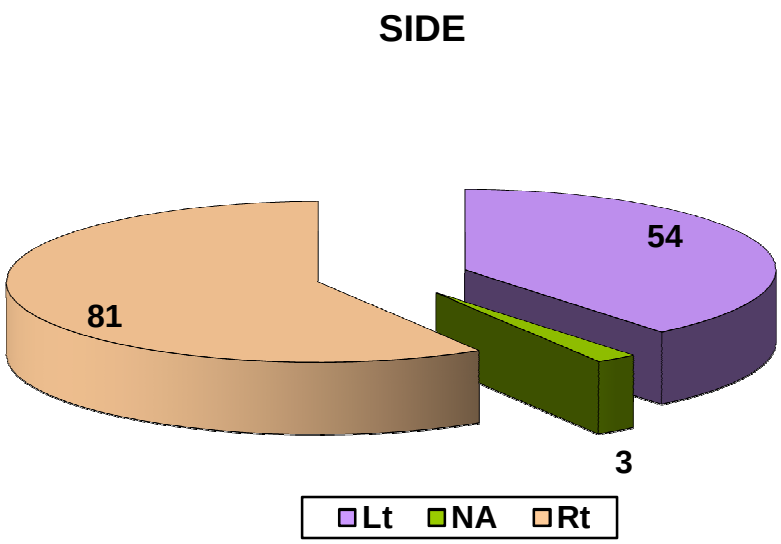
Per operatively 108 (78.3%) patients were found to be having ruptured ectopic. 1 (0.7%) patient had chronic ruptured ectopic .

17 (12.3%) patients had unruptured ectopic .

9 (6.5%) patients had tubal abortion .

In remaining 3 (2.2%) patients , 1 patient had negative laparotomy and 2 others were found to be having ovarian cyst per operatively .

SIDE OF THE ECTOPIC PREGNANCY

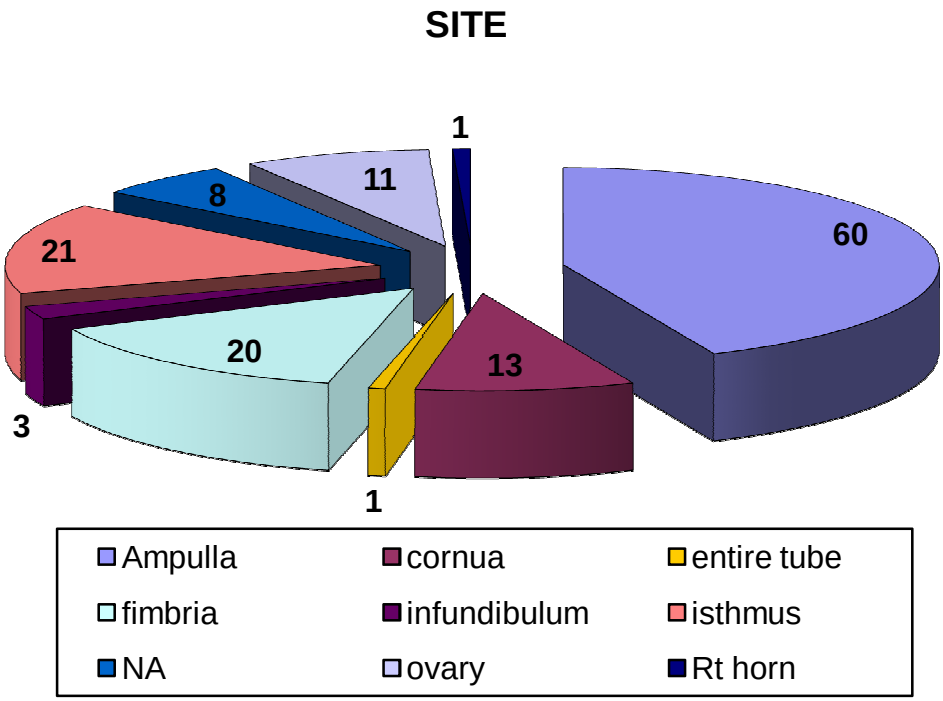


81 (58.7%) patients were found to be having right sided ectopic pregnancy .

54 (39.1%) patients were found to be having left sided ectopic pregnancy.

In remaining 3 (2.2%) patients , 1 patient had negative laparotomy and 2 others were found to be having ovarian cyst per operatively .

SITE OF THE ECTOPIC PREGNANCY



Site	No of patients	percentage
ampulla	60	43.5
cornua	13	9.4
fimbria	20	14.5
isthmus	21	15.2
infundibulum	3	2.2
Entire tube	1	0.7
ovary	11	8.0
Rt horn	1	0.7
Not applicable (NA)	8	5.8
total	138	

Most common site was ampulla followed by isthmus and fimbria .

Site could not be identified in medically managed patients.(5 patients).

In remaining 3 (2.2%) patients , 1 patient had negative laparotomy and 2 others were found to be having ovarian cyst per operatively . So site was not applicable in 8 patients (5+3).

AMOUNT OF HAEMOPERITONEUM (ml)

Haemoperitoneum in ml	**No of patients**	**percentage**
0-500	48	34.8
501-1000	31	22.5
1001-1500	23	16.7
1501-2000	13	9.4
2001-3000	4	2.9
Nil	14	10.1
NA	5	3.6
total	138	

Amount of clotted blood were weighed and converted into ml to simplify the data collection and analysis .

5 (3.6%) patients were managed medically. So per operative finding of haemoperitoneum was not applicable.

In remaining 133 (96.4%) patients , 40 (29%) patients had haemoperitoneum of more than one litre.

14 (10.1%) patients had no haemoperitoneum .

OTHER PER OPERATIVE FINDINGS

S.no	Other per operative findings	No of patients
1	Nil	62
2	e/o recanalisation	1
3	Active bleeding	2
4	Adhesions	26
5	Cystic ovaries	7
6	Haemorrhagic Rt ovary	1
7	Uterine anomalies	3 (2-bicornuate,1- arcuate)
8	Corpus luteal cyst	5(2-same side,3-opposite side)
9	Fimbrial cyst	2 (opposite side)
10	Endometriosis	1
11	B/L hydrosalphinx	1
12	Haematosalphinx	1
13	B/L dermoid cyst	1
14	Ovarian cyst	4 (1-complex, 1-twisted, 1-haemorrhagic, 1-simple)

BLOOD AND BLOOD PRODUCTS TRANSFUSION

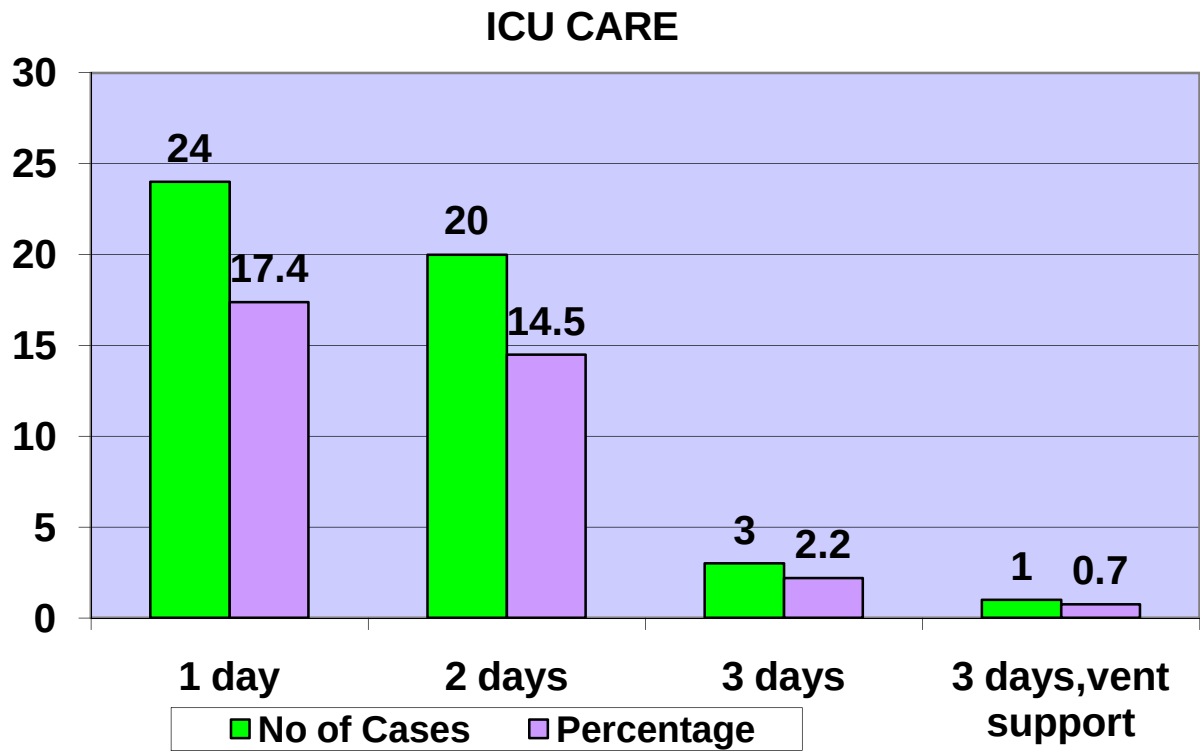
No of units of blood & blood products	No of patients
nil	36
1 WB	36
2 WB	32
3 WB	13
4 WB	6
1 WB,1 PCV	1
2 WB, 1 PCV	1
2 WB,2 PCV	1
3 WB, 4 FFP	1
3 WB,3 FFP	1
4 WB,2FFP	2
4 WB,4 FFP	2
4 WB, 4 FFP,4 PLTS	1
5 WB ,3 FFP	1
5 WB,4 FFP,2 PLTS	1
1 PCV	1
2 PCV	2
Total	138

36 (26.1%) patients required no blood transfusion.

65 (47.1%) patients required more than one unit of blood / blood products transfusions.

14 (10.1%) patients required more than 3 units of blood / blood products transfusions.

ICU CARE



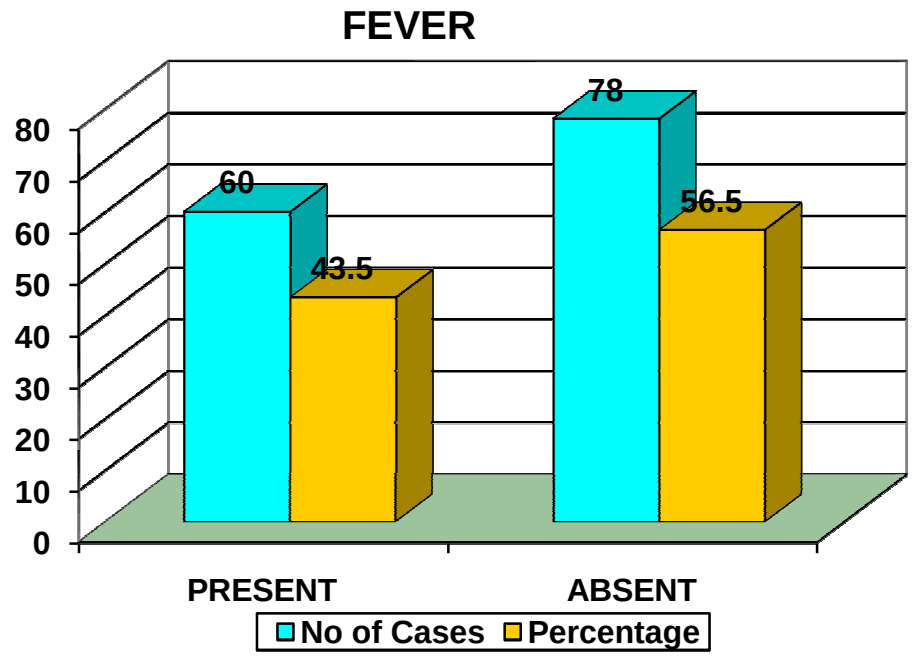
90 (65.2%) patients required no ICU care.

48 (34.8%) patients required ICU care. Most of them required ICU care for one or two days.

Only 4 (2.9%) patients required ICU care for more than 2 days .

One patient required ventilator support .

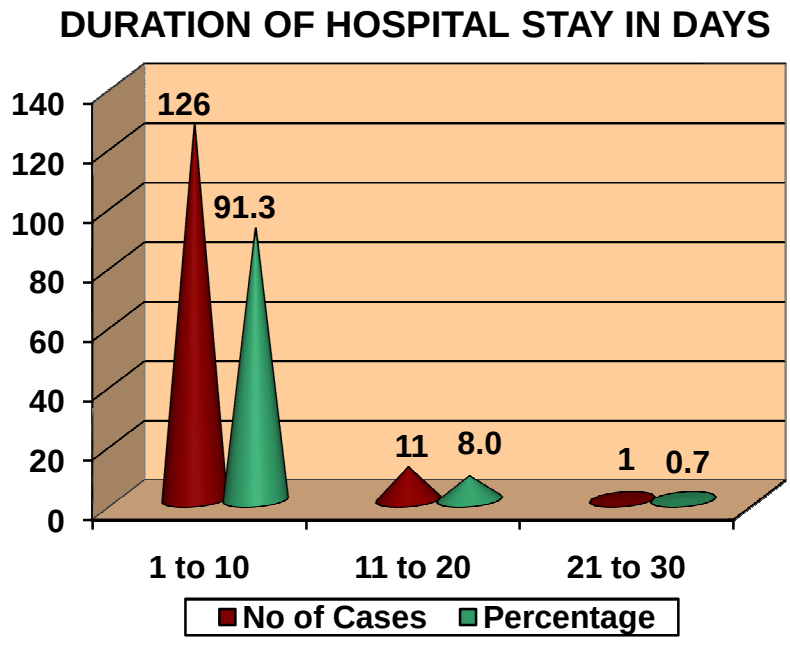
FEVER



Among the study population 60 (43.5%) patients had fever , especially post operatively .

78(56.5%) patients had no fever during the hospital stay .

DURATION OF HOSPITAL STAY

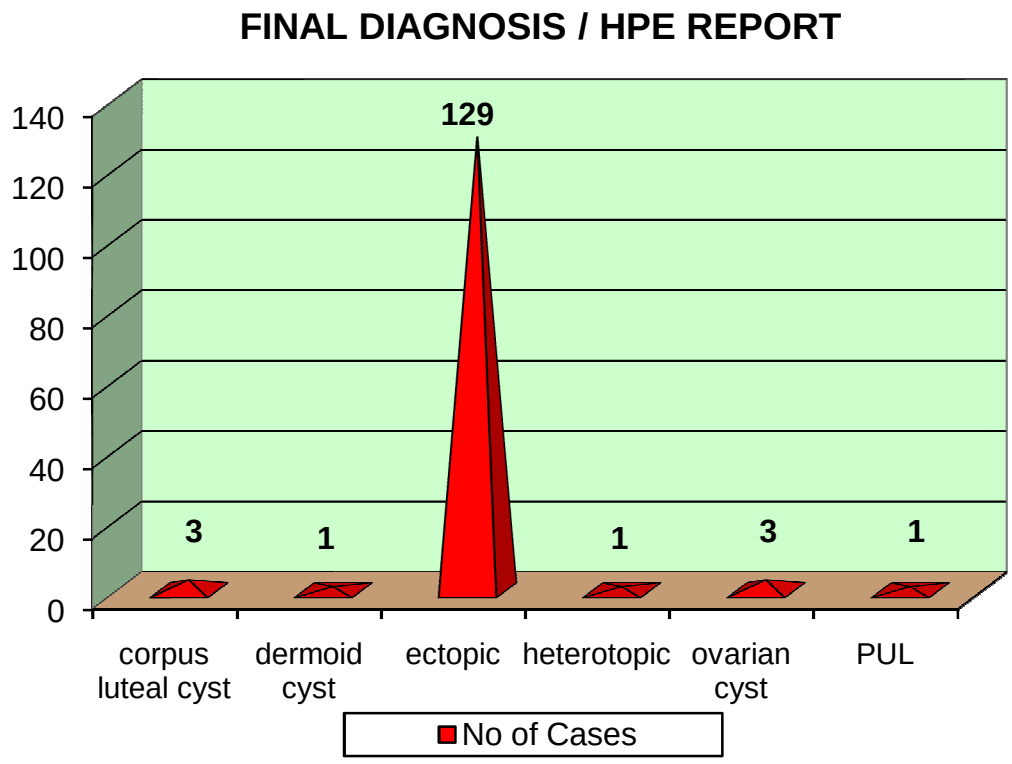


Most of the patients were discharged on 7th or 8th post operative day after suture removal.

126 (91.3%) patients were discharged within 10 days.

12 (8.7%) patients required prolonged hospital stay for more than 10 days.

FINAL DIAGNOSIS



Out of 138 patients , the final diagnosis of ectopic pregnancy was made in 129 patients ,which was confirmed by HPE reports in 124 patients. 5 patients underwent medical treatment . So,tissue was not available for HPE diagnosis. But all 5 patients were responded to medical management and treated successfully.

corpus luteal cyst was diagnosed in 3 patients only after HPE reports.

1 patient had dermoid cyst, which was confirmed by HPE.

Location of pregnancy was not identified in one patient.

Heterotopic pregnancy was confirmed by HPE in one patient .

1 patient was diagnosed to be having an ovarian cyst per operatively.

Final diagnosis of ovarian cyst was made in 2 patients only after HPE report .

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF SYMPTOMS FOR DIAGNOSING ECTOPIC PREGNANCY

Patient's Symptoms	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
amenorrhoea						
yes	95	5	73.6	44.4	95.0	10.5
no	34	4				
abdominal pain						
yes	124	7	96.1	22.2	94.7	28.6
no	5	2				
bleeding p/v						
yes	59	0	45.7	100	100	11.4
no	70	9				
ameno – pain						
yes	90	4	69.8	55.6	95.7	11.4
no	39	5				
ameno- bleed p/v						
yes	47	0	36.4	100	100	9.9
no	82	9				
pain-bleed p/v						
yes	56	0	43.4	100	100	11.0
no	73	9				
class-triad						
yes	44	0	34.1	100	100	9.6
no	85	9				

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF SYMPTOMS FOR DIAGNOSING ECTOPIC PREGNANCY

Patient's Symptoms	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
amenorrhoea						
yes	95	5	73.6	44.4	95.0	10.5
no	34	4				
abdominal pain						
yes	124	7	96.1	22.2	94.7	28.6
no	5	2				
bleeding p/v						
yes	59	0	45.7	100	100	11.4
no	70	9				
ameno – pain						
yes	90	4	69.8	55.6	95.7	11.4
no	39	5				
ameno- bleed p/v						
yes	47	0	36.4	100	100	9.9
no	82	9				
pain-bleed p/v						
yes	56	0	43.4	100	100	11.0
no	73	9				
class-triad						
yes	44	0	34.1	100	100	9.6
no	85	9				

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF EXAMINATON FINDINGS FOR DIAGNOSING ECTOPIC PREGNANCY

Examination findings	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
abd distension						
yes	55	2	42.6	77.8	96.5	8.6
no	74	7				
abd tenderness or guarding						
yes	74	4	57.4	55.6	94.9	8.3
no	55	5				
abd distension &tenderness or guarding						
yes	42	1	32.6	88.8	97.7	8.4
no	87	8				

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF P/V FINDINGS FOR DIAGNOSING ECTOPIC PREGNANCY

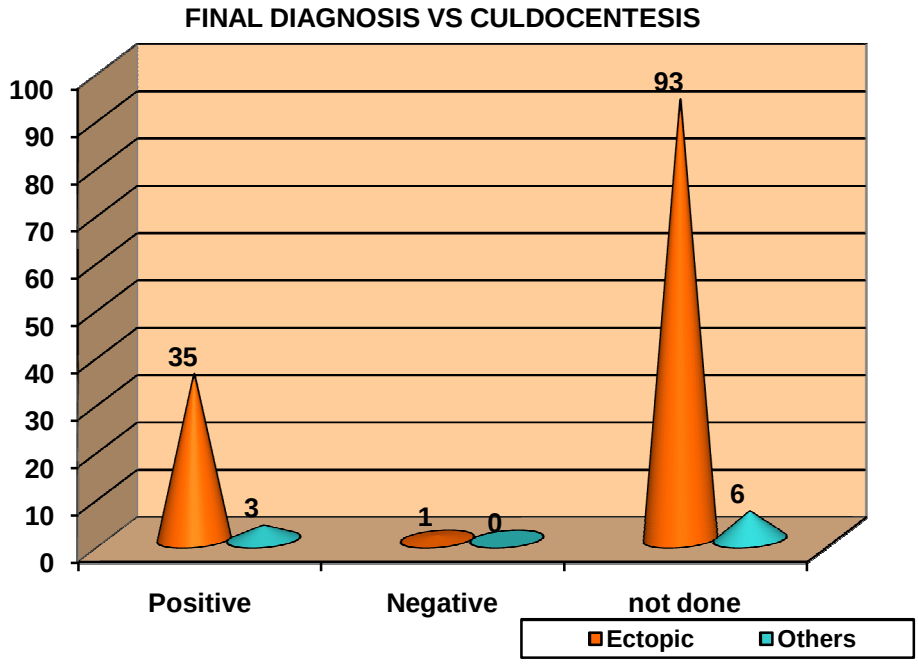
Per vaginal findings	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
forniceal fullness						
yes	36	3	27.9	66.7	92.3	6.1
no	93	6				
forniceal tenderness						
yes	75	4	58.1	55.5	94.9	8.5
no	54	5				
adnexal mass						
yes	3	0	2.3	100	100	6.7
no	126	9				
f.fullness & tenderness						
yes	19	0	14.7	100	100	7.6
no	110	9				
f.tenderness & adnex.mass						
yes	2	0	1.4	100	100	6.6
no	127	9				

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF ULTRASOUND FINDINGS FOR DIAGNOSING ECTOPIC PREGNANCY

Ultrasound findings	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
empty ut & adnex.mass						
yes	13	2	10.7	77.8	86.7	5.7
no	116	7				
empty ut & free fluid						
yes	33	1	25.6	88.9	97.1	7.6
no	96	8				
empty ut , adnex.mass & free fluid						
yes	64	4	52.7	55.6	94.1	7.1
no	65	5				

SENSITIVITY , SPECIFICITY,POSITIVE AND NEGATIVE PREDICTIVE VALUES OF UPT AND CEVICAL EXCITATION TEST FOR DIAGNOSING ECTOPIC PREGNANCY

Examination findings	ectopic pregnancy		sensitivity	specificity	PPV	NPV
	yes	no				
UPT						
positive	127	5	98.4	44.4	96.2	66.7
negative	2	4				
cervical excitation test						
positive	96	4	74.4	55.6	96.0	13.2
negative	33	5				



GESTATIONAL AGE AT RUPTURE FOR SPECIFIC SITES

	≤ 35 days n (%)	**36-56 days n (%)**	**> 57 days n (%)**	**Not known n (%)**	**Total n (%)**
Ampulla	5 (4.5)	32 (29.4)	6 (5.5)	5 (4.5)	48 (43.9)
Fimbria	2 (1.8)	6 (5.5)	6 (5.5)	3 (2.8)	17 (15.6)
Isthmus	1 (0.9)	11 (10.1)	4 (3.7)	4 (3.7)	20 (18.4)
Infundibulum	0 (0)	1 (0.9)	1 (0.9)	0 (0)	2 (1.9)
Ovary	2 (4.8)	5 (4.5)	1 (0.9)	1 (0.9)	9 (8.3)
Cornua	1 (0.9)	3 (2.8)	5 (4.5)	3 (2.8)	12 (11.0)
Rudimetary horn	0 (0)	0 (0)	1 (0.9)	0 (0)	1 (0.9)
Total	11 (10.1)	58 (53.2)	24 (22)	16 (14.7)	109 (100)

Total no of ruptured ectopic was 109 including 1 chronic ruptured ectopic .

Most of the ectopic pregnancies were found to be ruptured between 6 to 8 weeks.

≥ 1.5 L OF HAEMOPERITONEUM FOR SPECIFIC SITES OF ECTOPIC PREGNANCY

	No of cases	Percentage
Ampulla	7	29.15
Isthmus	7	29.15
Fimbria	1	4.20
Cornua	9	37.50
Total	24	100.00

Ruptured Cornual ectopic pregnancies were most commonly associated with massive haemoperitoneum followed by isthmic and ampullary pregnancies .

MORBIDITIES ASSOCIATED WITH RUPTURED AND UNRUPTURED ECTOPIC PREGNANCIES

	Ruptured	Unruptured	Tubal abortion
Total no of cases	109	17	9
Fever	56	1	3
Anaemia	43	1	0

Ruptured ectopic pregnancies were associated with higher morbidities.

DISCUSSION

In the present study, the incidence of ectopic pregnancy was 8.7 per 1000 deliveries. In a study conducted by shraddha shetty k et. al in mangalore , the incidence was 5.6/1000 deliveries[6] . In a study conducted by Rashmi et.al the incidence was 1: 399 pregnancies [7] . In a study conducted by porwal sanjay et al , the incidence was found to be 2.46 per 1000 deliveries[8] .

Majority of the patients (63.8%) were in the age group of 21 to 30 years in our study. Similar results were found in Smita singh et.al and Samiya Mufti et.al studies[70,65] . This corresponds to the age of peak sexual activity and reproduction . There are studies stating that age related tubal changes increase the incidence of ectopic pregnancy [63,64] .

In our study multigravida (75.4%) were found to be more prone to have an ectopic pregnancy. This result was similar to other studies conducted by Shraddha Setty et.al and Laxmi karki et al[6,257] . In Laxmi karki study 61% were multiparous women.

Majority (54.3%) of the patients presented with 5 to 8 weeks of amenorrhoea representing the time period required for the growing ectopic gestation to distend the tube and cause symptoms . This results are consistent with those from Tang BD et.al and Panti A et.al[10,13] .

Among the nulliparous women 62.5% had history of subfertility for more than one year .Similar findings were obtained in Tanveer Shafquat et.al study[99].

The commonest risk factors among the study population were previous LSCS, tubal surgeries , previous ectopic pregnancies , prior H/O abortions , H/O infertility and pelvic inflammatory disease. Similar risk factors were noted in various other studies[6,8] . The increasing trend in caesarean section was found to be associated with increased risk of ectopic pregnancy . 6 patients had H/O MTP pills intake. The significance of this risk factor needs further studies . 7 patients gave history of ovulation induction . Advancement in infertility treatment was associated with significant risk of ectopic pregnancy.

In our study 25 (18.1%) cases were post sterilisation ectopic pregnancies. Among the sterilisation methods, concurrent and puerperal sterilisation were found to be associated with higher number of ectopic pregnancies. Ectopic pregnancies most commonly occurred 3 to 10 years after the sterilisation surgery.

The most common presenting symptom was abdominal pain which was found in 94.9% of patients , followed by amenorrhoea in 72.5% of the patients. The classic triad was present only in 31.9% of cases . In porwal sanjay et al study , 90% reported amenorrhoea and 87.5% reported abdominal pain [8]. In some cases gastrointestinal and vaso vagal

symptoms are the presenting complaints. In our study the classic triad has 100 % specificity and positive predictive value , but low sensitivity .

The most common examination findings were tachycardia (51.4%) and hypotension (12.3%) . 6.5% of the patients presented with shock. Tachycardia was not present in all cases of ruptured ectopic pregnancy. 48.6% of patients were severely anaemic with Hb less than 5 gms%.

In per abdominal examination abdominal tenderness (56.5%) was the most common finding. In per vaginal examination forniceal tenderness (57.2%) was the most common finding. Cervical excitation test was positive in 72.5%. Culdocentesis was done only in 39 patients. Among them 38 were positive. In p/v the presence of adnexal mass has highest specificity and positive predictive value (100%) .

Urine pregnancy test and ultrasound examination were done in all patients. Serum beta hCG was sent only in haemodynamically stable patients with unruptured ectopic pregnancy to decide for medical management and in doubtful cases. UPT has highest (98.4%) sensitivity, but the specificity was low. In ultrasound examination empty uterus,adnexal mass and free fluid were strongly suggestive of ectopic pregnancy, especially in the presence of positive UPT.

Medical management criteria was applicable in 11 patients. Among them only 5 patients had medical management. Remaining were multiparous women who wanted definitive surgery rather than medical

management. Multi dose methotrexate regimen was used in all 5 patients and were treated successfully.

Treatment delay for more than 2 hrs was present in 36 (26%) patients. The most common reason for time delay was late diagnosis of ectopic pregnancy .In these cases diagnosis was misguided by atypical presentation, wrong referral diagnosis and atypical ultrasound findings. 8 patients had admitted in wrong wards and then transferred to O &G ward . Time delay was found to be associated with more morbidity.

In surgically managed group all 132 patients underwent laparotomy. Total/partial salphingectomy and salphingo oophorectomy were the common surgeries performed. In developing countries open method by laparotomy still remains the most commonly used management for ectopic pregnancy. But the trend is changing towards the laparoscopic surgery and conservative management. In Samiya Mufti et.al study the surgical management was by open method in all cases [65].

During laparotomy 109 (79%) cases were found to be ruptured including one chronic rupture. In developing countries still majority of cases present late with rupture. This was evidenced by many studies[6,7,65].

Ampulla was the most common site of the ectopic pregnancy found in 43.5% of the patients . This result was similar to other studies [7,70] . During laparotomy 124 patients were found to be having ectopic pregnancy. One patient was found to be having a rare variety of

pregnancy known as heterotopic pregnancy. One patient had negative laparotomy. One patient was found to be having dermoid cyst. One patient had ovarian cyst. Remaining 5 patients were diagnosed as ectopic pregnancy per operatively, then diagnosis was changed to gynaec lesions (3 corpus luteal cyst and 2 simple ovarian cyst) after HPE report.

Additional findings during laparotomy were corpus luteal cyst in 5 patients, fimbrial cyst in 2 patients , uterine anomalies in 3 patients, ovarian pathology in 13 patients, endometriosis , haematosalphinx and hydrosalphinx each in 1 patient. Adhesions were found in 26 patients.

Only one patient had hysterectomy for uncontrolled bleeding from the ruptured site of cornual ectopic pregnancy . Only one patient required post operative ventilatory support. No maternal mortality was found during the study period. This result was consistent with Rashmi et.al study[7] .

40 (29%) patients had haemoperitoneum of more than one litre. 65(47.1%) patients required more than one unit of blood transfusion .Massive haemoperitoneum was most commonly found in cornual and isthmic ruptured ectopic pregnancies.

Post operative anaemia and fever were found to be more common in ruptured ectopic pregnancies when compared to unruptured ectopic pregnancies.

12 (8.7%) patients required prolonged hospital stay for more than 10 days. The factors associated with prolonged hospital stay were medical management , anaemia correction , post operative fever and late diagnosis.

CONCLUSION AND RECOMMENDATIONS

1. Ectopic pregnancy is famous for its diversity of clinical presentations and atypical presentations. Strong suspicion is required for its early diagnosis.
2. Amenorrhoea is not necessary for the diagnosis of ectopic pregnancy. UPT and ultrasound should be advised to all reproductive age group patients presenting with abdominal pain , bleeding p/v with or without amenorrhoea.
3. Sterilisation could not rule out the possibility of an ectopic pregnancy. So, we should advise the patients to come for check up if she misses the periods as early as possible.
4. The recommended sterilisation techniques should be followed strictly.
5. To join the global trend of conservative management for ectopic pregnancy, early diagnosis before rupture is important.
6. Ultrasound is the simple and gold standard diagnostic method for ectopic pregnancy in low resource settings.
7. Routine first trimester ultrasound should be done in all pregnant women at the booking visit itself .
8. UPT kits and sonographic equipments should be made available in all primary health centres and emergency gynaecological units.
9. Staffs should be trained adequately to use and interpret the sonographic images.

10. Early diagnosis and referral is the key factor in reducing the maternal morbidity and in preserving the future fertility.
11. Because of the high incidence of tubal rupture in our set up, community education is required to inform the women to attend the health facilities as early as possible once they have symptoms .

DATA SHEET

PATIENT PROFILE

Name : Age : I.P.no :

Address :

Socio economic status :

Date and time of admission :

Mode of admission : Referral /Self

In case of referral the referral diagnosis :

Mode of transport :

Obstetric code :

LMP :

Period of amenorrhoea :

UPT : Positve/ Weakly positive/ Negative

Married since :

Duration of subfertility :

Past obstetric history :

- Miscarriage
- Normal vaginal delivery
- Caesarean section
- Previous ectopic gestation
- Gestational trophoblastic disease

Sterilisation status :

Sterilised / not sterilised

If sterilised , by what method

1) Concurrent sterilisation
2) Puerperal sterilisation
3) Laparoscopic sterilisation
4) TAT by open method

TIME SINCE STERILISATION :

OTHER CONTRACEPTIVE USE :

1) Barrier method
2) Intra uterine devices
3) Oral contraceptive pills

    Combined pills / POP / Emergency contraceptive pills

4) Others

RISK FACTORS :

Previous LSCS

H/O abortion

H/O infertility

Ovulation induction

Artificial Reproductive techniques.

Previous ectopic pregnancy

Pelvic inflammatory diseases

Sterilisation

Tubal recanalisation surgery

Other pelvic surgeries

Intrauterine contraceptive device use

Combined oral contraceptive pills

Progesterone only pills

Emergency contraceptive pills

MTP pills

Smoking

Others

PRESENTING SYMPTOMS :

Abdominal pain

Amenorrhoea

Bleeding p/v

Vasovagal symptoms

Gastro intestinal symptoms

Shoulder tip pain

Others

EXAMINATION :

Puse rate

Pulse volume

Blood pressure

Temperature

Consciousness

Pallor

Tachycardia

PER ABDOMINAL FINDINGS

Abdominal distension

Abdominal tenderness

Guarding

PER VAGINAL EXAMINATION

Forniceal tenderness

Forniceal fullness

Adnexal mass

Others

CERVICAL EXCITATION TEST : positive / negative

CULDOCENTESIS : positive / negative / not done

INVESTIGATIONS

Haemoglobin

Renal function test

Liver function test

Blood grouping & typing

Serum beta hCG

ULTRASOUND : Transabdominal / Transvaginal / Both

ULTRASOUND FINDINGS :

Empty uterus

Free fluid

Adnexal mass with size

Extra uterine gestational sac with size

Embryonic cardiac activity

Other findings

OTHER IMAGINGS : Doppler scan /CT/ MRI / not done

CT/ MRI /Doppler findings , if taken :

CLINICAL DIAGNOSIS :

TIME INTERVAL BETWEEN ADMISSION AND ONSET OF DEFINITIVE TREATMENT :

TIME DELAY : yes / no

REASONS FOR TIME DELAY

1) Late diagnosis

2) Waiting for beta hCG / CT/MRI reports

3) Admissions in other wards

4) Doubtful diagnosis

5) others

MANAGEMENT : Medical / Surgical

MEDICAL MANAGEMENT : Single dose MTX / Multiple dose MTX

SURGICAL MANAGEMENT : Laparotomy / Laparoscopic

Date and time of surgery :

Type of surgery :

1) Partial salphingectomy – unilateral / bilateral
2) Total salphingectomy – unilateral / bilateral
3) Salphingo oophorectomy
4) Salphingostomy
5) Hysterectomy
6) Cornual wedge resection
7) Others

Per operative findings :

1) Rupured / unruptured / chronic rupture / tubal abortion
2) Side
3) Site
4) Amount of haemoperitoneum
5) Other findings
    - Adhesions
    - Corpus luteal cyst – same side / opposite side
    - Ovarian cyst
    - Uterine anomalies
    - Endometriosis
    - Tubal pathology
    - Other findings

Duration of surgery :

Complications during surgery :

NUMBER OF UNITS OF BLOOD AND BLOOD PRODUCTS TRANSFUSED :

ICU CARE (In days) :

VENTILATORY SUPPORT REQUIRED / NOT

FEVER : Present / Absent

DURATION OF HOSPITAL STAY (IN DAYS):

HPE REPORT :

FINAL DIAGNOSIS :

BIBLIOGRAPHY

1. Walker JJ. Ectopic pregnancy. ClinObstet Gynecol. 2007; 50: 89–99.
2. Rajkhowa, M., Trends in the incidence of ectopic pregnancy in England and Wales from 1966 to 1996. BJOG, 200. **107**((3)): p. 369-74.
3. Geoffrey C. Ectopic Pregnancy. In: Obstetrics by Ten Teachers. Chamberlain G (ed), 16th edition. ChurchillLivingstone Pub. London. 1997: 162-165.
4. Kwawukume EY, Idrissa A. Ectopic Pregnancy. In: Comprehensive Obstetrics in the Tropics. Kwawukume EY and Emuveyan EE (Eds). Asante Hittscher Printing, Accra; 2000: 211-218.
5. Osaheni LLawani , Okechukwu B Anozie, Paul O Ezeonu . Ectopic pregnancy: a life-threatening gynecological emergency. International Journal of Women's Health 2013:5 515–521
6. Shraddha shetty k et.al ; a clinical study of ectopic pregnancies in a tertiary care hospital of Mangalore. Innovative Journal of Medical and Health Science 4 : 1 Jan - Feb(2014) 305-309.
7. Rashmi A Gaddagi, AP Chandrashekhar.A Clinical Study of Ectopic Pregnancy.JCDR 2012;6:867-869
8. Gupta R, Porwal S, Swarnkar M, Sharma N, Maheshwari P. Incidence, trends and risk factors for Ectopic Pregnancies in a tertiary care hospital of Rajasthan. JPBMS 2012; 16 (07):1-3
9. Rotas, M.A., S. Haberman, and M. Levgur, Cesarean scar ectopic pregnancies: etiology, diagnosis, and management. Obstet Gynecol, 2006. **107**((6)): p. 1373-81.
10. Tang, A., D. Baartz, and S.K. Khoo, A medical management of interstitial ectopic pregnancy: a 5-year clinical study. Aust N Z J Obstet Gynaecol,, 2006. **46**((2)): p. 107-11.
11. Barnhat K T, G.C., Reinll B, usefulness of pipelle endometrial biopsy in the diagnosis of women at risk of ectopic pregnancy. AMJ obstet.Gynecol, 2003. **188**: p. 906-9.
12. Aboulfoutouh, I. I.; Youssef, M. A.; Zakaria, A. E.; Mady, A. A. & Khattab, S. M. (2011).Cervical twin ectopic pregnancy after *In Vitro* fertilization-Embryo

transfer (IVFET): case report. *Gynecol Endocrinol*, (April 2011), [Epub ahead of print]

13. Panti A, Ikechukwu NE, lukman OO, Yakubu A, Egondu SC, Tanko BA. Ectopic pregnancy at Usmanu Danfodiyo University Teaching Hospital Sokoto: a ten year review. *Ann Niger Med*. 2012;6(2):87–91.
14. Abdul FI. Ectopic pregnancy in Ilorin: a review of 278 cases. *Niger J Med*. 2000;9(3):92–96.
15. Arpita N. A restospective and prospective study of maternal mortality in a rural tertiary care hospital of Central India. Indian Journal of Community Health, 2013. **25**(1).
16. Jurkovic D, Wilkinson H:Diagnosis and management of ectopic pregnancy. *BMJ* 2011;342:d3397doi: 10.1136/bmj.d3397
17. Murray, H., Diagnosis and treatment of ectopic pregnancy. CMAJ, 2005. **173**((8)): p. 905-12.
18. Amoko, D.H. and G. A. Buga., Clinical presentation of ectopic pregnancy in Transkei, South Africa. East Afr Med J,, 1995. **72**((12)): p. 770-3.
19. Goyaux N, Leke R, Keita N, Thonneau P. Ectopic pregnancy in African developing countries. ActaObstetGynecolScand 2003; 82:305-12
20. Karaer A, Avsar FA, Batioglu S. Risk factors for ectopic pregnancy: a case-control study. Aust N Z J ObstetGynaecol. 2006; 46:521–527
21. Turner C, Horner P, et al. British Fertility Society Impact of *Chlamydia trachomatis* in the reproductive setting: British Fertility Society Guidelines for practice. Hum Fertil (Camb) 2010; 13:115–125.
22. Monga A. Ectopic pregnancy. In: Monga A, Baker P, editors. *Gynaecology by Ten Teachers*. 18th ed. London: Hodder Education; 2006:97–99.
23. Ectopic Pregnancy Clinical Presentation Author: Vicken P Sepilian, MD, MSc; Chief Editor: Michel E Rivlin, MD more...Updated: May 2, 2014
24. Varma R, Gupta J, Tubal ectopic pregnancy.Clin Evid (online) 2009;2009: pii;1406
25. Timmerman D. Predictive models for the early diagnosis of ectopic pregnancy.Verh K AcadGeneeskd Belg. 2004; 66 (2):155-71.

26. Barnhart KT. Clinical practice. Ectopic pregnancy. N Engl J Med. 2009; 361:379–387.
27. John A. Rock, Howard W. Jones III. Te Linde's Operative Gynaecology. 10 th edition. Lippincott , Williams & Wilkins, a Wolters Kluwer business. USA. 2008. P[798].
28. F, Gary Cunningham,Kenneth J . Leveno, Steven L. Bloom, John C. Hauth, Dwight J. Rouse, Catherine Y. Spong. Williams Obstetrics. 23rd edition. McGraw- Hill Companies. USA .2010.p[238.]
29. Shaw, S.K. Dey, H.O.D. Critchley, Current knowledge of the aetiology of human tubal ectopic pregnancy. J.L.V. Available from: www.ncbi.nlm.nih.gov/pmc.
30. Tulandi T. Surgical treatment of ectopic pregnancy and prognosis for subsequent fertility. Up to date 12.3,2004:1-17.
31. Awojobi OA and Ogunsina S. Ectopic pregnancy in a rural practice. Nigerian Journal of Medicine, 2001;10(3):139-140.
32. Marion LL and Meeks GR. Ectopic pregnancy: history, incidence, epidemiology, and risk factors. Clin Obstet Gynecol 2012; 55: 376-386.
33. Farquhar CM. Ectopic pregnancy. Lancet 2005; 366: 583-591.
34. Tay JI, Moore J and Walker JJ. Ectopic pregnancy. BMJ 2000; 320: 916-919.
35. Shaw JL, Dey SK, Critchley HO and Horne AW. Current knowledge of the aetiology of human tubal ectopic pregnancy. Hum Reprod Update 2010; 16: 432-444.
36. Shao R. Understanding the mechanisms of human tubal ectopic pregnancies: new evidence from knockout mouse models. Hum Reprod 2010; 25: 584-587.
37. Shao R, Wang X, Wang W, Stener-Victorin E, Mallard C, Brannstrom M and Billig H. From mice to women and back again: Causalities and clues for Chlamydia-induced tubal ectopic pregnancy. Fertil Steril 2012; 98: 1175-1185
38. Shao R, Zhang SX, Weijdegard B, Zou S, Egecioglu E, Norstrom A, Brannstrom M and Billig H. Nitric oxide synthases and tubal ectopic pregnancies induced by Chlamydia infection: basic and clinical insights. Mol Hum Reprod 2010; 16: 907-915.

39. Shao R, Zou S, Wang X, Feng Y, Brannstrom M, Stener-Victorin E and Billig H. Revealing the Hidden Mechanisms of Smoke-Induced Fallopian Tubal Implantation. Biol Reprod 2012; 86: 131.
40. Hunter RH. Components of oviduct physiology in eutherian mammals. Biol Rev Camb Philos Soc 2012; 87: 244-255.
41. Seshagiri PB, Sen Roy S, Sireesha G, Rao RP. Cellular and molecular regulation of mammalian blastocyst hatching. J Reprod Immunol 2009; 83: 79-84.
42. RCOG 2004 BJOG: an International Journal of Obstetrics and Gynaecology 111, pp. 1322–1332
43. Sharma D, Singhal SR, Singhal SK. Uterus didelphys, a rare cause for tubal sterilization failure. Aust N Z J Obstet Gynaecol 1998;38(3): 327– 328.
44. Makar AP, Vanderheyden JS, Schatteman EA, Albertyn GP,Verkinderen JJ, Van Marck EA. Female sterilization failure after bipolar electrocoagulation: a 6 year retrospective study. Eur J Obstet Gynecol Reprod Biol 1990;37(3):237–246
45. Rock JA, Parmley TH, King TM, Laufe LE, Su BS. Endometriosis and the development of tuboperitoneal fistulas after tubal ligation.Fertil Steril 1981;35(1):16–20
46. Badawy S, Gilman T, Mroziewicz E. The role of recanalization in tubal pregnancy after sterilization. Int Surg 1979;64(5):49–51.
47. Grunert GM. Late tubal patency following tubal ligation. Fertil Steril1981;35(4):406– 408.
48. Hernandez FJ. Tubal ligation and pregnancy: mechanism of recanalization after tubal ligation. Fertil Steril 1975;26(5):392– 396.
49. McCausland AM. Recanalization and fistulization of the fallopian tubes are thought to be the causes of pregnancies following female sterilization. Am J Obstet Gynecol 1981;139(1):114–115.
50. Horne AW, King AE, Shaw E, McDonald SE, Williams AR, Saunders PT, Critchley HO. Attenuated sex steroid receptor expression in fallopian tube of women with ectopic pregnancy. JClin Endocrinol Metab 2009; 94: 5146-5154

51. Lim HJ, Wang H. Uterine disorders and pregnancy complications: insights from mouse models. J Clin Invest 2010; 120: 1004-1015
52. Ruijin Shao1,4, Yi Feng1,2, Shien Zou3, Birgitta Weijdegård1, Gencheng Wu2, Mats Brännström4, Håkan Billig1.The role of estrogen in the pathophysiology of tubal ectopic pregnancy. Am J Transl Res 2012;4(3):269-278 www.ajtr.org /ISSN:1943-8141/AJTR1206002
53. Wang H, et al. Aberrant cannabinoid signalling impairs oviductal transport of embryos. *Nat Med*.2004;10(10):1074–1080.
54. Hunter RH. Tubal ectopic pregnancy: a pathophysiological explanation involving endometriosis.*Hum Reprod*. 2002;17(7):1688–1691
55. Dixon RE, et al. Chlamydia infection causes loss of pacemaker cells and inhibits oocyte transport in the mouse oviduct. *Biol Reprod*. 2009;80(4):665–673.
56. Wang H, Dey SK, Maccarrone M. Jekyll and hyde:two faces of cannabinoid signaling in male and female fertility. *Endocr Rev*. 2006;27(5):427–448.
57. Wang H, et al. Fatty acid amide hydrolase deficiency limits early pregnancy events. *J Clin Invest*. 2006;116(8):2122–2131.
58. Chang TC and Mendell JT. microRNAs in vertebrate physiology and human disease. Annu Rev Genomics Hum Genet 2007; 8: 215-239.
59. Lee EJ, Baek M, Gusev Y, Brackett DJ, Nuovo GJ and Schmittgen TD. Systematic evaluation of microRNA processing patterns in tissues, cell lines, and tumors. RNA 2008; 14: 35-42.
60. Liang Y, Ridzon D, Wong L and Chen C. Characterization of microRNA expression profiles in normal human tissues. BMC Genomics 2007; 8: 166.
61. Wang L, Feng Y, Zou S, Brannstrom M, He L, Billig H and Shao R. Linking DNA methylation to the onset of human tubal ectopic pregnancy. Am J Transl Res 2013; 5: 116-125.
62. Sepilian VP, Wood E. Ectopic pregnancy .eMedicine Specialities-Obstetrics & Gynaecology Updated on May 7, 2010; pp. 1-5. Online (Accessed 2010 June 6).

63. Menon S, Sammel MD, Vichnin M, Barnhart KT. Risk factors for ectopic pregnancy: a comparison between adult and adolescent women. J Paediatr Adolesc Gynecol 2007; 20: 181-5.
64. Sindos M, TogiaA, Sergentanis TN, Kabagiannis A, Malamas F, Farfaras A, et al. Ruptured ectopic pregnancy: risk factors for a life-threatening condition. Arch Gynecol Obstet 2009; 279: 621-3.
65. Samiya Mufti. Shagufta Rather, Samina Mufti, Reyaz A Rangrez, Wasiqa, Khalida ECTOPIC PREGNANCY: AN ANALYSIS OF 114 CASES JK-Practitioner 2012;17(4):20-23
66. Majhi AK, Roy N, Karmakar KS, Banerjee PK. Ectopic pregnancy - an analysis of 180 cases. J Indian Med Assoc 2007; 105: 308-12.
67. Westrom L, Bengtsson LPH, Mardh PA. Incidence trends and risk of ectopic pregnancy in a population of women. Br Med J 1981; 282; 15-18.
68. Rubin GL, Peterson HB, Dorfman SF, et al. Ectopic Pregnancy in USA 1970 through 1978. JAMA 1983;l 249 :1725
69. Anorlu RI, Oluwole A, Abudu OO, Adebajo S. Risk factors for ectopic pregnancy in Lagos, Nigeria. Acta Obstet Gynecol Scand 2005; 84:184-8.
70. Smita Singh1Mahendra G2, Vijayalakshmi S3, Ravindra S Pukale4. Clinical study of ectopic pregnancy in a rural set up: A two year survey. print ISSN: 22249 4995 | eISSN: 2277 8810.Volume 4|Issue 1|Jan – Mar 2014.
71. Yuk JS[1], Kim YJ, Hur JY, Shin JH. Association between socioeconomic status and ectopic pregnancy rate in the Republic of Korea.Int J Gynaecol Obstet. 2013 Aug;122(2):104-7. doi: 10.1016/j.ijgo.2013.03.015. Epub 2013 May 30.
72. Napolitano PG. Vu K, Rosa C. Pregnancy after failed tubal sterilization. J. Reprod. Med.,1996;41:609-13.
73. Pisarska MD, Carson SA. Incidence and risk factors for ectopic pregnancy. Clin. Obstet. Gynecol., 1999;42:2-8.
74. Peterson HB, Xia Z, Hughes 3M, et al. The risk of ectopic pregnancy after tubal sterilization. US Colloborative Review of Sterilization Working Group. N. EngI. 3. Med., 1997;336:762-67.

75. Hulka JF. Reich H. Textbook of laparoscopy 2nd ed. Philadelphia; WB Saunders, 1994:136 -43.
76. Soderstrom RM , Levy BS, Engel T. Reducing bipolar sterilisation failures. Obstet Gynecol 1989;74: 60-3.
77. Muhiu G, Rogo KO. Ruptured tubal pregnancy following tubal sterilization. East Afr Med J 1987;64:333-6.
78. Shah JP, Parulekar SV, Hinduja IN. Ectopic pregnancy after tubal sterilization. J Postgrad Med 1991;37:17-20.
79. Sultana A, Khan U. Ectopic pregnancy following reversal of tubal ligation. Ann Abbasi Shaheed Hosp 2001; 6:323-8.
80. Ameh N, Madugu NH, Bawa US, Adelaiye MS, Akpa M. Tubal ectopic pregnancy after bilateral tubal ligation: a case report. Niger J Med 2006;15:453-4.
81. Sinéad M. O'Neill,Esben Agerbo,Louise C. Kenny mail,Tine B. Henriksen,Patricia M. Kearney,Richard A. Greene,Preben Bo Mortensen,Ali S. Khashan.Cesarean Section and Rate of Subsequent Stillbirth, Miscarriage, and Ectopic Pregnancy: A Danish Register-Based Cohort Study. Published: July 01, 2014. DOI: 10.1371/journal.pmed.1001670
82. O'Neill SM, Khashan AS, Kenny LC, Greene RA, Henriksen TB, et al. (2013) Caesarean section and subsequent ectopic pregnancy: a systematic review and meta-analysis. BJOG 120: 671–680 doi:10.1111/1471-0528.12165
83. Michalas S[1], Minaretzis D, Tsionou C, Maos G, Kioses E, Aravantinos D.Pelvic surgery, reproductive factors and risk of ectopic pregnancy: a case controlled study. Int J Gynaecol Obstet. 1992 Jun;38(2):101-5.
84. Edelman DA. Contraceptive practice and ectopic pregnancy. IPPF Medical Bulletin 1980;14:1-2. [PUBMED]
85. Brar MK, Kaur JS. A study of microsurgical renastomosis of the fallopian tubes for reversal of sterilization. J Obst Gyn India 2000;6:75-7
86. Royal College of Obstetricians and Gynaecologists.*Management of Acute Pelvic Inflammatory Disease*. Greentop Guideline No.32. London,UK: RCOG, 2008

87. Bevan C, Johal B, Mumtaz G, Ridgway G, Siddle N. Clinical, laparoscopic and microbiological findings in acute salpingitis: report on a United Kingdom cohort. *BJOG* 1995;102:407–14
88. US Department of Health and Human Services.Healthy People 2010. *Sexually Transmitted Diseases*. Atlanta, GA: Centers for Disease Control and Prevention, 2010
89. Chow WH, Daling JR, Cates W Jr, et al. Epidemiology of ectopic pregnancy. Epidemiol Rev 1987;9:70–94.
90. Coste J, Job-Spira N, Fernandez H, et al. Risk factors for ectopic pregnancy: a case-control study in France, with special focus on infectious factors. Am J Epidemiol 1991;133:839–49.
91. Weström L. Influence of acute pelvic inflammatory disease on fertility. Am J Obstet Gynecol 1975;121:707–13.
92. Cacciatore B, Stenman U-H, Ylostalo P. Early screening for ectopic pregnancy in high-risk symptom-free women. *Lancet* 1994:343:517–8
93. Egger M, Low N, Smith GD, et al. Screening for chlamydial infections and the risk of ectopic pregnancy in a county in Sweden: ecological analysis. BMJ 1998;316:1776–80.
94. Sivalingam VN, Duncan WC, Kirk E, Shephard LA, Horne AW (2011).Diagnosis and management of ectopic pregnancy. J Fam Plann Reprod Health Care 37: 231–240.
95. Magers T, Talbot P, DiCarlantonio G, Knoll M, Demers D, et al. (1995) Cigarette smoke inhalation affects the reproductive system of female hamsters. Reprod Toxicol 9: 513–525.
96. Neri A, Eckerling B (1969) Influence of smoking and adrenaline (epinephrine) on the uterotubal insufflation test (Rubin test). Fertil Steril 20: 818–828.
97. Shaw JL, Oliver E, Lee KF, Entrican G, Jabbour HN, et al. (2010) Cotinine exposure increases Fallopian tube PROKR1 expression via nicotinic AChRalpha-7 : a potential mechanism explaining the link between smoking and tubal ectopic pregnancy. Am J Pathol 177: 2509–2515.

98. Bouyer J, Coste J, Shojae T, Pouly JL, Fernandez H, Gerband L, et al. Risk factors for ectopic pregnancy: a comprehensive analysis based on a large case control population based studies in France. Am J Epidemiol 2003; 157: 185-94.
99. Shafquat T, Wahab S, Bawar S, Rahim R. Relation of age, parity and duration of subfertility as risk factors for ectopic pregnancy, Gomal J Med Sci 2013; 11:171-3.
100. Ego A, Subtil D, Cosson M, Legoueff F, Houfflin – Debarge U, Querleu D. Survival analysis of fertility after ectopic pregnancy. Fertil Steril 2001;75:560-566.
101. Job-Spira N, Bouyer J, Pouly JL, Germain E, Coste J, Aublet-Cuvelier B, Fernandez H. Fertility after ectopic pregnancy: first results of a population-based cohort study in France. Hum Reprod 1996; 11: 99-104.
102. Tharaux-Deneux C, Bouyer J, Job-Spira N, Coste J, Spira A. Risk of ectopic pregnancy and previous induced abortion. Am J Public Health 1998; 88: 401-5.
103. Bo Zhu, Gu-feng Xu, Yi-feng Liu, Fan Qu, Wei-miao Yao, Yi-min Zhu, Hui-juan Gao, Dan Zhang. Heterochronic bilateral ectopic pregnancy after ovulation induction.Journal of Zhejiang University SCIENCE B August 2014, Volume 15, Issue 8, pp 750-755.
104. Combined bilateral ectopic and intrauterine pregnancy following ovulation induction with the low-dose step-up protocol in a patient with polycystic ovary syndrome. Yarali, Hakan; Bukulmez, Orhan; Gurgan, Timur // Archives of Gynecology & Obstetrics;Jul2000, Vol. 264 Issue 1, p37
105. Case Report of Heterotopic Pregnancy with Clomiphene Citrate. Naki, M.M. // Fertility Weekly;10/2/2006, p3
106. Clayton, H.B., Schieve, L.A., Peterson, H.B., *et al.*, 2006. Ectopic pregnancy risk with assisted reproductive technology procedures. *Obstet. Gynecol.*, **107**(3):595–604.[doi:10.1097/01.AOG.0000196503.78126.62]
107. Nazari, A., Askari, H.A., Check, J.H., *et al.*, 1993. Embryo transfer technique as a cause of ectopic pregnancy in *in vitro* fertilization. *Fertil. Steril.*, **60**(5):919–921

108. Pope CS, Cook EK, Arny M, Novak, Grow DR,. Influence of embryo transfer depth on in vitro fertilization and embryo transfer outcome. Fertil and Steril 2004; 81: 51-58.
109. Zouves C, Erenus M, Gomel V. Tubal ectopic pregnancy after in vitro fertilization and embryo transfer; A role for proximal occlusion or salphingectomy after failed distal tubal surgery.Fertil and Steril 1991.
110. Comprehensive Gynaecology, 3rd edition. Missouri, St Louis: *Mosby* 1997; 432.
111. Latchaw G, Takacs P, Gaitan L, Geren S, Burzawa J. Risk factors associated with the rupture of tubal ectopic pregnancy. GynecolObstet Invest. 2005; 60 (3):177-80.
112. Bhattacharya S, McLernon DJ, Lee AJ, Bhattacharya S (2012) Reproductive OutcomesFollowing Ectopic Pregnancy: Register-Based Retrospective Cohort Study. PLoS Med 9(6): e1001243. doi:10.1371/journal.pmed.1001243.
113. Klentzeris DL. Ectopic pregnancy. Textbook of Gynaecology,(3rd edn). Edited by Robert W Shaw, W Patrick, Sautter and Stuart L Stanton 2003; 371-86.
114. Bernoux A, Job-Spira N, Germain E, Coste J, Bouyer J (2000) Fertility outcome after ectopic pregnancy and use of an intrauterine device at the time of the index ectopic pregnancy. Human Reprod 15: 1173–1177.
115. Aziz S, Wafi BA, Swadi HA (2011) Frequency of ectopic pregnancy in a Medical Centre, Kingdom of Saudia Arabia . J Pak Med Assoc 61: 221-224.
116. Weström L, Bengtsson LP, Mardh PA. The risk of pelvic inflammatory disease in women using intrauterine contraceptive devices compared to nonusers. Lancet 1976;2:221–4.
117. Vessey MP, Yeates D, Flavel R, et al. Pelvic inflammatory disease and the intrauterine device: findings of a large cohort study. Br Med J 1981;282:855–7.
118. Hodgson BJ, Talo A. Spike bursts in rabbit oviduct: effects of estrogens and progesterone. Am J Physiol. 1978;234:439-45.
119. Sheffer-Mimouni G, Pauzner D, Maslovitch S, Lessing JB, Gamzu R. Ectopic pregnancies following emergency levonorgestrel contraception.Contraception. 2003;67(4):267-9.

120. Croxatto HB, Diaz S, Salvatierra AM, Morales P, Ebensperger C,Brandeis A. Treatment with Norplant subdermal implants inhibits sperm penetration through cervical mucus in vitro. Contraception.1987;36:193-201.
121. Schwarz UI, Buschel B, Kirch W. Unwanted pregnancy on selfmedication with St. John's wort despite hormonal contraception. Br J Clin Pharmacol. 2003;55:112-3.
122. Paltieli Y, Eibschitz I, Ziskind G, Ohel G, Silbermann M, Weichselbaum AJ. High progesterone levels and ciliary dysfunction—a possible cause of ectopic pregnancy. J Assist Reprod Genet. 2000;17(2):103-6.
123. Liukko P, Erkkola R, Laakso L. Ectopic pregnancies during use of low dose progestógenos for oral contraception. Contraception. 1977;16:575- 80.
124. Shoupe D, Mishell DR, Bradford L. The significance of bleeding patterns in Norplant users. Obstet Gynecol. 1991;77:256-62.
125. Faúndes A, Brache V, Alvarez F. Emergency contraception—clinical and ethical aspects. Int J Gynaecol Obstet. 2003,82:297-305.
126. Parazzini F[1], Ferraroni M, Tozzi L, Ricci E, Mezzopane R, La Vecchia C. Induced abortions and risk of ectopic pregnancy. Hum Reprod. 1995 Jul;10(7):1841-4.
127. Doyle MB, Decherney AH, Diamond MP. Epidemiology and etiology of ectopic pregnancy. Obstet Gynecol Clin North Am 1991;18:1–152.
128. Bouyer J[1], Coste J, Fernandez HPouly JLJob-Spira N.Sites of ectopic pregnancy: a 10 year population-based study of 1800 cases. Hum Reprod. 2002 Dec;17(12):3224-30.
129. Ushakov FB, Elchalal U, Aceman PJ, Schenker JG. Cervical pregnancy: past and future. Obstet Gynecol Surv 1997;52:45-59.
130. Raskin MM. Diagnosis of cervical pregnancy by ultrasound: a case report. Am J Obstet Gynecol 1978;130:234-5.
131. Timor-Tritsch IE, Monteagurdo A, Mandeville EO, Peisner DB, Anaya GP, Pirronw EC. Successful management of a viable cervical pregnancy by local injection of methotrexate guided by transvaginal ultrasonography. Am J Obstet Gynecol 1994;170:737-9.

132. Spiegelberg O. Zur Casuistik der Ovariaschwangerschaft. Arch Gynekol 1873;13:73.
133. Kraemer B, Kraemer E, Guengoer E, et al. Ovarian ectopic pregnancy: diagnosis, treatment, correlation to Carnegie Stage 16 and review based on a clinical case. Fertil Steril 2009;92:392.e13-5.
134. Zhang Y, Chen YS, Wang JJ, Lu ZY, Hua KQ. Analysis of 96 cases with cesarean scar pregnancy. Zhonghua Fu Chan Ke Za Zhi 2010;45:664-8.
135. Lin EP, Bhatt S, Dogra VS. Diagnostic clues to ectopic pregnancy. Radiographics 2008 Oct;28(6):1661-1671.
136. Troiano RN, McCarthy SM. Mullerian duct anomalies: imaging and clinical issues. Radiology 2004 Oct;233(1):19-34.
137. Chan LY, Fok WY, Yuen PM. Pitfalls in diagnosis of interstitial pregnancy. Acta Obstet Gynecol Scand 2003 Sep;82(9):867-870.
138. Soriano D, Vicus D, Mashiach R, Schiff E, Seidman D, Goldenberg M. Laparoscopic treatment of cornual pregnancy: a series of 20 consecutive cases. Fertil Steril 2008 Sep;90(3):839-843.
139. Fylstra DL, Soper DE. Chapter 21, ectopic pregnancy. In: Gilstrap III L, Cunningham FG, Vandorsten JP, eds. Operative obstetrics, 2nd ed. Philadelphia: McGraw-Hill; 2002:355-78.
140. Yildizhan R, Kurdoglu M, Kolusari A, Erten R: Primary omental pregnancy. Saudi Med J 2008, 29:606-609.
141. Clark JFJ, Guy RS. Abdominal pregnancy. Am J Obstet Gynecol 1996;96:511-20.
142. Studdiford WE: Primary peritoneal pregnancy. Am J Obstet Gynecol 1942, 44:487-491.
143. Ang LP, Tan AC, Yeo SH: Abdominal pregnancy: a case report and literature review. Singapore Med J 2000, 41:454-457.
144. Alto WA: Abdominal pregnancy. Am Fam Physician 1990, 41:209-214.
145. Fox EJ, Mevs FF. Simultaneous bilateral tubal pregnancies, report of 2 cases. Obstet Gynecol 1963;21:499–501.

146. Fishback HR. Bilateral simultaneous tubal pregnancy. Am J Obstet Gynecol 1939;37:1035.
147. Norris S. Bilateral simultaneous tubal pregnancy. Can Med Assoc J 1953;68:379–81.
148. Bansal, P.M.C., Ectopic pregnancy. WWW.slideshare.net,, 2011.
149. Dart, R.G., B. Kaplan, and K. Varaklis, Predictive value of history and physical examination in patients with suspected ectopic pregnancy. Ann Emerg Med,, 1999. **33**(3): p. 283-90.
150. Pam IC, Otubu JAM. Ectopic pregnancy. In: Agboola A, ed. *Textbook of Obstetrics and Gynaecology for Medical Students*, 2nd edn. London: Heinemann Education Publishers, 2006;101–5.
151. Shaw's Textbook of Gynaecology by V G Padubidri and S N Daftary.15th edition published by Elsevier.
152. Stovall TG, Kellerman AL, Ling FW, Buster JE. Emergency department diagnosis of ectopic pregnancy. *Ann Emerg Med*. Oct 1990;19(10):1098-103. [Medline].
153. Kruszka PS, Kruszka SJ.Evaluation of acute pelvic pain in women. *Am Fam Phys* 2010;82:141–7
154. Vyas, P.S., Epidermiology,diagnosis and management of ectopic pregnancy. LTMG Hospital,Sion, Mumbai,, 1993-1998.
155. Abbott J, Emmans LS, Lowenstein SR. Ectopic pregnancy: ten common pitfalls in diagnosis. *AmJ Emerg Med*. Nov 1990;8(6):515-22.
156. DeCherney AH, Minkin MJ, Spangler S. Contemporary management of ectopic pregnancy. J Reprod Med. Oct 1981;26(10):519-23. [Medline].
157. Brenner PF, Roy S, Mishell DR Jr. Ectopic pregnancy. A study of 300 consecutive surgically treated cases. JAMA. Feb 15 1980;243(7):673-6. [Medline].
158. Hibbard LT. Diagnosis of ectopic pregnancy. Obstet Gynecol. Apr 1956;7(4):453-8. [Medline].
159. Cartwright PS, Vaughn B, Tuttle D. Culdocentesis and ectopic pregnancy. J Reprod Med. Feb 1984;29(2):88-91. [Medline].

160. Romero R, Copel JA, Kadar N, Jeanty P, Decherney A, Hobbins JC. Value of culdocentesis in the diagnosis of ectopic pregnancy. Obstet Gynecol. Apr 1985;65(4):519-22. [Medline].
161. Vermesh M, Graczykowski JW, Sauer MV. Reevaluation of the role of culdocentesis in the management of ectopic pregnancy. Am J Obstet Gynecol. Feb 1990;162(2):411-3.(Medline).
162. Nayama M, Gallais A, Ousmane N, Idi N, Tahirou A, Garba M, et al. [Management of ectopic pregnancy in developing countries: example of a Nigerian reference maternity]. Gynecol Obstet Fertil. Jan 2006;34(1):14-8. [Medline].
163. Chard T. Pregnancy tests: a review. Hum Reprod. 1992;7:701-10.
164. Olshaker JS. Emergency department pregnancy testing. J Emerg Med. 1996;14:59-65.
165. Minaretzis D, Tsionou C, Tsandoulas E. Clinical evaluation of a sensitive urine pregnancy test in the gynecological emergencies. Eur J Obstet Gynecol Reprod Biol. 1991;39:123-6.
166. Close R. Abdominal and pelvic pain in the nonpregnant patient. In: Tintinalli JE, Kelen GD, Stapczynski JS, editors. Emergency medicine. Philadelphia: McGraw-Hill; 2003.
167. Bastian LA, Diagnostic efficiency of home pregnancy test kits. A meta analysis Arch Farm Med, 2008. **7**(5): p. 465-469.
168. R. D. Nerenz, H. Song, A. M. Gronowski. Screening Method to Evaluate Point-of-Care Human Chorionic Gonadotropin (hCG) Devices for Susceptibility to the Hook Effect by hCG Core Fragment: Evaluation of 11 Devices. Clinical Chemistry, 2014; 60 (4): 667 DOI: 10.1373/clinchem.2013.217661.
169. Gracia C.R. Barnhart KT., Diagnosing ectopic pregnancy: decision analysis comparing six strategies. Obstet. Gynecol, 2001. **97**: p. 464-70.
170. Silva C, Sammel MD, Zhou L, et al. Human chorionic gonadotropin profile for women with ectopic pregnancy. Obstet Gynecol. 2006; 107:605–610. [PubMed: 16507931]

171. Li TC, Tristram A, Hill AS, et al. A review of 254 ectopic pregnancies in a teaching hospital in the Trent Region, 1977-1990. Hum Reprod. 1991; 6:1002–1007. [PubMed: 1722218]
172. McCord ML, Muram D, Buster JE, et al. Single serum progesterone as a screen for ectopic pregnancy: exchanging specificity and sensitivity to obtain optimal test performance. Fertil Steril. 1996; 66:513–516. [PubMed: 8816609]
173. Nama V, Manyonda I. Tubal ectopic pregnancy: diagnosis and management. Arch Gynecol Obstet. 2009; 279:443–453. [PubMed: 18665380]
174. Barnhart K, Mennuti MT, Benjamin I, et al. Prompt diagnosis of ectopic pregnancy in an emergency department setting. Obstet Gynecol. 1994; 84:1010–1015. [PubMed: 7970455]
175. Romero R, Kadar N, Jeanty P et al. Diagnosis of ectopic pregnancy: value of the discriminatory human chorionic gonadotropin zone. Obstet Gynecol 1985; 66: 357–360.
176. Aleem FA, DeFazio M & Gintautas J. Endovaginal sonography for the early diagnosis of intrauterine and ectopic pregnancies. Hum Reprod 1990; 5(6): 755–758.
177. Morin L, Van den Hof MC. Diagnostic Imaging Committee, Society of Obstetricians and Gynaecologists of Canada. Ultrasound evaluation of first trimester pregnancy complications. J Obstet Gynaecol Can. 2005; 27:581–591. [PubMed: 16100636].
178. Royal College of Obstetricians and Gynaecologists and Royal College of Radiologists Faculty of Clinical Radiology. Guidance on Ultrasound Procedures in Early Pregnancy. RCOG Press; London, UK: 2005.
179. Ahmed AA, Tom BD, Calabrese P. Ectopic pregnancy diagnosis and the pseudo-sac. Fertil Steril. 2004; 81:1225–1228. [PubMed: 15136081]
180. Reyftmann L, Dechaud H, Hedon B. Alert for heterotopic pregnancy. Fertil Steril. 2007; 88:759–60. [PubMed: 17681335]
181. Condous G, Okaro E, Khalid A, et al. The accuracy of transvaginal ultrasonography for the diagnosis of ectopic pregnancy prior to surgery. Hum Reprod. 2005; 20:1404–1409. [PubMed: 15695311]

182. Perriera L, Reeves MF. Ultrasound criteria for diagnosis of early pregnancy failure and ectopic pregnancy. Semin Reprod Med. 2008; 26:373–382. [PubMed: 18825605]
183. Condous G, Lu C, Van Huffel SV, et al. Human chorionic gonadotrophin and progesterone levels in pregnancies of unknown location. Int J Gynaecol Obstet. 2004; 86:351–357. [PubMed:15325852]
184. Condous G. The management of early pregnancy complications. Best Pract Re Clin Obstet Gynaecol 2004; 18: 37–57.
185. Goldstein S & Timor-Tritsch IE. Ultrasound in gynecology. New York: Churchill Livingstone, 1995. Chapter 15, 228.
186. Kirk E, Daemen A, Papageorghiou AT et al. Why are some ectopic pregnancies characterized as pregnancies of unknown location at the initial transvaginal ultrasound examination? Acta Obstet Gynecol Scand 2008; 87(11): 1150–1154.
187. Jermy K, Thomas J, Doo A et al. The conservative management of interstitial pregnancy. BJOG 2004; 111: 1283–1288
188. Jurkovic D, Hacket E & Campbell S. Diagnosis and treatment of early cervical pregnancy: a review and a report of two cases treated conservatively. Ultrasound Obstet Gynecol 1996; 8: 373–380.
189. Jurkovic D, Hillaby K, Woelfer B et al. First-trimester diagnosis and management of pregnancies implanted into the lower uterine segment Cesarean section scar. Ultrasound Obstet Gynecol 2003; 21: 220–227.
190. Braffman BH, Coleman BG, Ramchandani P et al. Emergency department screening for ectopic pregnancy: a prospective US study. Radiology 1994; 190: 797–802.
191. Shalev E, Yarom I, Bustan M et al. Transvaginal sonography as the ultimate diagnostic tool for the management of ectopic pregnancy: experience with 840 cases. Fertil Steril 1998; 69: 62–65.
192. Atri M, Valenti DA, Bret PM et al. Effect of transvaginal sonography on the use of invasive procedures for evaluating patients with a clinical diagnosis of ectopic pregnancy. J Clin Ultrasound 2003; 31: 1–8.

193. Kirk E, Papageorghiou AT, Condous G et al. The diagnostic effectiveness of an initial transvaginal scan in detecting ectopic pregnancy. Hum Reprod 2007; 22: 2824–2828.
194. Barnhart KT, Sammel MD, Rinaudo PF, et al. Symptomatic patients with an early viable intrauterine pregnancy: HCG curves redefined. Obstet Gynecol. 2004; 104:50–55. [PubMed: 15229000]
195. Tamai K, Koyama T, Togashi K. MR features of ectopic pregnancy. *Eur Radiol* 2007; 17:3236–3246.
196. Parker RA III, Yano M, Tai AW, Friedman M, Narra VR, Menias CO. MR imaging findings of ectopic pregnancy: a pictorial review. *Radio- Graphics* 2012; 32:1445–1460
197. Wolfman DJ. Invited commentary. *RadioGraphics* 2009; 20:2003–2005
198. Kanal E, Barkovich AJ, Bell C, et al. ACR guidance document for safe MR practices. *AJR* 2007; 188:1447–1474
199. Singh AK, Desai H, Novelline RA. Emergency MRI of acute pelvic pain: MR protocol with no oral contrast. *Emerg Radiol* 2009; 16:133–141.
200. Takahashi A[1], Takahama J, Marugami N, Takewa M, Itoh T, Kitano S, Kichikawa K. Ectopic pregnancy: MRI findings and clinical utility. Abdom Imaging. 2013 Aug;38(4):844-50. doi: 10.1007/s00261-012-9969-0.
201. Horne AW, Shaw JL, Murdoch A, et al. Placental growth factor: a promising diagnostic biomarker for tubal ectopic pregnancy. J Clin Endocrinol Metab. 2011; 96:E104–E108. [PubMed: 21047920]
202. Lundorff P, Thorburn J, Hahlin M, et al. Laparoscopic surgery in ectopic pregnancy. A randomized trial versus laparotomy. Acta Obstet Gynecol Scand. 1991; 70:343–348. [PubMed: 1836087]
203. Vermesh M, Silva PD, Rosen GF, et al. Management of unruptured ectopic gestation by linear salpingostomy: a prospective, randomized clinical trial of laparoscopy versus laparotomy. Obstet Gynecol. 1989; 73:400–404. [PubMed: 2464777
204. Murphy AA, Nager CW, Wujek JJ, et al. Operative laparoscopy versus laparotomy for the management of ectopic pregnancy: a prospective trial. Fertil Steril. 1992; 57:1180–1185. [PubMed: 1534771]

205. Robson SJ, O'Shea RT. Undiagnosed ectopic pregnancy: a retrospective analysis of 31 'missed' ectopic pregnancies at a teaching hospital. Aust N Z J Obstet Gynaecol. 1996; 36:182–185. [PubMed: 8798311]
206. Lund, JJ.- Early ectopic pregnancy treated nonsurgically. J. Obstet. Br. Empire, 62: 70-6,1955.
207. Elito Jr J, Montenegro NA, Soares RC, Camano L. Unruptured ectopic pregnancy: diagnosis and treatment. State of art. Rev Bras Ginecol Obstet. 2008 Mar;30(3):149-59.
208. Han KK; Elito Jr J, Camano L. Conduta expectante para gravidez tubária íntegra. Rev Bras Ginecol Obstet. 1999; 21(8):465-70.
209. Elito Jr J, Han KK, Camano L. Tubal patency following surgical and clinical treatment of ectopic pregnancy. Sao Paulo Med J. 2006 Sep 7;124(5):264-6.
210. Rantala M, Ma¨kinen J. Tubal patency and fertility outcome after expectant management of ectopic pregnancy. Fertil Steril 1997;68:1043–6.
211. Debby A, Golan A, Sadan O, Zakut H, Glezerman M. Fertility outcome following combined methotrexate treatment of unruptured extrauterine pregnancy. BJOG 2000; 107: 626–30. 3.
212. Glatstein IZ, Sleeper LA, Law Y, Simon A, Adoni A, Laufer N, et al. Observer variability in the diagnosis and management of the hysterosalpingogram. Fertil Steril 1997;67:233–7
213. Mol BW, Swart P, Bossut PM, Van Der Veen F. Hysterosalpingography an important tool in predicting fertility outcome? Fertil Steril 1997;67:663–9.
214. Elito Jr J, Han KK, Camano L. Tubal patency after clinical treatment of unruptured ectopic pregnancy. Int J Gynaecol Obstet. 2005a; 88(3):309-13.
215. Stovall TG, Ling FW, Gray LA, Carson SA, Buster JE. Methotrexate treatment of unruptured ectopic pregnancy: a report of 100 cases. Obstet Gynecol 1991;77:749– 53.
216. Li MC, Hertz R, Spencer, DB. Effect of methotrexate therapy upon choriocarcinoma and chorioadenoma. Proc. Sci. Exp. Biol. Med., 93: 361-9, 1956.
217. Barnhart K, Coutifaris C, Esposito M. The pharmacology of methotrexate. Expert Opin Pharmacother 2001;2:409–17.

218. Tanaka T, Hayashi H, Kutsuzawa T, Fujimoto S, Ichinoe K. Treatment of intersticial ectopic pregnancy with methotrexate: report of a successful case. Fertil Steril 1982;37:851-5.
219. Stovall TG, Ling FW, Buster JE. Outpatient chemotherapy of unruptured ectopic pregnancy. Fertil Steril 1989;51:435–8.
220. Stovall TG, Ling FW. Single-dose methotrexate: an expanded clinical trial. Am J Obstet Gynecol 1993;168:1759-65.
221. Elito J Jr, Reichmann AP, Uchiyama MN, Camano L. Predictive score for the systemic treatment of unruptured ectopic pregnancy with a single dose of methotrexate. Int J Gynaecol Obstet 1999a;67:75–9.
222. Pisarska MD, Carson SA, Buster JE. Ectopic pregnancy. Lancet 1998;351:1115–20.
223. Barnhart KT, Gosman G, Ashby R, Sammel M. The medical management of ectopic pregnancy: a meta-analysis comparing ‘‘single dose and multidose’’ regimens. Obstet Gynecol 2003;101:778–84.
224. Lipscomb GH, McCord ML, Stovall TG, Huff G, Portera SG, Ling FW. Predictors of success of methotrexate treatment in women with tubal ec- topic pregnancies. N Engl J Med 1999;341:1974–8.
225. Brown DL, Felker RE, Stovall TG, Emerson DS, Ling FW. Serial endovaginal sonography of ectopic pregnancies treated with methotrexate. Obstet. Gynecol., 77: 406-9, 1991.
226. Atri M, Bret PM, Tulandi T, Senterman MK. Ectopic pregnancy: evolution after treatment with transvaginal methotrexate. Radiology 1992;185:749–53.
227. Lipscomb GH, Puckett KJ, Bran D, Ling FW. Management of separation pain after singledose methotrexate therapy for ectopic pregnancy. Obstet Gynecol 1999;93:590–3.
228. Soares, RC; Elito Jr J, Han KK, Camano L. Endometrial thickness as an orienting factor for the medical treatment of unruptured tubal pregnancy. Acta Obstet Gyn Scand, 2004:83(3): 289-92.
229. Elito Jr J, Han KK, Camano L. Values of beta-human chorionic gonadotropin as a risk factor for tubal obstruction after tubal pregnancy. Acta Obstet Gynecol Scand. 2005b; 84(9):864-7.

230. Lipscomb GH, Stovall TG, Ling FW. Nonsurgical treatment of ectopic pregnancy. N Engl J Med 2000;343:1325–9.
231. Feichtinger, W.; Kemeter, P.- Conservative treatment of ectopic pregnancy by transvaginal aspiration under sonographic control and MTX injection. Lancet. 1987; 1: 381-92.
232. Pansky, M.; Bubowsky, I.- Local methotrexate injection: a nonsurgical treatment of ectopic pregnancy. Am. J. Obstet. Gynecol. 1989; 161: 363-8.
233. Fernandez, H.; Benifla, J. A.- Metotrexate treatment of ectopic pregnancy - 100 cases treated by primary transvaginal injection under sonographic control. Fertil. Steril. 1993; 59: 773-7.
234. Benifla JL, Fernandez H, Sebban E, Darai E, Frydman R, Madelenat P. Alternative to surgery of treatment of unruptured interstitial pregnancy: 15 cases of medical treatment. Eur J Obstet Gynecol Reprod Biol 1996;70:151-6.
235. Sagiv R, Golan A, Arbel-Alon S, Glezerman M. Three conservative approaches to treatment of interstitial pregnancy. J Am Assoc Gynecol Laparosc 2001;8:154-8.
236. Lin YS, Chen CL, Yuan CC, Wang PH. Successful rescue of an early interstitial pregnancy after failed systemic methotrexate treatment: a case report. J Reprod Med 2007;52:332-4
237. Chetty M, Elson J. Treating non-tubal ectopic pregnancy. Best Pract Res Clin Obstet Gynaecol. 2009 Aug;23(4):529-38
238. Parker J, Bisits A. Laparoscopic surgical treatment of ectopic pregnancy: salpingectomy or salpingostomy? Aust N Z J Obstet Gynaecol. 1997; 37:115–117. [PubMed: 9075562]
239. Clausen I. Conservative versus radical surgery for tubal pregnancy. A review. Acta Obstet Gynecol Scand. 1996; 75:8–12. [PubMed: 8561006]
240. Thornton KL, Diamond MP, DeCherney AH. Linear salpingostomy for ectopic pregnancy. Obstet Gynecol Clin North Am. 1991; 18:95–109. [PubMed: 1923258]
241. Mukul LV, Teal SB. Current management of ectopic pregnancy. Obstet Gynecol Clin North Am. 2007; 34:403–19. [PubMed: 17921007

242. Gilman, A.; Goodman, LS.; Goodman, A.; Calabresi, P.; Chabner, BA. Antineoplastic agents. In: Gilman, A.; Goodman, LS.; Goodman, A., editors. The Pharmacologic Basis of Therapeutics. 8th edn. Macmillan Publishing; New York, NY: 1990. p. 1275-1276.
243. Rulin MC. Is salpingostomy the surgical treatment of choice for unruptured tubal pregnancy? Obstet Gynecol. 1995; 86:1010–1013. [PubMed: 7501323]
244. Timor-Tritsch IE, Monteagudo A, Matera C, Veit CR. Sonographic evolution of corneal pregnancies treated without surgery. Obstet Gynecol 1992;79:1044-9.
245. Ackerman TE, Levi CS, Dashefsky SM, Holt SC, Lindsay DJ. Interstitial line: sonographic finding in interstitial (cornual) ectopic pregnancy. Radiology 1993;189:83-7.
246. De La Veja GA, Avery C, Nemiroff R, Marchiano D. Treatment of early cervical pregnancy with cerclage, carboprost, curettage and balloon tamponade. Obstet Gynecol. 2007; 109:505-7.
247. Xu B, Wang YK, Zhang YH, Wang S, Yang L, Dai SZ. Angiographic uterine artery embolization followed immediate curettage: an efficient treatment for controlling heavy bleeding and avoiding recurrent bleeding in cervical pregnancy. J Obstet Gynaecol Res 2007; 33:190-4.
248. Monteagudo A, Minior VK, Stephenson C, Monda S, Timor-Tritsch E. Non-surgical management of live ectopic pregnancy with ultrasound-guided local injection: a case series. Ultrasound Obstet Gynecol 2005; 25:282-8.
249. Mesogitis S, Pilalis A, Daskalakis G, Papantoniou N, Antsaklis A. Management of early viable cervical pregnancy. BJOG 2005; 112:409-11.
250. Bai SW, Lee JS, Park JH, Kim JY, Jung KA, Kim SK, Park KH. Failed methotrexate treatment of cervical pregnancy. Predictive factors. J Reprod Med. 2002; 47:483-8.
251. Chuang J, Seow KM, Cheng WC, Tsai YL, Hwang JL. Conservative treatment of ectopic pregnancy in a caesarean section scar. BJOG. 2003 Sep;110(9):869-70.

252. Einenkel J, Baier D, Horn L-C et al. Laparoscopic therapy of an intact primary ovarian pregnancy with ovarian hyper- stimulation syndrome. Hum Reprod 2000; 15(9): 2037–2040.
253. Ayinde OA, Aimakhu CO, Adeyanju OA et al. Abdominal pregnancy at the University College Hospital, Ibadan: a ten-year review. Afr J Reprod Health 2005; 9: 123–127.
254. Oki T, Baba Y, Yoshinaga M et al. Super-selective arterial embolization for uncontrolled bleeding in abdominal pregnancy. Obstet Gynecol 2008; 112: 427–429.
255. Chin HY, Chen FP, Wang CJ, Shui LT, Liu YH, Soong YK. Heterotopic pregnancy after in vitro fertilization-embryo transfer. Int J Gynaecol Obstet 2004;86:411–6.
256. Fernandez H, Gervaise A. Ectopic pregnancies after infertility treatment: modern diagnosis and therapeutic strategy. Hum Reprod Update 2004;10:503–13.
257. R. C (Karki) L, Pradhan B, Duwa S. Annual Analysis of Ectopic Pregnancy in Tertiary Care Hospital. PMJN 2011;11 :5-8

Sl.No	Name	Age(in years)	I.P.No	D.O.A	Obstetric Code	LMP	Period of Amenorrhoea (in Days)	UPT	Duration of Subfertility	Sterilisation Status	Time Since ST (Years)	Method of ST	Other Contra Ceptive Use	Risk Factors	Clinical Symptoms	B.P(in mmHg)	P.Rt(per minute)	Hb %	Sr.β HsCG(M IU/ml)	P/A Findings	P/V Findings	Cervix Excitation Test	Coldecentesis	Ultrasound Findings	Additional Imaging	Clinical Diagnosis	Time interval Between Admission and Start of definitive Treatment	Time Delay Due to	Medical Management Criteria	Management	Registered (R) / Unregistered (UR)	Side	Site	Haemoperitoneum (in ml)	Other Findings	No.Of Units of Blood / Blood Products Transfusion	ICU Care	Fever	Duration of Hospital Stay (in Days)	Final Diagnosis/HPE report
1	Jeyadevi	22	39910	13.07.12	p4m1	14.05.12	60	P	3 m	NA	NA	NA	nil	nil	AM,LAP,BPV	130/80	114	9	ND	T	FF	P	ND	EU,Rt A mass	ND	ectopic	1 hr 20 min	NA	NA	RSO	R	Rt	ovary	1000	nil	1 WB	1 day	PR	7	ectopic
2	poo esakkiammal	41	40190	15.07.12	G3P2L2	25.05.12	49	P	NA	NS	NA	NA	nil	PID,prev LSCS	AM,LAP,BPV	100/70	98	5.7	ND	T	FT	P	ND	EU,Rt A mass,FL	ND	ectopic	1 hr 50 min	NA	NA	RSO,Lt salphingectomy	R	Rt	Ampulla	1250	haemorrhagic right ovary	2 WB	1 day	PR	7	ectopic
3	Velthai	29	42587	27.07.12	p4m1	16.06.12	41	P	5 y	NA	NA	NA	nil	nil	LAP,vomit	80/40	112,F	4.4	ND	D,T	FT	P	ND	EU,Lt A mass,FL	ND	ectopic	30 min	NA	NA	Lt partial salphingectomy	R	Lt	isthmus	1600	nil	3 WB	3 days	AB	7	ectopic
4	poomari	44	43500	01.08.12	G3P2L1	16.06.12	45	N	NA	NS	NA	NA	nil	PID	vomit,dysuria	120/70	104	4.5	ND	D,T	FF	N	P	EU,Rt A mass,FL	ND	ectopic	24 hrs	late diagnosis	NA	RSO	R	Rt	ovary	1200	Rt twisted ovarian mass	2 WB	2 days	PR	10	Rt ovarian cyst
5	mary	38	42892	30.07.12	G3P1L1A1	06.06.12	54	P	NA	NS	NA	NA	nil	prev ectopic	AM,LAP	120/80	80	13	21929	nil	nil	N	ND	EU,Lt A mass of 12 mm	ND	ectopic	18 hrs	late diagnosis	NA	Lt totals alphingectomy	R	Lt	fimbria	100	dense adhesions	nil	nil	AB	9	ectopic
6	alagu	28	45696	15.08.12	G2A1	30.06.12	46	P	2 y	NA	NA	NA	nil	prev abortion	AM,LAP,BPV	140/90	80	11.1	1332	D	FT	N	ND	EU,Rt A. mass, FL	ND	ectopic	1 hr 15 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	250	nil	nil	nil	PR	7	ectopic
7	vennila	22	47179	23.08.12	p4m1	4.07.12	50	P	6m	NA	NA	NA	nil	nil	AM,LAP,BPV	120/70	86	12	ND	T	FT	P	ND	EU,Rt A.mass,FL	ND	ectopic	1 hr 30 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	600	nil	1 WB	nil	AB	7	ectopic
8	veerammal	21	48243	29.08.12	p4m1	22.06.12	68	P	nil	NA	NA	NA	nil	nil	AM,LAP,BPV	100/70	96	8.1	ND	D	FT	P	ND	EU,Lt A.mass,FL	ND	ectopic	1 hr	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	1 000	nil	2 WB	1 day	PR	9	ectopic
9	Mumtaj	24	48703	01.09.12	G3P2L2	16.07.12	47	P	nil	NS	NA	NA	nil	nil	AM,LAP,giddi	80/50	130	5.4	ND	D,T	FT,FF	P	P	EU,FL	ND	ectopic	30min	NA	NA	LSO	R	Lt	isthmus	1100	Lt haemorrhagic ovarian cyst	2 WB	2 days	PR	12	ectopic
10	seetha	30	48795	02.09.12	p4m1	11.07.12	53	P	nil	NA	NA	NA	nil	nil	AM,LAP,BPV	100/70	80	11	ND	D	nil	N	P	EU,Rt A. mass, FL	ND	ectopic	1 hr	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	650	nil	1 WB	nil	AB	7	ectopic
11	michaelammal	35	49591	05.09.12	p4m1	08.07.12	59	P	5y	NA	NA	NA	nil	nil	AM ,LAP,diarr	60/?	>120,F	6.6	ND	D,T	FT	P	P	EU,FL	ND	ectopic	30 min	NA	NA	Lt partial salphingectomy	R	Lt	isthmus	1500	nil	4 WB	3 days	AB	8	ectopic
12	subbulakshmi	24	49771	06.09.12	G2P1L1	26.07.12	42	P	NA	NS	NA	NA	nil	nil	AM,LAP	90/50	116	6.7	ND	D,T	FT	P	P	EU,FL	ND	ectopic	45 min	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	1800	nil	4 WB	2 days	PR	8	ectopic
13	amutha	35	50118	08.09.12	G2P1L1	19.07.12	51	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,vomit	110/70	72	10	ND	D	nil	N	ND	EU,Rt A.mass, FL	ND	ectopic	1hr	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	150	nil	nil	nil	AB	7	ectopic
14	bavu sundari	35	50846	12.09.12	p4m1	29.07.12	45	P	10y	NA	NA	NA	nil	nil	AM,LAP	110/70	80	8	ND	nil	FT	P	ND	EU,Rt A. mass, FL	ND	ectopic	1hr 20 min	NA	NA	Rt partial salphingectomy	R	Rt	isthmus	500	nil	1 WB	nil	AB	7	ectopic
15	muthulakshmi	24	51048	13.09.12	p4m1	04.07.12	71	P	1 y	NA	NA	NA	nil	nil	AM,LAP	80/40	126	9.2	ND	D	FT,FF	P	P	EU,FL	ND	ectopic	2 hr 30 min	IMCU admn	NA	Lt partial salphingectomy	R	Lt	Ampulla	1700	nil	3 WB	2days	PR	8	ectopic
16	mary stella	31	51378	15.09.12	G3P2L2	01.08.12	45	P	NA	NS	NA	NA	nil	PID	AM,LAP	100/70	92	8.7	ND	D	FT	P	ND	EU,FL	ND	ectopic	50 min	NA	NA	B/L total salphingectomy	R	Lt	isthmus	400	Rt haematosalphinx	1 WB	nil	AB	7	ectopic
17	esakkiammal	29	51829	18.09.12	G3P2L2	27.08.12	22	P	NA	NS	NA	NA	nil	nil	LAP	120/70	76	12.3	ND	nil	FT	P	ND	EU,Rt A. sac 3.5 cm	ND	ectopic	11 hrs	late diagnosis	A	B/L total salphingectomy	UR	Rt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
18	sajitha	28	52291	20.09.12	G3P2P2	29.07.12	53	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	70/40	112,F	5.6	ND	nil	FT	P	ND	EU,Rt A. mass, FL	ND	ectopic	30 min	NA	NA	B/L total salphingectomy	R	Rt	Ampulla	1100	nil	2 WB	1 day	PR	8	ectopic
19	chithra	26	52508	21.09.12	p4m1	10.08.12	42	P	1 y	NA	NA	NA	nil	nil	AM,LAP,BPV	110/70	82	11.6	ND	nil	nil	P	ND	EU,Rt A. mass, FL	ND	ectopic	25min	NA	NA	Rt total salphingectomy	R	Rt	isthmus	200	nil	nil	nil	AB	7	ectopic
20	thanamani	34	55737	09.10.12	G4P2L2A1	27.08.12	43	P	NA	S	9Y	TAT	NA	prev ectopic,LSCS,tubal sx	AM,LAP	120/80	78	12	ND	nil	FT	P	ND	EU,Rt A. mass 5cm	ND	ectopic	1 hr 10 min	NA	NA	RSO	tubal abortion	Rt	fimbria	50	adhesions	1 WB	nil	PR	7	ectopic
21	karupee	25	57069	16.10.12	G2P1L1	12.09.14	34	P	NA	NS	NA	NA	nil	nil	LAP,vomit	80/30	>120,F	5.1	ND	D	FT	P	ND	EU,Rt A.mass, FL	ND	ectopic	3 hrs	sx side admn	NA	Rt partial salphingectomy	R	Rt	cornua	2000	nil	4 WB	2 days	AB	10	ectopic
22	lakshmi	33	58466	25.10.12	G2P1L1	14.08.12	72	P	NA	NS	NA	NA	nil	nil	AM,LAP,BPV	110/70	86	8.8	ND	nil	nil	N	ND	EU,Lt A.viable 9 wks getation	ND	ectopic	1hr	NA	NA	Lt totals alphingectomy	UR	Lt	ent.tube	nil	bowel loops adherent	nil	nil	PR	7	ectopic
23	perinbam	35	59774	31.10.12	p4m1	19.08.12	73	P	3y	NA	NA	NA	nil	nil	AM,LAP	110/70	82	8.7	ND	nil	FT	P	ND	EU,FL	ND	ectopic	30 min	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	500	nil	1 WB	nil	PR	10	ectopic
24	maharasi	27	61324	09.11.12	G2P1L1	20.09.14	50	P	NA	NS	NA	NA	nil	prev LSCS, prev RSO	AM,LAP	120/80	88	8.7	ND	T	nil	N	ND	EU,Rt A. mass, FL	ND	ectopic	50 min	NA	NA	Rt cornual resection	R	Rt	cornua	1000	Lt side corpus luteal cyst	2 WB	1 day	AB	8	ectopic
25	rejina	33	63153	18.11.12	G2P1L1	16.08.12	94	N	NA	NS	NA	NA	nil	nil	AM	120/80	78	13.5	ND	nil	FF	N	ND	EU,Lt A. mass	ND	ectopic	2 hrs	NA	NA	B/L ovarian cystectomy	NA	NA	NA	nil	B/L ovarian dermoid cyst	nil	nil	AB	7	dermoid cyst
26	vasanthi	29	63203	18.11.12	G2P1L1	30.09.12	49	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	80/?	116,F	6.3	ND	nil	FF	P	ND	EU,FL	ND	ectopic	30 min	NA	NA	Lt totals alphingectomy	R	Lt	fimbria	1200	nil	3 WB	1 day	PR	9	ectopic
27	balammal	25	64640	25.11.12	p4m1	29.10.12	27	P	1y	NA	NA	NA	nil	nil	LAP	120/70	92	9.2	ND	D,T	FT	P	P	EU,FL	ND	ectopic	30 min	NA	NA	Lt partial salphingectomy	R	Lt	isthmus	900	nil	2 WB	nil	PR	7	ectopic
28	eswari	36	65544	29.11.12	G3A2	20.10.12	40	P	12y	NA	NA	NA	nil	abortions, PID	AM,LAP,BPV	140/90	120	7	ND	D,T	FT,FF	P	P	EU,Lt A. mass, FL	ND	ectopic	20 min	NA	NA	Lt partial salphingectomy	R	Lt	isthmus	1000	B/L hydrosalphinx	2 WB	1 day	PR	13	ectopic
29	muthumari	28	66879	04.12.12	G3A2	20.10.12	45	P	3y	NA	NA	NA	nil	abortions	AM,LAP	130/80	110	8.8	ND	nil	A.Mass	P	P	EU,Rt A. mass, FL	ND	ectopic	35 min	NA	NA	Lt totals alphingectomy	R	Lt	Ampulla	400	adhesions	2 WB	nil	AB	7	ectopic
30	deivanai	28	66736	05.12.12	p4m1	05.10.12	61	P	4y	NA	NA	NA	nil	ovulation induction	AM,LAP	110/70	86	10.9	ND	nil	nil	N	ND	EU,Lt A. viable fetus of 8 wks	ND	ectopic	1 hr 30 min	NA	NA	Lt partial salphingectomy	UR	Lt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
31	sagayamary	27	69009	17.12.12	G2P1L1	23.11.12	24	P	NA	NS	NA	NA	nil	prev LSCS	LAP,vomit,giddi	120/80	96	8.4	ND	G	FT	P	ND	EU,FL	ND	ectopic	3 hrs	sx side admn	NA	Lt totals alphingectomy	R	Lt	Ampulla	1000	adhesions	2 WB	nil	PR	7	ectopic
32	pulamadi	33	70033	22.12.12	G3P2L2	14.11.12	38	P	NA	S	4Y	PS	NA	tubal surgery	LAP	120/70	84	11.7	ND	nil	FF	P	ND	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	tubal abortion	Rt	fimbria	25	nil	nil	nil	AB	7	ectopic
33	thirupathi	32	71437	30.12.12	G3P2L2	29.10.12	62	P	NA	S	9Y	LS	NA	tubal surgery	AM,LAP,BPV	100/60	106	8.7	ND	D,T	nil	N	ND	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	B/L total salphingectomy	R	Rt	fimbria	700	nil	1 WB	nil	AB	7	ectopic
34	ajeswari	29	36	01.01.13	p4m1	11.11.12	51	P	4 y	NA	NA	NA	nil	nil	AM,LAP,BPV	110/70	92	10.2	ND	nil	FT,FF	P	ND	EU,FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	800	Lt ovarian corpus luteal cyst	2 WB	1 day	AB	9	ectopic
35	sindu	28	1665	08.01.13	G3P2L2	10.11.12	59	P	NA	S	3Y	coni	NA	tubal sx,prev LSCS	AM,LAP,BPV	100/70	88	12.5	ND	nil	nil	N	ND	normal study	doppler -Lt A.mass	ectopic	18 hrs	late diagnosis	A	B/L total salphingectomy	UR	Rt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
36	aruna	33	4029	19.01.13	G5P2L2A2	19.09.12	62	P	NA	S	7Y	TAT	NA	abortions, tubal sx	AM,LAP,BPV	120/80	118	7.3	ND	nil	nil	P	ND	EU,Lt A. viable 9 wks getation	ND	ectopic	40min	NA	NA	RSO,Lt salphingectomy	R	Lt	fimbria	600	nil	2 PCV	nil	AB	8	ectopic
37	roselin	25	4337	21.01.13	G4P3L3	25.12.12	27	P	NA	NS	NA	NA	nil	PID	AM,LAP	120/80	88	11.7	ND	nil	FT,A.Mass	P	ND	EU,Rt A. mass of 6.5 cm	ND	ectopic	30 min	NA	NA	RSO,Lt salphingectomy	R	Rt	Ampulla	400	Rt ovary unhealthy	1 WB	nil	AB	7	ectopic
38	Ramalakshmi	23	2812	13.01.13	G2A1	11.12.12	33	WP	6y	NA	NA	NA	nil	abortion	LAP	120/70	82	11.3	ND	nil	nil	N	ND	EU,FL	doppler -Lt A.mass	ectopic	8 days	late diagnosis	NA	LSO	R	Lt	Ampulla	600	adhesions	1 WB	nil	PR	26	ectopic
39	jagadeeswari	35	4850	23.01.13	G2P1L1	01.12.12	53	P	NA	NS	NA	NA	nil	nil	AM,LAP,giddi	100/70	88	9.3	ND	D,T	FT,A.Mass	P	ND	EU,Rt A. mass, FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	R	Rt	isthmus	1250	nil	2 WB	nil	AB	7	ectopic
40	pethanatchi	25	5570	26.01.13	G3P2L2	29.12.12	28	N	NA	NS	NA	NA	nil	PID	LAP	110/80	84	12	ND	nil	FF	N	ND	EU,Rt A.mass of 4.5 cm	MRI- inflam.patho	ectopic	8 days	late diagnosis	NA	RSO,Lt salphingectomy	UR	Rt	ovary	nil	nil	1 WB	nil	AB	17	ectopic
41	vadha	23	7976	06.02.13	G2P1L1	NK	NK	P	NA	NS	NA	NA	IUCD	IUCD	LAP	120/70	76	9	ND	nil	nil	N	ND	EU,Lt A. cyst of 5cm	ND	ectopic	5 hrs	late diagnosis	NA	Lt ovariotomy,D&C	NA	NA	NA	nil	Lt ovary enlarged,cystic	nil	nil	AB	7	ovarian cyst
42	lakshmi	29	9622	13.02.13	G3P2L2	09.01.13	35	P	NA	NS	NA	NA	EC	em.pills	LAP,BPV	110/70	126	12	ND	T	FT,FF	P	ND	EU,Rt A. cyst ,FL	ND	ectopic	30 min	NA	NA	RSO,Lt salphingectomy	R	Rt	ovary	200	fresh oozing from Rt ovary	nil	nil	AB	7	ectopic
43	amutha	27	10352	17.02.13	G2P1L1	02.01.13	46	P	NA	NS	NA	NA	nil	nil	AM,LAP	110/70	86	8.8	ND	nil	nil	N	ND	EU,Lt &Rt A. mass,FL	ND	ectopic	4hrs 30 min	late diagnosis	NA	LSO	R	Lt	ovary	800	Rt ovary cystic	2 WB	nil	PR	8	ectopic
44	muppidathy	32	13342	02.03.13	G3P2L2	26.01.13	35	P	NA	NS	NA	NA	nil	MTP pills	AM,LAP,BPV	100/60	92	8.2	ND	D,T	FT	P	P	EU,Rt A. mass, FL	ND	ectopic	20 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	100	nil	nil	nil	AB	7	ectopic
45	amutha	25	14319	07.03.13	G2P1L1	19.01.13	57	P	NA	NS	NA	NA	nil	nil	AM,LAAP,BPV	100/60	88	9.6	ND	D,T	FT	P	ND	EU,Rt A. mass, FL	ND	ectopic	20 min	NA	NA	RSO	R	Rt	Ampulla	300	nil	1 WB	nil	PR	8	ectopic
46	stella	26	14677	09.03.13	G6P2L2A3	24.01.13	44	P	NA	S	4Y	TAT	NA	abortions, tubal sx	AM,LAP	120/80	84	13	ND	T	FT	P	ND	EU,Lt A. mass,FL	ND	ectopic	1 hr	NA	NA	B/L total salphingectomy	tubal abortion	Lt	fimbria	50	nil	nil	nil	PR	7	ectopic
47	mariammal	27	15413	12.03.13	G2P1L1	21.01.13	50	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP	80/50	96	7.2	ND	D	FT,FF	P	P	EU,FL	ND	ectopic	20 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	1300	nil	3 WB	1day	PR	8	ectopic
48	lakshmi	27	15689	14.03.13	G4P2L2A1	06.02.13	36	P	NA	S	7Y	LS	NA	abortion,tubal sugery	LAP	110/70	76	12	ND	T	FT,FF	P	ND	EU,Lt A.mass,FL	ND	ectopic	40 min	NA	NA	B/L total salphingectomy	R	Lt	Ampulla	500	nil	1 WB	nil	PR	7	ectopic
49	chandra	29	17312	21.03.13	p4m1	16.01.13	64	P	10y	NA	NA	NA	nil	ART	AM,LAP,BPV	80/50	108	8.2	ND	D,T	FF	P	P	EU,FL	ND	ectopic	30 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	1500	nil	2 WB,1PCV	2 days	PR	9	ectopic
50	usha	28	17787	24.03.13	G3A2	NK	NK	P	8y	NA	NA	NA	nil	abortions	LAP,BPV	110/60	102	8.6	ND	D,T	FF	P	P	EU,FL	ND	ectopic	20 min	NA	NA	Lt totals alphingectomy	R	Lt	isthmus	1500	nil	3 WB	2 days	PR	8	ectopic

Sl.No	Name	Age(in years)	I.P.No	D.O.A	Obstetric Code	LMP	Period of Amenorrhoea (in Days)	UPT	Duration of Subfertility	Sterilisation Status	Time Since ST (Years)	Method of ST	Other Contra Ceptive Use	Risk Factors	Clinical Symptoms	B.P(in mmHg)	P.Rt(per minute)	Hb %	Sr.β HCG(MIU/ml)	P/A Findings	P/V Findings	Cervix Excitation Test	Coldecentesis	Ultrasound Findings	Additional Imaging	Clinical Diagnosis	Time interval Between Admission and Start of definitive Treatment	Time Delay Due to	Medical Management Criteria	Management	Registered (R) / Unregistered (UR)	Side	Site	Haemoperitoneum (in ml)	Other Findings	No.Of Units of Blood / Blood Products Transfusion	ICU Care	Fever	Duration of Hospital Stay (in Days)	Final Diagnosis/HPE report
51	saravanapriya	27	18653	28.03.13	p#mi	12.02.13	44	P	nill	NA	NA	NA	nill	nill	AM,LAP,BPV	110/80	86	8.8	ND	D,T	FT	P	P	Bicorn.ut., FL	ND	ectopic	21 hrs	late diagnosis	NA	Lt partial salphingectomy	tubal abortion	Lt	Ampulla	150	arcuate uterus	1 WB	nil	AB	7	ectopic
52	pattathy	35	21384	09.04.13	G3P2L2	NK	NK	P	NA	NS	NA	NA	nil	nill	LAP,BPV	110/70	106	7.6	ND	D	nill	N	P	EU,Lt A. mass,FL	ND	ectopic	7 hrs	s x side admn	NA	Lt total salphingectomy	R	Lt	isthmus	800	nil	2 WB	nil	PR	13	ectopic
53	muhabath nisha	22	22994	15.04.13	G2P1L1	NK	NK	N	NA	NS	NA	NA	nil	MTP pills	LAP	120/70	76	7.8	ND	T	FT	P	ND	EU,Rt A. mass of 4.5 cm	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	UR	Rt	infundib	nil	nil	nil	nil	PR	10	ectopic
54	vasuki	28	24648	22.04.13	G3P2L2	03.03.13	50	P	NA	S	5Y	con	NA	prev LSCS,tubal surgery	AM,LAP	120/70	80	12	2010	nil	FT	P	ND	EU,Rt A. mass of 3.2 cm	ND	ectopic	2hrs	NA	A	RSO,Lt salphingectomy	UR	Rt	Ampulla	nil	adhesions	nil	nil	AB	7	ectopic
55	muthu kumari	31	24737	22.04.13	G5P2L2A2	NK	NK	P	NA	S	1Y	con	NA	abortion, LSCS,tubal sx	LAP	90/60	88	11	ND	D	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	2hrs	NA	NA	RSO,Lt salphingectomy	R	Rt	fimbria	300	adhesions	1 WB	nil	AB	7	ectopic
56	usharani	28	24916	23.04.13	G3P2L2	14.03.13	40	P	NA	S	3Y	PS	NA	tubal surgery	AM,LAP	120/80	86	11	ND	T	FT	P	ND	EU,Lt A. mass,FL	ND	ectopic	20 min	NA	NA	LSO,Rt salphingectomy	R	Lt	Ampulla	500	adhesions	1 WB	nil	PR	8	ectopic
57	muthuselvi	22	26913	02.05.13	G2P1L1	05.03.13	58	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP	110/80	88	9.8	ND	T	FT	P	ND	EU,Rt A.mass,FL	ND	ectopic	1 hr 1.5 min	NA	NA	Rt total salphingectomy	R	Rt	isthmus	550	nil	1 WB	nil	AB	7	ectopic
58	vasanthi	30	30231	16.05.13	G3P1L1A1	NK	NK	P	NA	NS	NA	NA	nil	prev ectopic, prev LSCS	LAP	110/70	84	10	ND	T	FT,FF	P	P	EU,Lt A. mass,FL	ND	ectopic	30min	NA	NA	Lt total salphingectomy	R	Lt	Ampulla	500	Rt tube absent	1 WB	nil	PR	8	ectopic
59	muthulakshmi	31	30380	17.05.13	G2P1L1	13.04.12	34	P	NA	NS	NA	NA	nil	prev LSCS	LAP,BPV	100/70	82	9	2126	T	FT	P	P	EU,Lt A. mass,FL	ND	ectopic	40 min	NA	NA	LSO	R	Lt	Ampulla	400	adhesions	1 WB	nil	PR	8	ectopic
60	Elizabeth	23	32383	26.05.12	G2P1L1	07.04.13	49	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP	120/80	78	11.7	ND	nil	FF	P	N	EU,FL	ND	ectopic	1 hr	NA	NA	Lt total salphingectomy	tubal abortion	Lt	fimbria	100	nil	nil	nil	PR	8	ectopic
61	subbulakshmi	23	33904	28.05.13	p#mi	28.03.13	61	P	6m	NA	NA	NA	nil	PID	AM,LAP	90/60	114	7.7	ND	D,T	FF,FT	P	P	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	RSO	R	Rt	fimbria	1500	ovary was adherent	3 WB	2days	PR	9	ectopic
62	shanthi	34	36434	14.06.13	G5P3L3A1	05.05.13	40	P	NA	S	3Y	TAT	NA	abortion,LSCS,tubal sx	AM,LAP	120/70	108	8.1	ND	T	FT	P	P	EU,Lt A. mass,FL	ND	ectopic	30 min	NA	NA	RSO,Lt salphingectomy	R	Rt	ovary	300	e/o ST on both sides	2 WB	nil	PR	8	corpus luteal cyst
63	selvi	28	37925	21.06.13	p#mi	01.05.13	51	P	6y	NA	NA	NA	COC	OCP	AM,LAP,BPV	130/70	92	6.8	ND	T	FT,FF	P	P	EU,Lt A. mass,FL	ND	ectopic	35 min	NA	NA	Lt total salphingectomy	R	Lt	fimbria	800	nil	2 WB	nil	AB	8	ectopic
64	subbulakshmi	29	38381	24.06.13	G4P1L1A2	14.05.13	41	P	NA	NS	NA	NA	IUCD	abortions	AM,LAP,vomit	80/40	>120,F	5.9	ND	D,T	FT	P	P	EU,FL	ND	ectopic	20 min	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	1900	nil	4 WB,4FFP	2 days	PR	10	ectopic
65	subbulakshmi	22	38771	26.06.13	p#mi	11.05.13	46	P	2y	NA	NA	NA	nil	nill	AM,LAP	110/80	86	10	ND	T	FT,FF	P	P	EU,Rt A. mass,FL	ND	ectopic	25 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	250	nil	1 WB	nil	PR	8	ectopic
66	selvi	37	39089	27.06.12	G3P2L2	NK	NK	P	NA	NS	NA	NA	nil	nill	LAP	130/80	78	11	5500	T	FT	P	ND	EU,Rt A.mass of 2.5cm	ND	ectopic	1 hr 30 min	NA	NA	Rt partial salphingectomy	UR	Rt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
67	thangamari	32	40629	04.07.13	G3P2L2	25.05.13	40	P	NA	S	6Y	con	NA	prev LSCS,tubal sx	AM,LAP,BPV	90/60	98	8	ND	D,T	FT,FF	P	ND	EU,Rt A. mass,FL	ND	ectopic	15 min	NA	NA	B/L total salphingectomy	R	Rt	Ampulla	1200	E/O ST on Lt side	2 WB	1 day	PR	9	ectopic
68	latha	25	42080	11.07.13	G2P1L1	NK	NK	P	NA	NS	NA	NA	nil	nill	LAP,BPV	110/70	96	7.4	ND	T	FT	P	ND	EU,Rt A.mass,FL	ND	ectopic	4 hrs	late diagnosis	NA	Rt comual resection	R	Rt	cornua	2000	nil	3 WB,4FFP	2 days	AB	8	ectopic
69	Vijaya	26	44210	22.07.13	G2P1L1	02.06.13	50	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	100/70	96	8.8	ND	D,T	FF	P	P	EU,Rt A. mass,FL	ND	ectopic	30 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	700	nil	1 WB	nil	AB	8	ectopic
70	jeyabharathi	28	45074	26.07.13	G2A1	27.05.13	60	P	3y	NA	NA	NA	nil	prev ectopic	AM,LAP	120/70	92	10.2	ND	T	FT	P	ND	EU,FL	ND	ectopic	45 min	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	400	Rt ovary,tube-absent	1 WB	nil	PR	8	ectopic
71	boopathy	39	45652	29.07.13	G4P3L3	29.05.13	61	P	NA	NS	NA	NA	IUCD	IUCD	AM,LAP,BPV	120/70	82	10	ND	T	FT	P	P	EU,Rt A. mass,FL	ND	ectopic	30 min	NA	NA	hysterectomy	R	Rt	cornua	1500	nil	5 WB,4FFP,2PLTS	2 days	PR	10	ectopic
72	lakshmi	34	46151	01.08.13	G4P2L2A1	NK	NK	P	NA	NS	NA	NA	nil	abortion	LAP,giddi	130/60	100	6.4	ND	D,T	nill	P	P	EU,FL	ND	ectopic	16 hrs	s x side admn	NA	B/L total salphingectomy	R	Lt	isthmus	1300	Rt fimbrial cyst	3 WB	1day	PR	8	ectopic
73	ratchiyar	30	47757	09.08.13	G3P2L2	21.06.13	49	P	NA	NS	NA	NA	IUCD	IUCD	AM,LAP	120/70	104	7.5	ND	D,T	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	30 min	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	1150	nil	2 WB	1 day	PR	8	ectopic
74	Jesintha	32	47634	08.08.13	P2L2	22.06.13	47	N	NA	S	8Y	con	NA	prev LSCS,tubal sx	AM,LAP	120/60	96	6.9	ND	T	FT	P	P	EU,Rt A. mass,FL	ND	ectopic	1 hr	NA	NA	RSO	R	Rt	ovary	150	adhesions,Rt ovarian cyst	1 WB	nil	PR	8	ovarian cyst
75	syedalil fathima	34	47798	10.08.13	G3P2L2	01.07.13	40	P	NA	S	10Y	con	NA	prev LSCS,tubal sx	AM,LAP	100/70	120	10	ND	D,T	FF	P	ND	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	B/L total salphingectomy	R	Lt	fimbria	1350	Rt ovary adherent	2 WB	1day	PR	8	ectopic
76	bakkiyalakshmi	27	49126	16.08.13	p#mi	25.06.13	52	P	6y	NA	NA	NA	nil	ovulation induction	AM,LAP	80/50	120	6.9	ND	D,T	FT	P	P	EU,Rt A. mass,FL	ND	ectopic	15 min	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	1600	nil	3 WB,3FFP	2 days	PR	8	ectopic
77	Rani	30	49284	17.08.13	G4P3L3	20.06.13	58	P	NA	NS	NA	NA	IUCD	IUCD	AM,LAP	110/70	78	12	1156	nil	nil	P	ND	EU,Rt A. mass of 3 cm	ND	ectopic	6 hrs	hCG report	A	Medical, MTX- multi dose	UR	Rt	NA	NA	NA	nil	nil	AB	14	ectopic
78	valli	31	51516	28.08.13	G2P1L1	10.07.13	49	P	NA	NS	NA	NA	POP	prev LSCS,POP	AM,LAP,BPV	80/50	124	8	ND	D,T	FF	P	ND	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	R	Rt	cornua	1000	Rt corpus luteal cyst	4 WB	1 day	PR	9	ectopic
79	seevalamuthu	40	51514	28.08.13	G3P2L2	NK	NK	P	NA	S	6Y	PS	NA	PID,tubal surgery	LAP,BPV	120/70	96	7.6	ND	D,T	FF	P	ND	EU,Lt A. mass,FL	ND	ectopic	1 hr	NA	NA	LSO,Rt salphingectomy	chr.R	Lt	fimbria	500	adhesins	1 WB	nil	PR	8	ectopic
80	mariammal	40	52172	01.09.13	G3P1L1A1	11.07.13	52	P	NA	NS	NA	NA	nil	abortion,LSCS,PID	AM,LAP,BPV	110/60	80	8.7	ND	D,T	FT	P	ND	EU,Lt A.mass,FL	ND	ectopic	30 min	NA	NA	LSO	R	Lt	cornua	1100	ovary was adherent	2 WB	1 day	PR	8	ectopic
81	mani	30	54509	14.09.13	G2P1L1	05.07.13	71	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	110/80	88	10.3	ND	T	FT	P	ND	EU,Rt A. mass of 4.5 cm	ND	ectopic	1 hr 30 min	NA	NA	Rt total salphingectomy	UR	Rt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
82	kanagavalli	30	55832	20.09.13	G3P2L2	NK	NK	P	NA	NS	NA	NA	nil	nil	LAP,diwr	90/70	>120	4.5	ND	D,T	FT	P	P	EU,Lt A. mass,FL	ND	ectopic	15 min	NA	NA	Lt cornual resection	R	Lt	cornua	1500	nil	4 WB,2FFP	2 days	PR	9	ectopic
83	mariselvi	32	53957	11.09.13	G2P1L1	27.07.13	46	P	NA	NS	NA	NA	nil	nill	AM,LAP	100/60	80	10.4	ND	nil	nill	P	ND	Bicorn. ut with G.sac	MRI-heterotopic	heterotopic	9 days	late diagnosis	NA	Lt total salphingectomy,MVA	R	Lt	Ampulla	1100	uterus enlarged to 8 wks	4 WB	1day	PR	18	heterotopic
84	Subbammal	38	57179	27.09.13	p#mi	30.07.13	59	P	10y	NA	NA	NA	nil	MTP pills	AM,LAP	110/60	>120	6.9	ND	D,T	FT,FF	P	P	IUP,5wks,FL	ND	ectopic	1hr 1.5 min	NA	NA	Rt total salphingectomy	R	Rt	cornua	2300	nil	4 WB,2FFP	2 days	PR	9	ectopic
85	maheswari	26	58390	04.10.13	p#mi	NK	NK	WP	nill	NA	NA	NA	nil	nill	LAP,BPV	120/80	106	10.6	4702	D,T	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	40 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	300	nil	1 WB	nil	AB	7	ectopic
86	usha	28	58671	05.10.13	G3P2L2	08.09.13	27	P	NA	S	1Y	con	NA	prev LSCS,tubal sx	LAP,BPV	100/70	76	11	ND	nill	nill	N	ND	EU,Rt A. mass of 5.7cm	ND	ectopic	1 hr	NA	NA	RSO,Lt salphingectomy	R	Rt	fimbria	50	adhesions	nil	nil	AB	7	ectopic
87	bharathy	29	60183	13.10.13	G2P1L1	23.08.13	51	WP	NA	NS	NA	NA	nil	nill	AM,LAP,giddi	90/60	126	5.7	ND	D	FT,FF	P	ND	EU,FL	ND	ectopic	1 hr	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	1350	nil	3 WB	2 days	PR	9	ectopic
88	Rani	32	60298	14.10.13	p#mi	19.08.13	66	P	9 months	NA	NA	NA	nil	PID	AM,LAP	100/70	78	10.6	ND	T	FT	P	ND	EU,Lt A. mass,FL	ND	ectopic	2 hrs	NA	NA	Lt partial salphingectomy	R	Lt	infundib	250	nil	nil	nil	AB	7	ectopic
89	Ramajeyam	32	61132	18.10.13	G3P2L2	02.09.13	46	P	9m	S	10Y	con	NA	prev LSCS,tubal sx	AM	90/60	72	12.7	ND	nil	nill	N	ND	EU,Rt tubal ring, viable 7wks	ND	ectopic	1 hr 10 min	NA	NA	B/L total salphingectomy	tubal abortion	Rt	Ampulla	100	nil	nil	nil	AB	7	ectopic
90	Ramalakshmi	20	60883	17.10.13	p#mi	05.09.13	42	P	6m	NA	NA	NA	nil	nill	AM,LAP,BPV	110/70	102	6.5	ND	D,G	FT	P	ND	EU,FL	ND	ectopic	25 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	900	nil	2 WB	1 day	AB	7	ectopic
91	shoba	32	62414	24.10.13	G2A1	31.08.13	54	P	5y	NA	NA	NA	nil	prev ectopic,PID,ART	AM,LAP	110/70	84	10.8	ND	G	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	6 hrs	late diagnosis	NA	Rt partial salphingectomy	R	Rt	Ampulla	500	Lt tube absent	1 PCV	nil	AB	7	ectopic
92	chermakani	26	62387	24.10.13	G2P1L1	30.08.13	55	P	NA	NS	NA	NA	IUCD	IUCD	AM	120/60	88	9.6	2356	nil	nil	N	ND	EU,Rt A.sac ,live 6wks fetus	ND	ectopic	1 hr	NA	A	LSO	tubal abortion	Lt	Ampulla	50	oozing from left ovary	nil	nil	AB	7	ectopic
93	Jasper pristilla	26	63081	28.10.13	G3P2L2	05.09.13	53	P	NA	NS	NA	NA	nil	prev LSCS,MTP pills	AM,LAP,BPV	90/60	106	5.9	ND	T	FT	P	ND	EU,FL	ND	ectopic	1 hr 30 min	NA	NA	B/L total salphingectomy	R	Lt	isthmus	900	nil	2 WB	nil	AB	7	ectopic
94	Meenatchi	27	63421	30.10.13	G3P1L1A1	24.09.13	36	WP	NA	NS	NA	NA	B	abortion	LAP,BPV	110/70	100	8.5	ND	T	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	30 min	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	200	endometriosis	1 WB	nil	AB	8	ectopic
95	Sudalai vadivu	27	63577	31.10.13	G4P2L2A1	15.09.13	46	P	NA	NS	NA	NA	nil	abortion,PID	AM,LAP,BPV	120/80	92	10	ND	T	FF	P	ND	EU,FL	ND	ectopic	35 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	850	nil	2 WB	nil	AB	8	ectopic
96	selvi	25	65019	07.11.13	p#mi	18.09.13	50	P	2.5y	NA	NA	NA	nil	PID	AM,LAP,BPV	120/80	78	12	39,738	T	nil	N	ND	EU,Rt ovary- 1cm follicle	ND	ectopic	7 days	late diagnosis	NA	RSO	UR	Rt	ovary	100	nil	nil	nil	AB	7	ectopic
97	maharasi	25	67969	21.11.13	G2P1L1	05.10.13	47	P	NA	NS	NA	NA	LAM	prev LSCS	LAP,BPV	120/70	82	11	ND	T	FT	N	ND	EU,Rt ovary - multi follicles	ND	ectopic	4 hrs	late diagnosis	NA	Rt total salphingectomy	R	Rt	isthmus	100	adhesions	nil	nil	AB	7	ectopic
98	amutha	32	67976	21.11.13	G4P2L2A1	12.10.13	40	WP	NA	NS	NA	NA	IUCD	abortion,IUCD	AM,LAP,BPV	130/70	80	11.8	440	nil	nil	N	ND	EU,small Rt A. mass	ND	ectopic	2 days	late diagnosis	A	Medical, MTX- multi dose	UR	Rt	NA	NA	NA	nil	nil	AB	12	ectopic
99	subha ozanna	29	69161	27.11.13	G3P2L2	29.10.13	29	N	NA	NS	NA	NA	IUCD	nill	LAP	140/70	102	9.3	ND	nil	nil	N	ND	IUCD in situ,Rt A. mass,FL	ND	ectopic	30 min	NA	NA	RSO	R	Rt	ovary	1100	nil	2 WB	nil	AB	8	corpus luteal cyst
100	krishna rani	28	69969	01.12.13	G2P1L1	09.10.13	53	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	100/60	86	11.6	ND	nil	nil	N	ND	EU,Rt A. mass,FL	ND	ectopic	12 hrs	late diagnosis	NA	RSO	R	Rt	Ampulla	200	Rt tube &ovary adherent	1 WB	nil	AB	8	ectopic
101	lakshmi	28	70029	01.12.13	G3P2L2	20.10.13	42	P	NA	NS	NA	NA	nil	prev LSCS,MTP pills	AM,LAP,giddi	80/60	126	8.8	ND	D,T	FT	P	P	EU,FL	ND	ectopic	30 min	NA	NA	B/L total salphingectomy	R	Lt	isthmus	2100	Rt fimbrial cyst	4 WB,4FFP,4PLTS	2 days	PR	8	ectopic
102	selvi	35	71782	10.12.13	G3P2L2	10.11.13	30	P	NA	NS	11Y	LS	NA	tubal surgery	LAP	100/60	82	11.5	ND	nil	FF	N	ND	EU,Lt A. cyst,FL	CT-pelvic cyst,8 cm	ectopic	24 hrs	late diagnosis	NA	RSO & LSO	R	Rt	fimbria	200	adhesions,Lt ovarian cyst	1 WB	nil	AB	8	ectopic
103	pathirakali	26	71637	09.12.13	G4P2L2A1	05.10.13	65	P	NA	NA	S	1.5Y	con	NA	abortion,LSCS,tubal sx	AM,LAP,BPV	110/70	90	11.5	ND	nil	nil	N	ND	EU,Lt A. mass,FL	ND	ectopic	40 min	NA	NA	RSO,Lt salphingectomy	R	Rt	fimbria	200	E/O ST on Lt side	nil	nil	AB	8	ectopic
104	kulanthai	24	73310	17.12.13	p#mi	18.10.13	60	P	2y	NA	NA	NA	nil	ovulation induction	AM,LAP	80/40	>120,F	3.1	ND	D,G	FF	P	ND	EU,Lt A. mass,FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	R	Rt	cornua	2900	Lt corpus luteal cyst	5 WB,3FFP	3 days	AB	10	ectopic

Sl.No	Name	Age(in years)	I.P.No	D.O.A	Obstetric Code	LMP	Period of Amenorrhoea (in Days)	UPT	Duration of Subfertility	Sterilisation Status	Time Since ST (Years)	Method of ST	Other Contra Ceptive Use	Risk Factors	Clinical Symptoms	B.P(in mmHg)	P.Rt(per minute)	Hb %	Sr.β HCG(M IU/ml)	P/A Findings	P/V Findings	Cervix Excitation Test	Coldecentesis	Ultrasound Findings	Additional Imaging	Clinical Diagnosis	Time interval Between Admission and Time of definitive Treatment	Time Delay Due to	Medical Management Criteria	Management	Registered (R) / Unregistered (UR)	Side	Site	Haemoperitoneum (in ml)	Other Findings	No.Of Units of Blood / Blood Products Transfusion	ICU Care	Fever	Duration of Hospital Stay (in Days)	Final Diagnosis/HPE report
105	kannika	30	73966	21.12.13	G2P1L1	NK	NK	P	NA	NS	NA	NA	LAM	IMTP pills	LAP,BPV	110/70	98	10	ND	T	FT	P	P	EU,Rt A. mass,FL	ND	ectopic	40min	NA	NA	RSO,Lt salphingectomy	R	Rt	Ampulla	300	Rt tube &ovary adherent	1 WB	nil	PR	7	ectopic
106	muthuselvi	35	74441	23.12.13	G4P3L3	NK	NK	WP	NA	S	14Y	LS	NA	PID,tubal surgery	LAP,const,vomit	90/70	80	11	508	nil	FF	N	ND	EU,Rt A.mass,FL	ND	ectopic	21 hrs	late diagnosis	NA	B/L total salphingectomy	R	Rt	isthmus	700	E/O ST on Lt ,ovary cystic	1 WB	nil	AB	7	ectopic
107	Jeyalakshmi	35	74440	23.12.13	p/imi	24.10.13	60	P	8y	NA	NA	NA	nil	ART	AM,LAP,BPV	100/70	88	9.8	ND	D,T	FT	P	ND	EU,Rt A.mass,FL	ND	ectopic	45 min	NA	NA	Rt partial salphingectomy	R	Rt	isthmus	1700	uterus &bowel adherent	3 WB	1 day	PR	9	ectopic
108	muppidathy	39	75756	31.12.13	G7P6L2	15.11.12	46	WP	NA	S	10Y	PS	NA	tubal surgery	AM,LAP	120/80	72	11	ND	D,T	FT	P	ND	EU,Lt A.mass,FL	ND	ectopic	45 min	NA	NA	LSO,Rt salphingectomy	R	Lt	ovary	250	E/O recanalisation on Lt	1WB	nil	AB	8	corpus luteal cyst
109	subbulakshmi	33	1307	08.01.14	G2A1	22.11.13	47	P	14y	NA	NA	NA	nil	Ovul.induction,recan.sx	AM,LAP,BPV	110/70	82	9.4	ND	D,T	FT	P	ND	EU,Lt A. ring,FL	ND	ectopic	1 hr 20 min	NA	NA	LSO	R	Lt	Ampulla	550	bicorn uterus,adhesions	1 WB	nil	AB	8	ectopic
110	muthulakshmi	21	75	01.01.14	p/imi	18.11.13	44	P	11m	NA	NA	NA	nil	nil	AM,LAP,BPV	120/70	76	14	872	nil	nil	N	ND	EU,Lt A. mass of 3.2 cm	doppler-Lt A.cyst,no flow	ectopic	3 days	late diagnosis	A	Medical, MTX- multi dose	UR	Lt	NA	NA	NA	nil	nil	AB	14	ectopic
111	murugeshwari	23	4485	25.01.14	G3P1L1A1	17.11.13	69	P	NA	NS	NA	NA	nil	abortion,LSCS,PID	AM,LAP,BPV	130/80	78	9.5	386	nil	nil	N	ND	EU,Lt A. mass, local fluid	ND	ectopic	48 hrs	late diagnosis	NA	Lt partial salphingectomy	tubal abortion	Lt	Ampulla	75	Lt corpus luteal cyst	nil	nil	AB	7	ectopic
112	selvi	23	4867	28.01.14	G3P2L2	NK	NK	P	NA	S	3Y	con	NA	prev LSCS,tubal sx	LAP	100/60	92	9.2	ND	T	nil	N	ND	EU,Rt A. mass,FL	ND	ectopic	35 min	NA	NA	RSO,Lt salphingectomy	R	Rt	cornua	1500	e/o ST on Lt side	2 WB	1day	AB	8	ectopic
113	sathya	26	6120	03.02.14	G2P1L1	02.12.13	63	P	NA	NS	NA	NA	nil	prev LSCS	AM,BPV,anuria	110/70	116	9.2	ND	nil	nil	N	ND	EU,,FL	ND	ectopic	40 min	NA	NA	LSO	R	Lt	cornua	1600	Adhesions	2 PCV	1 day	AB	8	ectopic
114	chandra	26	6473	05.02.14	G3P2L2	NK	NK	P	NA	S	7Y	PS	NA	tubal surgery,PID	LAP,BPV	110/70	120	12	ND	D,T	nil	P	ND	EU,Rt A. mass,FL	ND	ectopic	1 hr	NA	NA	RSO,Lt salphingectomy	R	Rt	Ampulla	650	e/o ST on Lt ,Rt ovary friable	2 WB	nil	PR	8	ectopic
115	padma	29	6719	06.02.14	p/imi	19.12.13	49	P	7m	NA	NA	NA	nil	PID	AM,LAP,BPV	110/70	84	9.4	ND	D,T	FT	P	ND	EU,Lt A. G.sac of 1.6 cm,no FP	ND	ectopic	4hrs 30 min	hCG report	A	Medical, MTX- multi dose	UR	Rt	NA	NA	NA	nil	nil	AB	15	ectopic
116	parvathy	25	7197	09.02.14	G2P1L1	17.11.13	84	P	NA	NS	NA	NA	nil	prev LSCS	AM,BPV	100/60	92	4	ND	nil	nil	N	ND	EU,Rt A. mass,FL	ND	ectopic	38 hrs	late diagnosis	NA	removal of Rt horn ectopic	R	Rt	Rt horn	1000	bicornuate uterus	2WB,2PCV	2 days	AB	9	ectopic
117	eskkiammal	30	8410	15.02.14	p/imi	24.12.13	53	P	4m	NA	NA	NA	nil	nil	AM,LAP,BPV	110/80	112	7.6	ND	D,T	FT	P	P	EU,Lt A. mass,FL	ND	ectopic	20 min	NA	NA	Lt partial salphingectomy	R	Lt	isthmus	300	nil	2 WB	nil	AB	8	ectopic
118	Ramalakshmi	29	13990	14.03.14	p/imi	05.02.14	37	P	6m	NA	NA	NA	nil	nil	AM,LAP	110/80	86	11	ND	nil	nil	N	ND	EU,Rt A. G.sac,FP 7 wks,FH +	ND	ectopic	1 hr	NA	NA	Rt partial salphingectomy	UR	Rt	Ampulla	nil	nil	nil	nil	AB	7	ectopic
119	shanthi	29	14199	16.03.14	G3P1L1A1	03.02.14	41	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP	90/60	88	10.5	ND	D	FF	P	ND	EU,Rt A. mass,FL	ND	ectopic	45 min	NA	NA	RSO	R	Rt	infundib	900	Rt tube &ovary adherent	2 WB	nil	AB	8	ectopic
120	sugirtha	30	15916	24.03.14	p/imi	21.01.14	62	P	10y	NA	NA	NA	nil	ovulation induction	AM,LAP	100/70	92	8.8	ND	D,G	FT,FF	P	ND	EU,Rt A.mass,FL	ND	ectopic	20 min	NA	NA	Rt total salphingectomy	R	Rt	fimbria	1450	active bleed from fimbria	3 WB	1 day	AB	8	ectopic
121	kerpagavalli	25	16591	28.03.14	G2A1	05.02.14	51	P	1y	NA	NA	NA	nil	abortion	AM,LAP	130/90	100	10.2	ND	T	FT,FF	P	ND	EU,,FL	ND	ectopic	1 hr	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	450	nil	1 WB	nil	AB	7	ectopic
122	lakshmi	25	17284	31.03.14	G3A2	NK	NK	P	2.5y	NA	NA	NA	nil	abortions	LAP,anasarca	80/40	140,F	3.1	ND	D,G	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	20 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	2000	Rt ovary cystic	4 WB,4FFP	3 days,V	PR	10	ectopic
123	muniammal	32	19677	13.04.14	G2P1L1	28.02.14	44	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,oliguria	80/60	124	6.3	ND	D,T	FT	P	ND	EU,,FL	ND	ectopic	3 hrs	sx side admn	NA	Lt partial salphingectomy	R	Lt	Ampulla	1550	nil	4 WB	2 days	PR	9	ectopic
124	Rama prabha	33	22006	25.04.14	G6P1L1A4	NK	NK	P	NA	NS	NA	NA	nil	abortions,LSCS	LAP	150/90	82	10.9	ND	T	FT,FF	P	ND	EU,Lt A.mass,FL	ND	ectopic	18 hrs	late diagnosis	NA	LSO	R	Lt	fimbria	550	Lt tube & ovary adherent	1WB,1 PCV	nil	AB	8	ectopic
125	selvi	30	19723	13.04.14	G4A3	03.03.14	41	P	10y	NA	NA	NA	nil	abortions ,prev ectopic	AM,LAP,BPV	100/60	80	11	ND	T	FT	P	ND	EU,Rt A. mass,FL	ND	ectopic	50 min	NA	NA	Rt partial salphingectomy	R	Rt	Ampulla	250	Lt -only proximal tube +	1 WB	nil	AB	7	ectopic
126	sudha	35	21300	21.04.14	p/imi	23.02.14	57	P	2y	NA	NA	NA	nil	nil	AM,LAP,giddi	100/70	92	8.1	ND	D,G	FT	P	ND	EU,,FL	ND	ectopic	40 min	NA	NA	Rt partial salphingectomy	R	Rt	isthmus	1600	nil	3 WB	1 day	PR	9	ectopic
127	sukirtha	27	23663	03.05.14	G2P1L1	22.03.14	42	WP	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,vomit	110/70	88	12	1126	nil	nil	N	ND	EU,Rt A. sac 1.3 cm,Rt ovary cyst	ND	ectopic	12 hrs	late diagnosis	A	Rt total salphingectomy,Rt ovarian cystectomy	UR	Rt	cornua	nil	comp.Rt ov. cyst of 6x4cm	nil	nil	AB	8	ectopic
128	viji	27	24257	06.05.14	p/imi	04.03.14	63	P	3y	NA	NA	NA	nil	ovulation induction	AM,LAP,Gidd	100/70	102	11	46193	nil	nil	N	P	EU,,FL	MRI-Rt cornual ectopic	ectopic	45 min	NA	NA	Rt total salphingectomy	R	Rt	cornua	2500	active bleed at rupture site	3 WB	2 days	PR	9	ectopic
129	thirumalaiselvi	31	26157	15.05.14	G3P2L1	27.03.14	49	P	NA	NS	NA	NA	nil	prev LSCS,PID	AM,LAP,BPV	110/70	80	8.9	ND	nil	nil	N	P	EU,,FL	ND	ectopic	3 hrs	late diagnosis	NA	Lt partial salphingectomy	tubal abortion	Lt	Ampulla	50	nil	nil	nil	AB	7	ectopic
130	jeyalakshmi	26	26805	19.05.14	p/imi	25.03.14	55	WP	1.5y	NA	NA	NA	nil	nil	AM,BPV	120/80	90	12	956	nil	nil	N	ND	EU,Rt A.sac of 1cm, FP + ,no FH	ND	ectopic	1 day	late diagnosis	A	Medical, MTX- multi dose	UR	Rt	NA	NA	NA	nil	nil	AB	15	ectopic
131	babitha	24	30213	03.06.14	G3A2	20.04.14	44	P	2y	NA	NA	NA	nil	abortion,ovul.induction	AM,LAP,BPV	110/70	120	7.4	ND	D,T	FF	P	ND	EU,,FL	ND	ectopic	30 min	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	800	nil	2 WB	nil	AB	8	ectopic
132	thangaselvi	25	30204	03.06.14	G3P2L2	NK	NK	P	NA	S	4Y	PS	NA	tubal surgery	LAP,giddi	110/70	100	10	ND	nil	FT	P	P	EU,Rt A. mass,FL	ND	ectopic	7 hrs	IMCU admn	NA	RSO,Lt salphingectomy	R	Rt	ovary	1400	e/o ST on both sides	2 WB	1 day	PR	8	ectopic
133	nalvelkumari	24	32919	16.06.14	G2P1L1	11.05.14	36	P	NA	NS	NA	NA	nil	prev LSCS	LAP,vomit	110/70	122	11	ND	D	FT,FF	P	P	EU,Rt A. G.sac , FP 7 wks,FH +	ND	ectopic	18 hrs	sx side admn	NA	Rt total salphingectomy	R	Rt	fimbria	800	nil	1 WB	nil	AB	8	ectopic
134	balammal	34	32933	16.06.14	G5P2L2A2	01.05.14	46	P	NA	S	5Y	con	NA	abortion,LSCS,tubal sx,PID	AM,LAP	90/60	108	9	ND	D,G	nil	P	P	EU,,FL	ND	ectopic	30 min	NA	NA	B/L total salphingectomy	R	Rt	fimbria	900	e/o ST ,fresh bleeing	2WB,1PCV	1 day	PR	8	ectopic
135	Iyyammal	31	32374	14.06.14	G3P1L1A1	09.05.14	36	WP	NA	NS	NA	NA	nil	abortion ,LSCS	LAP	120/70	92	10	1784	nil	FF	N	ND	normal study	doppler-ring of fire	ectopic	5 days	doubt	A	Negative laparotomy, D&C	NA	NA	NA	nil	nil	nil	nil	AB	12	PUL
136	Shanthi	33	33713	20.06.14	G2A1	26.04.14	55	P	4y	NA	NA	NA	nil	abortion	AM,LAP,BPV	140/90	80	12	ND	nil	FT,FF	P	ND	bicorn.ut,no G.sac	ND	ectopic	1hr 45 min	NA	NA	Lt partial salphingectomy	R	Lt	Ampulla	250	nil	1 WB	nil	AB	7	ectopic
137	padma	24	33817	20.06.14	G3P2L2	01.05.14	50	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,giddi	100/60	98	9.7	ND	nil	nil	P	ND	EU,Rt A. mass,FL	ND	ectopic	2 hrs	NA	NA	Rt total salphingectomy	R	Rt	Ampulla	750	nil	2 WB	nil	AB	8	ectopic
138	selvarani	32	33390	18.06.14	G2P1L1	14.04.14	65	P	NA	NS	NA	NA	nil	prev LSCS	AM,LAP,BPV	80/40	130,F	6.5	ND	D	FT	P	P	EU,,FL	ND	ectopic	20 min	NA	NA	LSO	R	Lt	isthmus	2000	nil	3 WB	2 days	PR	9	ectopic

KEY TO MASTERCHART

D.O.A	-	Date of admission
I.P.NO	-	In patient number
LMP	-	Last menstrual period
NK	-	Not known
ND	-	Not done
UPT	-	Urine pregnancy test
P	-	Positive
N	-	Negative
WP	-	Weakly positive
Y	-	Year
m	-	month
hr	-	hour
min	-	minute
S	-	Sterilised
NS	-	Not Sterilised
A	-	Applicable
NA	-	Not applicable
F	-	Feeble
PR	-	Present
AB	-	Absent
Rt	-	Right
Lt	-	Left

R	-	Ruptured
UR	-	Unruptured
ST	-	Sterilisation
LS	-	Laparoscopic sterilisation
PS	-	Puerperal sterilisation
TAT	-	Total abdominal tubectomy
Con	-	Concurrent sterilisation
IUCD	-	Intrauterine contraceptive device
EC	-	Emergency contraception
COC	-	Combined oral contraceptive
POP	-	Progesterone only contraceptive
B	-	Barrier method
LAM	-	Lactational amenorrhoea
MTP	-	Medical termination of pregnancy
PID	-	Pelvic inflammatory disease
ART	-	Artificial reproductive technique
Sx	-	Surgery
LSCS	-	Lower segment caesarean section
AM	-	Amenorrhoea
LAP	-	Lower abdominal pain
BPV	-	Bleeding per vaginum
D	-	Abdominal Distension
T	-	Abdominal tenderness
FF	-	Forniceal fullness

FT	-	Forniceal tenderness
A.mass	-	Adnexal mass
EU	-	Empty uterus
FL	-	Free fluid
G.sac	-	Gestational sac
Bicorn.ut	-	Bicornuate uterus
FP	-	Fetal pole
FH	-	Fetal heart
RSO	-	Right salphingo oophorectomy
LSO	-	Left salphingo oophorectomy
hCG	-	human chorionic gonadotropin
Infundib	-	Infundibulum
WB	-	Whole blood
PCV	-	Packed cell volume
PLTS	-	Platelets
FFP	-	Fresh frozen plasma
V	-	Ventilatory support