EFFICACY OF VARIOUS MEDICAL MANAGEMENT OF OVARIAN ENDOMETRIOMA - A RETROSPECTIVE OBSERVATIONAL STUDY

A Dissertation submitted in partial fulfilment of the requirements of the Tamil Nadu Dr. MGR Medical University for the degree of MS (Obstetrics & Gynaecology) examination to be held in May 2022

Registration number - 221916407

CERTIFICATE

This is to certify that the dissertation titled “EFFICACY OF VARIOUS MEDICAL MANAGEMENT OF OVARIAN ENDOMETRIOMA- A RETROSPECTIVE OBSERVATIONAL STUDY” is the original research work done by Dr.Hana Christy.T towards partial fulfilment of the requirements of the Tamil Nadu Dr M.G.R Medical University for the degree of MS (Obstetrics and Gynaecology) examination to be held in May 2022.

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DECLARATION CERTIFICATE

I hereby declare that this dissertation titled "EFFICACY OF VARIOUS MEDICAL MANAGEMENT OF OVARIAN ENDOMETRIOMA- A RETROSPECTIVE OBSERVATIONAL STUDY" was carried out by me under the guidance and supervision of Dr Vaibhav Londhe, Professor of Obstetrics and Gynaecology Unit II, Christian Medical College, Vellore.

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Efficacy of various medical management of ovarian endometrioma- A retrospective observational study.
Dr. Hana Christy.T, PG Registrar, Employment Number:29850, Obstetric & Gynaecology unit II office, Dr. Vaibhav Londhe, Employment Number:28279, OG2 Office, Dr.Emily Divya Ebenezer, Obstetrics & Gynaecology Unit 2.

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Dear Dr. Hana Christy.T,

I enclose the following documents:-

1. Institutional Review Board approval 2. Agreement

Could you please sign the agreement and send it to Dr. Suceena Alexander, Addl. Vice Principal (Research), so that the grant money can be released.

With best wishes,

Dr. Suceena Alexander
Secretary (Ethics Committee)
Institutional Review Board

Dr. Suceena Alexander, MD.,DM.,FASN.
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Ref: IRB Min. No. 13634 [OBSERVE] dated 02.12.2020.

Dear Dr. Hana Christy.T,

The Institutional Review Board **(Blue**, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Efficacy of various medical management of ovarian endometrioma- A retrospective observational study" on December 02, 2020.

The Committee reviewed the following documents:

1) IRB Application Format
2) Permission Letter
3) GCP Certificate
4) Cvs. Of Drs. Emily, Hana, Vaibhav.
5) No. of Documents 1 – 5.

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on December 02, 2020 in the New IRB Room, Christian Medical College, Vellore 632 004.

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Dr. Shyam Kumar NK	DMRD, DNB, FRCR, FRANZCR	Professor, Radiology, CMC, Vellore	Internal, Clinician
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Dr. Premila Abraham	M.Sc. Ph.D	Professor, Department of Biochemistry, CMC, Vellore	Internal Clinician

We approve the project to be conducted as presented.

Kindly provide the total number of patients enrolled in your study and the total number of Withdrawals for the study entitled: "Efficacy of various medical management of ovarian endometrioma- A retrospective observational study" on a monthly basis. Please send copies of this to the Research Office (research@cmcvellore.ac.in).

The Institutional Ethics Committee expects to be informed about the progress of the project, Any **adverse events** occurring in the course of the project, any **amendments in the protocol and the patient information / informed consent**. On completion of the study you are expected to submit a copy of the **final report**. Respective forms can be downloaded from the following link: http://172.16.11.136/Research/IRB_Polices.html in the CMC Intranet and in the CMC website link address: http://www.cmch-vellore.edu/static/research/Index.html.

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Fluid Grant Allocation:

A sum of 8,000/- INR (Rupees Eight thousand Only) will be granted for 24 months.

Yours sincerely,

Dr. Suceena Alexander
Secretary (Ethics Committee)
Institutional Review Board

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ABSTRACT

Title: Efficacy of various medical management of ovarian endometrioma- A retrospective observational study.

Methods: Ours was a retrospective observational study to look for the efficacy of medical management and the need for change in management -medical or surgical, in terms of regression of size of ovarian endometrioma. Charts of 300 patients who consulted CMC Vellore Gynecology OPD from 2017 to 2019 and on medical management for ovarian endometrioma for at least 6 months were reviewed.

This study was done as a chart review, on women in reproductive age group of 18 to 45years. These women were on a medical management for endometrioma for at least 6 months during the years 2017 to 2019. Most frequently used medical managements in our hospital for treatment of endometrioma were extended cycle OCP's or progestins like Injection DMPA, Dienogest and Mirena. Following which recruitment of patients according to the inclusion and exclusion criteria was done. The total women selected in the study were 300. Through online charts of patients, the change in size of endometriomas and menstrual regulation were collected and analyzed. Size of endometrioma after the new treatment was noted following repeat ultrasound scan. The details were obtained from the

online chart retrospectively. The efficacy of various medical managements on the endometrioma were compared from the time of starting medical management to after the change in management using Excel sheet.

Results: The efficacy of medical management in terms of decrease in size of endometrioma was slightly higher in extended cycle OCP group on comparison to inj. DMPA, dienogest and Mirena but statistically not proven. Thus, for a woman with endometrioma it is advisable to start with extended cycle OCP as first line management. Knowledge about breakthrough bleeding pattern has to be imparted to patients as it is commonest bleeding pattern noticed in patients on extended cycle OCPs.

Conclusion: Extended cycle OCP has slightly higher efficacy in management of endometriosis but not proven statistically. There is need for change in medical and surgical management in patients with endometrioma mainly due to persisting symptoms of dysmenorrhea, non-cyclical abdominal pain, heavy menstrual blood loss and dyspareunia.

TABLE OF CONTENTS:

TITLE

Efficacy of various medical management of ovarian endometrioma- A retrospective observational study.

INTRODUCTION

Endometriosis occurs commonly in reproductive age women. It is a chronic benign condition which can present with mild symptoms and can progress to severe chronic pelvic pain [1]. There is multiple hypothesis but still there is no clarity on the definite pathogenesis of endometriosis. Major symptoms related to endometriosis are dysmenorrhea, dyspareunia, and chronic pelvic pain. Infertility is one of the debilitating effects of endometriosis [1].

Endometriosis has high risk for invasion of areas like pelvic cavity, diaphragm and pleural cavity [2]. It can manifest as pelvic implants, ovarian endometriomas or invade surgical scar sites.

Treatment includes various medical and surgical managements. Management of endometriosis involves the following basic principles:

- Reduction of inflammation, thereby decreasing endometriosis related pain [2]
- Suppressing ovarian cycles and inhibiting the effect of oestrogen by either medical or surgical managements mostly decreases the growth and size of endometriosis and its implants [2]. Thereby there is relief from endometriosis related pain. However, this is only temporary, it should be noted once the suppression on estrogen is removed, there is noted return of the

endometriosis related symptoms of pain like dysmenorrhea, dyspareunia, and chronic pelvic pain.

- Use of medical management before surgery to get temporary pain relief of symptoms also decrease the size of endometriotic implants and endometriomas. Post-surgical use of continued suppression of estrogen by long term use of medical managements for 6 months to 1 year delays the recurrence of endometriotic implants and ovarian endometrioma. However studies show after laparoscopic surgical resection there is recurrence of 21.5% in 2 years and about 40 to 50% after 5 years after the surgery [2]. There have been few studies showing importance of usage of combined oral contraceptive pills after laparoscopic surgeries for suppression against recurrence of Endometrioma. However, there is need for studies to correlate efficacy of different modes of medical management of endometriosis, for better understanding in treating patients with endometriosis in the future[2].

In our study we are going to concentrate on the efficacy of different modes of medical management on treatment of ovarian endometriomas and their effect on menstrual cycles.

ABBREVIATIONS:

Inj.DMPA - Injection Depot medroxy progesterone acetate

OCP - Oral contraceptive pills

LNG-IUS - Levonorgestrel intra uterine system

MRI - Magnetic resonance imaging

USG - Ultrasound

NK cells - Natural killer cells

T cells - T lymphocytes

WNT4 - Wnt family member 4

GREB1 - Growth regulating estrogen receptor binding gene 1

SPE - Superficial peritoneal endometriosis

OMA - Ovarian endometrioma

DIE - Deep infiltrating endometriosis

AFC – Antral follicular count

IVF - Invitro fertilization

PID - Pelvic inflammatory disease

IBS - irritable bowel syndrome

STD - Sexually transmitted disease

rASRM- Revised American society of reproductive medicine

EFI - Endometriosis fertility index

AAGL - American association of gynecologist laparoscopists.

NSAIDs- Non steroidal anti-inflammatory drugs

COCs - Combined oral contraceptive pills

GnRH - Gonadotrophin releasing hormone.

AIMS & OBJECTIVES

AIMS:

To assess the efficacy of various types of medical management of ovarian endometrioma and the need for change in management-medical or surgical, among women of reproductive age group 18 to 45 years who were treated in CMC Vellore gynecology OPD for at least 6months.

OBJECTIVES:

Primary objective:

-To assess the efficacy of medical management in terms of regression of size of ovarian endometrioma retrospectively by viewing online OPD chart data from 2017 to 2019 over a period of 3years among women of reproductive age group 18 to 45years on medical management – extended cycle oral contraceptive pills or progestins for at least 6months in CMC Vellore gynaecology OPD.

Secondary objective:

-To assess the need for change in management -medical or surgical in regression of size of endometrioma retrospectively by viewing online OPD chart data from 2017 to 2019 over a period of 3years among women of reproductive age group 18 to 45years on medical management for at least 6months in CMC Vellore Obstetrics and gynecology OPD.

REVIEW OF LITERATURE

DEFINITION

Endometriosis is a condition in which there is presence of endometrial glands and stroma outside the normal uterine endometrium [3]. It usually affects women of reproductive age group. It is a chronic debilitating condition which progresses by inflammation, local invasion, and dissemination. It is usually diagnosed by ultrasound, MRI and by Laparoscopic findings. Laparoscopy is the gold standard for diagnosis of endometriosis [3]. Endometrioma is a pseudocyst of ovary, where endometriotic implants invaginate into the ovary. During each menstrual cycle, there is more inflammation, angiogenesis and growth of the endometriotic implant leading to Endometrioma [4]. The Menstrual implants progresses to form thick texture similar to chocolate, hence also called as chocolate cyst [4].

PREVALENCE

Endometriosis usually presents in adolescent and reproductive age woman [5].
It nearly affects 10% of the reproductive woman population around the world and around 30% to 70% of women known to have infertility [5]. In India a study done in 2016[5] by the O&G dept, Wayanad institute of medical sciences, Kerala prevalence of endometriosis was at a higher rate of 25% in reproductive age women and 73.33% in women with infertility. In a study [6] done by ashrafi et al on

673 women the prevalence of endometriosis in the total sample of women undergoing laparoscopy for various reasons for infertility treatment was 26.5%. Another study done on women with endometriosis there were about 17 to 44% of women developing ovarian endometrioma [4]. The true prevalence of endometriosis is confusing due to difference in risk factors distribution around the world [6].

RISK FACTORS

Endometriosis is known to affect woman differently depending on their risk factors [6]. Due to wider prevalence of endometriosis among infertile women, the need for identifying risk factors related to diagnosing early stage of disease for further evaluation and treatment is important [6]. In a study done by hemmings et al [7] prevalence of endometriosis among those who have experienced symptoms was from 31% to 55%. The maximum prevalence[7] was recorded for patients suffering from risk factors of infertility - 55%, pelvic mass - 47% and pelvic pain - 46%.

Below are some of the risk factors known to cause endometriosis in women [6]:

1. Symptoms of dysmenorrhea, dyspareunia and chronic pelvic pain
2. Increasing age
3. Low-income strata
4. Nulliparous

5. Underweight
6. Family history of endometriosis
7. Early menarche
8. Prolonged menstrual flow
9. Short cycle interval
10. Intercourse during menstrual cycle
11. Infertility.

According to a retrospective study done among 1282 woman in an infertility center in Iran in 2016[6], there is significant association for risk of endometriosis in woman with shorter menstrual cycle, low parity, dysmenorrhoea, dyspareunia, chonic pelvic pain, nulliparous, diarrhoea, constipation, family history of endometriosis, history of galactorrhoea and history of pelvic surgeries [6]. However duration of menstruation and early menarche age which were theoretically among risk factors for endometriosis, were not related to risk of endometriosis in this study. We found significant correlation between length of menstrual cycle and the presence of endometriosis, but other studies reported no significant association between length of cycle, length of menstruation, as well as age at menarche with the presence of endometriosis [7]. Some studies done shows inverse relationship to smoking and endometriosis, with significant mild benefits of smoking in women with endometriosis but few other studies find no significant relationship between smoking and endometriosis [7,8].

Recent evidences have all indicated that endometriosis is related with increased exposure to menstruation cycle like either shorter or longer cycles and heavy menstrual blood loss , which is clearly well along the lines of the the retrograde menstruation theory or sampsons theory [7].

PATHOPHYSIOLOGY

Endometriosis depends on estrogen hormone which leads to more inflammation and growth[1]. During each menstrual cycle there is cyclical shedding of the endometriotic tissue, similar to what happens in the normal endometrial tissues. It leads to more inflammation and symptoms like dysmenorrhea, dyspareunia and deep-seated pelvic pain mostly causing Infertility along with affecting quality of life[7]. There is also formation of endometriomas and deep-seated endometriosis, invading peritoneum and pelvic cavities[7]. They also form deposits on pelvic organs and rarely are found disseminated in distant organs and regions of body needing medical and sometimes surgical management once size increases leading to symptoms and signs of endometriosis[7].

Retrograde menstruation[1] is most common hypothesis used to explain migration of endometrial glands and stroma from uterus and getting implanted in peritoneal

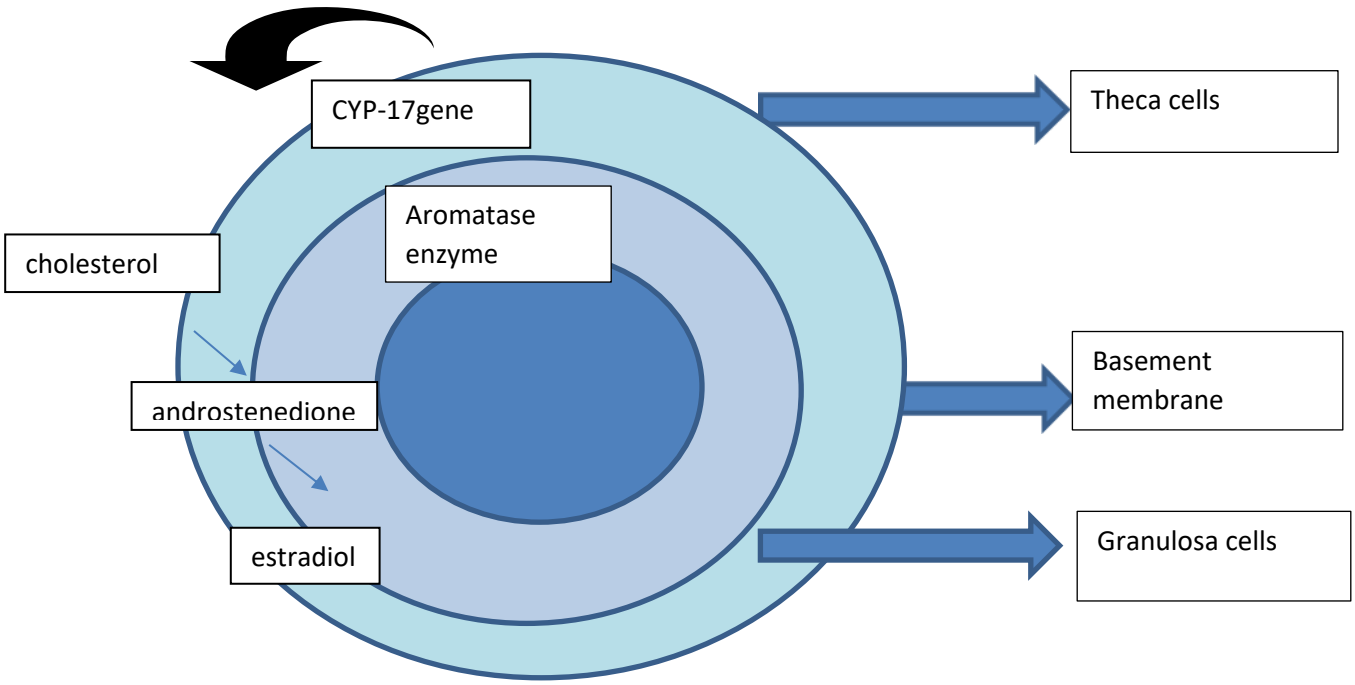
cavity and other areas, leading to formation of endometriotic implants or deposits and ovarian endometriomas. These lesions are hormonally active and respond to the cyclical changes in estrogen and progesterone, leading to the classical triad of endometriosis - dysmenorrhea, dyschezia and dyspareunia. As we said earlier estrogen is crucial for the growth and persistence of the endometriotic implants.

The main pathophysiology associated with Endometriosis consists of [1]:

1. Increased production of estradiol[1]
2. Increased intrinsic aromatase activity[1]
3. Increased production of inflammatory markers[1]
4. Progesterone resistance[1]

The mechanism of aromatase enzyme in production of estrogen is explained by two cell-two gonadotrophin theory [9]. It involves the 2 layers of the ovarian follicle namely theca cells and granulosa cells, theca cells have CYP-17 gene which codes for 17-hydroxylase enzyme which helps in conversion of cholesterol into androstenedione and testosterone, however it lacks aromatase enzyme [9]. However, the inner granulosa layer consists of aromatase enzyme but lacks CYP-17 gene. The granulosa cells depend on theca cells for androstenedione and use aromatase enzyme to convert the androstenedione and testosterone into estradiol, which is

necessary for endometriosis to thrive [9]. Aromatase inhibitors like letrozole have been known to stop this peripheral estrogen production.



OVARIAN FOLLICLE

2 cell- 2 gonodotrophin theory

PATHOGENESIS OF ENDOMETRIOSIS

Endometriosis is disease of chronic ailment. Its definite pathogenesis is still unknown and not all cases can be explained by simple theories of endometriosis [10]. The most commonly known theory is the retrograde menstruation theory also called as Sampson's theory, the shedding of menstrual endometrial tissue which are refluxed into the pelvic cavity, via the Fallopian tube, are deposited to form ectopic endometriotic lesions[10]. This theory directly explains the peritoneal lesions in endometriosis.

The most common site[1] of endometriosis by retrograde spread is the ovaries and then to anterior, posterior cul-de sac, broad ligament, fallopian tube, uterosacral ligaments, sigmoid colon, appendix, and round ligament
t. Other areas, which are less commonly involved[1], include the vagina, cervix, rectovaginal septum, cecum, ileum, inguinal canal, perineal scars, urinary bladder, ureter, and the umbilicus.

Rare cases of endometriosis of gastrointestinal tract, bones, vertebra, central nervous system, and lungs have been reported[1]. These extra peritoneal spread of endometriosis has been explained by lympho-vascular spread[10]. There have been studies on newer endometrial stem cell progenitors deposited by retrograde

menstruation and lymphovascular spread to extrauterine sites like abdominal wall, gastro intestinal tracts, bladder, cervix and vagina [10].

In addition to these theories, abnormal physiological processes like hormonal dysregulation, inflammatory markers and dysfunctional immune mediators all contribute to the pathogenesis of endometriosis [10]. Endometriosis-related pain[1] is attributed to the increase in inflammatory mediators, neurological dysfunction and estrogen-mediated neuro-modulation of the peripheral sensory neurons. Studies have found increased number of inflammatory cells like macrophages, proinflammatory cytokines like interleukin-1, 6, tumor necrosis factor and prostaglandins in the endometrial lesions[1]. There are the cause for chronic inflammation and symptoms of endometriosis related pain[1].

The following are the various theories of endometriosis formation and growth.

1. Implantation theory
2. Coelomic metaplasia theory
3. Induction theory
4. Venous and lymphatic dissemination
5. Immunological theory
6. Genetic factors
7. Molecular defects
8. Surgical entrapment

1. Implantation theory:

   Also known as Sampson's theory, refers to the retrograde menstruation causing reflux of the endometrial glands and stromal deposits from uterine endometrium to pelvic cavity[10]. This theory is supported by the fact that endometriosis is prevalent in women with obstructive Mullerian anomalies[11], short and heavy menstrual cycles and also due to the fact, the most dependent parts of pelvic cavity are affected such as ovary, cul-de-sac, uterosacral ligaments, rectovaginal septum and posterior region of uterine wall[11]. This theory is the most commonly used to explains endometriosis around the world. However certain conditions like MRKH syndrome (Mayer-Rokitansky Küster-Hauser's syndrome)[12,13]where there is absence of uterus

and in cases of men[13,14]under the influence of prolonged estrogen therapy this theory for endometriosis does not explain.

2. Coelomic metaplasia theory:

   Meyer postulated this theory. The serosa of all intra-abdominal organs are derived from coelomic epithelium[13]. Metaplasia of these coelomic epithelium into endometrium is then followed by development of endometriotic implants on peritoneal / different distant organs[13]. This theory explains endometriosis in the absence of menstruation[15]. This theory also explains the presence of endometriotic tissues in distant organs like lungs, spleen, liver, brain, gastro intestinal tracts, nail beds, pericardium and skin[13]. Endometriotic implants in lungs and brain can lead to morbid conditions with catamenial pneumothorax, hemoptysis, catamenial headache, seizures and subarachnoid bleed. [13]

3. Induction theory:

   This theory is derived from coelomic metaplasia theory[15]. During menstruation there is release of endogenous factors which induces the metaplasia of coelomic epithelium into endometrial tissue[15]. This theory also explains the distant micro metastasis of endometriotic tissue into organs like lungs, spleen, brain and skin[13].

4. Venous and lymphatic dissemination:

Dissemination by venous and lymphatic spread explains the widespread development of endometriosis in cervix, vagina, vulva, gastrointestinal tracts, lungs, pleural cavity, brain , lymph nodes and surgical scars[13].

5. Immunological theory:

This theory is explained by the presence of decreased cellular immunity against endometrial implants in women with endometriosis[15]. Change in immunity can be due to alteration of the cytokines and growth factors secreted by macrophages. There are changes in both cell mediated and humoral immunity[15]. The number of Macrophages in peritoneum are increased, however the numbers of T cell and NK(Natural killer cells) cells are decreased, these lead to remnants of the endometrial implants which causes further inflammation, increase in size of implants leading to adhesion and further symptoms of cyclical and non-cyclical pain for the women. [16]

The peritoneal fluid and the serum from endometriosis patient inhibits the activity of NK cells, mainly because they produce macrophages like TGF-Beta, IL-10 which makes the peritoneal environment unfavorable for NK cells[15]. Hence apoptosis of the endometriotic implants is not cleared compared to normal women, leading to increase in size of the endometriotic implants and with it there is increase in endometriosis symptoms of pain[15].

6. Genetic factors

Familial inheritance is noted in endometriosis[17]. Genetic factors represent around 51% of risk for endometriosis[17]. Patients with history of affected first-degree relative have risk of 7 to 10 times higher chance of developing endometriosis than others[17]. Earlier age of disease diagnosis and history of first degree relative predisposes to severe disease. There are studies showing polygenic inheritance[17,18]. Endometriosis that occurs in families tends to be more severe compared to sporadic cases[17]. This also suggests that there is more genetic predisposition in individuals with severe disease, and hence there is more likelihood to have affected siblings or offsprings[17]. Other factors which suggest a genetic predisposition to endometriosis include the similar age and earlier age of onset of disease[17].

Several studies have been done to identify the loci of genes that are at risk for encoding for endometriosis[17]. Some have been identified for example: WNT4, CDKN2B-AS1, and GREB1. WNT4 encodes for development of female reproductive tract and for gene products responsible for steroidogenesis[17]. The CDKN2B-AS1 encodes for gene that is responsible for tumor suppressor proteins such as p15, p16-NK4a, and p14ARF[17]. GREB1 encodes for an early-response gene, very important in the estrogen receptor regulated pathway[17]. There are no studies relating identification of genetic factors and prevention and treatment of endometriosis. Future studies

are needed to understand the effect of these gene expression on control of endometriosis to fully understand the disease of endometriosis.

7. Molecular defects:

   The endometriotic stromal and epithelial cells are associated with uniquely aberrant molecular pathways that were regulated by microRNAs[19]. In endometriotic stromal cells aberrant pathways interfere with immune cell production, induce inflammation and potential activation of epithelial-mesenchymal transformation to tumor cells[19]. Decreased NK cells lead to delayed apoptosis and hence increases risk for endometriosis[19].

8. Surgical entrapment:

   The reason for scar endometriosis can be explained by the fact, during surgeries like caesarean sections and hysterectomies, where peritoneum containing endometriotic implants get trapped between suture sites, leading to entrapment of endometrial implants[20]. Inability to exit the suture site causes entrapment of the endometrial tissue leading to increase in size of the endometrial implants, cyclical pain at the surgical scar site and formation of nodule/induration at the healed scar[21]. About 0.03% to 1.5% of caesarean sections and 0.01% to 0.06% of hysterectomy and episiotomy surgical sites have presented with scar endometriosis[21]. The treatment is usually complete

excision of the scar tissue or temporary suppression of pain by use of combined oral contraceptive pills[20].

PATHOLOGY

The endometriotic lesions are hormonally active and respond to the cyclical changes in estrogen and progesterone and may have a different appearance in various phases of the menstrual cycle[22]. They can be identified by gross examination of the specimen, direct laparoscopic evaluation of the cysts or the implants and by histological evaluation of the specimen[23].

However definitive diagnosis is made by histological examination of the glandular and stromal components[23]. The appearance of the glandular component can be altered by hormonal and metaplastic changes, as well as cytologic atypia and hyperplasia[23]. Hence definite knowledge of the pathological features of the endometriosis is important[23].

In a study done by Rizk et al in 2014 only 50% of laparoscopic biopsy specimens from areas suspicious for endometriosis were proven microscopically to be endometriosis[22]. The histologic diagnosis of endometriosis is usually made based on the below pathological features observed in endometriosis[23].

Pathological findings in endometriosis:

STROMAL COMPONENT	GLANDULAR COMPONENT	MISCELLANEOUS TUMORLIKE FINDINGS	INFLAMMATORY-REACTIVE CHANGES	RARE ASSOCIATED LESIONS
Foamy and pigmented histiocytes	Postmenopausal and treatment-related changes	Necrotic pseudoxanthomatous nodules	Infected endometriotic cysts	Peritoneal leiomyomatosis
Fibrosis	Pregnancy-related changes	Polypoid endometriosis	Pseudoxanthomatous salpingitis	Peritoneal gliomatosis
Elastosis	Metaplastic changes	Vascular invasion	Florid mesothelial hyperplasia	
Smooth muscle	Cytologic atypia	Perineural invasion	Liesegang rings	
Metaplasia	Hyperplasia		Multilocular peritoneal inclusion cysts (MPIC's)	
Myxoid change	Absence of glands (stromal endometriosis)			
Decidual change, including signet-ring–like cells				

Other features noticed on examination of gross specimen in endometriosis[23]

- There can be variations from red, brown, black, white, yellow, pink, or clear vesicles.
- There could be degree of hemorrhages, fibrosis, and inflammation depending on the duration of the lesions.

CLASSIFICATION OF ENDOMETRIOSIS BASED ON MACROSCOPIC APPEARANCE OF PELVIC ENDOMETRIOSIS.

1. **Superficial or peritoneal endometriosis:**

   The most common among the three subtypes of endometriosis is superficial peritoneal endometriosis (SPE)[24]. It accounts for around 80% of the endometriosis[24]. SPE however do not present with similar features. Even the location, extent and symptoms of pain varies from each other[24]. They can also be found to co-exist along with the other subtypes of endometriosis and sometimes with adenomyosis[24].

   It is present on dependent areas of pelvic cavity[25]. It is found on laparoscopic evaluation usually and occurs on superficial surfaces of the peritoneum[25]. The commonest sites are ovaries surfaces, over anterior and posterior surfaces of uterus, pouch of Douglas, uterosacral ligaments and broad ligaments[25]. Pelvic nodes are involved in 30% women[25].

   Gross appearance of lesion depends on early lesions looking red, hemorrhagic compared with old lesions looking brown or black, later on discoloration disappears and appears white[25].

   Clinical examination cannot be performed, only laparoscopic examination confirms the diagnosis[25]. Superficial endometriosis can cause cyclical lower abdominal pain which can progress to chronic pelvic pain if size of the

implant increases and causes more inflammation and adhesions[25]. There symptoms are similar to pelvic inflammatory diseases[25]. In an article published in 2017 on Superficial Peritoneal Endometriosis Fernando et al stated that the major complaints of women affected with superficial or peritoneal endometriosis is primary infertility, moderate to severe dysmenorrhea and deep dyspareunia[25]. There have been no theories yet on the natural progression of SPE, whether it regresses spontaneously or will worsen to different forms of subtypes of endometriosis[24]. Current guidelines for treatment are either excision by surgical removal, cautery method or suppression of the SPE medically[24].

2. **Ovarian endometrioma:**

Ovarian endometrioma (OMA) is a benign and most common gynecological disease with an incidence of 10% to 20% in women of reproductive age group[2]. It is most prevalent in women around the age of 40 to 44 years, and usually the incidence tends to fade after menopause[2]. OMA consists between 17% to 44% of patients with endometriosis[2]. Following are various theories for pathogenesis of endometrioma formation.

The pathogenesis of endometrioma is still not definite, many theories have been put forward, below are few of the common theories:

a) Inversion and invagination of ovarian cortex [26]:

This theory was first described by Hughesdon in 1957, he suggested that endometrial implants, located on the surface of the ovary are the reason for endometriomas. Hughesdon's theory suggests that, menstrual shedding and the endometrial implant bleeding are trapped which leads to a gradual invagination of the ovarian cortex, resulting in a psuedocyst[27]. Brosens et al.[28], also agreed with the above theory and reported the presence of menstrual shedding and blood accumulation at the site of the implants by ovariscopy. Hence this theory is the backbone for treatment of ovarian endometriomas.

b) Secondary involvement of functional ovarian cyst by endometriotic implants located on ovarian surface[26].
c) Metaplasia of coelomic epithelium on the ovary[26].
d) Primordial cells have also role in forming endometriomas[26].

Endometriomas are more usually located in the left hemipelvis and left ovary. There is a left lateral predisposition of endometrioma, could be the result of the presence of sigmoid colon in the left side of pelvis, preventing endometrial cell recycling in the pelvis.[29]

3. **Deep infiltrating endometriosis:**

Deep infiltrating endometriosis (DIE) is the most aggressive form, which affects 20% of women who suffer from endometriosis[30]. It can be defined as the presence of endometriotic lesions over 5 mm in depth under the peritoneal surface; others define it as a pathologic entity, which is called adenomyosis externa[30].

DIE is also defined as the infiltration of fibrous and muscular tissue in organs and structures affected by endometriosis, including endometrial tissue, with no reference to the extent of lesion depth underneath the peritoneum Deep infiltrating endometriosis[30].

Pathogenesis of DIE is explained mainly by metaplasia of coelomic epithelium and primordial cell theories[30]. Since only 10% women with endometriosis end up having DIE even though the rest 90% are having retrograde menstruation, this Sampson's theory of retrograde menstruation does not hold good in this case of DIE[30].

It is the most aggressive of the three sub types of endometrioses. It can affect the pelvis as a whole, causing change to the normal anatomy of pelvis and its pelvic organs. This leads to great impact on the women's quality of life[30].

Medical therapy can control the symptoms and stop the progress of the diseased[31]. But recurrence happens once medication is stopped once pain relief is achieved[2]. Hence patient should be aware of the recurrence and side effects of the medications used[2].Surgical treatment should be proposed

only when it is either failed hormonal therapy, side effects of medication used, severity of symptoms or as treatment for infertility[30].

CLINICAL FEATURES OF ENDOMETRIOSIS

Depending on the severity of endometriosis and site of the endometrial implants the clinical symptoms can vary[32]. Usually superficial peritoneal endometriosis is asymptomatic[32]. The symptomatic patients usually signify severity of the disease[32]. It takes around 7 years for an asymptomatic implant to become symptomatic implant[32]. The classical triad of endometriosis are dysmenorrhea, dyspareunia and chronic pelvic pain[32]. It also presents with low back pain, dyschezia, dysuria, or infertility[32]. Of these symptoms, chronic pelvic pain is the most common chief complaint among women who come for medical care[32]. The Pelvic pain usually increases in severity premenstrually and subsides after menses ends, but it can also occur without menstruation. The main cause for chronic pelvic pain is the involvement of peripheral spinal nerves. There is no involvement of autonomic nervous system[32].

The endometriotic lesions have enriched nerve roots which is the cause for the endometriosis related pain. In deep infiltrating lesions the nerve fiber density is

higher, especially DIE of gastro intestinal system are very densely innervated hence there incidence is diagnosed earlier due to early onset of symptoms of pain[32]. The intensity of the pain is usually found to be related to depth of the endometriotic implants. However, some studies have proved that there is no co-relation between the depth of penetration of the endometriotic lesion and the severity of endometriosis related pain.

In endometriosis symptoms are often vague and generalized. Hence can be misdiagnosed as irritable bowel syndrome, interstitial cystitis/painful bladder syndrome or even for recurrent cystitis-overactive bladder thus leading to inadequate treatment and diagnostic delay[32]. The interval between the onset of the first symptoms to the diagnosis of endometriosis may be of approximately 7–10 years[32].

The clinical manifestations of endometriosis varies and cannot be predicted about its course. Below are few of the most common clinical features of endometriosis[32]:

1. Dysmenorrhea
2. Dyspareunia
3. Chronic pelvic pain
4. Dyschezia
5. Uterosacral ligament nodularity

6. Adnexal masses
7. Infertility
8. Hematuria and dysuria
9. Perimenstrual tenesmus and loose stools
10. Heavy menstrual blood loss.

Below are the correlations of site and corresponding symptoms of endometriosis[32]:

1)	Bladder:	dysuria, gross hematuria during menses, urgency, tenesmus, suprapubic discomfort, and urinary incontinence.
2)	Ureters:	dysmenorrhea, dyspareunia, urinary symptoms, hydronephrosis and decline of renal function
3)	Round ligaments:	painful, palpable inguinal mass, nonspecific pelvic pain (intra-pelvic portion)
4)	Uterosacral ligaments:	severe pain and dyspareunia

5)	Vagina	dysmenorrhea, dyspareunia, postcoital spotting, prolonged menstruation not responding to medical therapy leading to anemia
6)	Rectosigmoid colon	cyclic pain during defecation, dyschezia, cyclic, bloating, constipation, bowel cramping, catamenial diarrhea, pencil-like stools, bowel obstruction
7)	Thoracic- diaphragmatic endometriosis	chest pain with right-sided predominance, scapular or cervical pain associated with menses, sometimes radiating to the arm, pneumothorax, dyspnea, hemoptysis.
8)	Sciatic nerve	cyclic sciatica, back pain, gluteal pain radiating to the dorsal thigh and lateral lower leg, sensory loss, reflex alterations, muscle weakness and paresis.

PROBLEMS RELATED TO ENDOMETRIOSIS

Endometriosis Associated Pain

The pelvis is highly vascularized and enervated. There is also multiple other factors, contributes to the pain syndrome that is associated with endometriosis[33].

1. Peritoneal fluid in women with endometriosis contains high levels of nerve growth factors that promote neurogenesis[33].
2. Nerve density within endometriotic nodules is increased[33].
3. Cytokines and prostaglandins produced by mast cells and other inflammatory cells are attracted to ectopic endometrial implants, leading to more pain in the region[33].
4. Nerve fiber entrapment within endometriotic implants leads to conditions like cyclical sciatic pain, weakness, and sensory loss can all stem from endometriotic entrapment of the sciatic, femoral, or lumbosacral nerve roots[33].
5. Central sensitization: Patients become highly sensitive to subsequent painful stimuli because of endometriosis-induced neuroplastic changes in descending pathways that modulate pain perception[33].

PROBLEMS RELATED TO ENDOMETRIOMA

The endometrioma negatively impacts the antral follicle count(AFC) on the affected side in comparison to the non-affected side. The endometrioma significantly lowers AFC[26].

The presence of an endometrioma lowers number of oocytes retrieved[4] during IVF.

There is also a risk of malignant transformation in endometriomas (0.7%). Clear cell and endometrioid carcinoma are the most commonest cancer that is found in endometrioma[26].

DIAGNOSIS

Definite diagnosis of endometriosis is a very important for early diagnosis and treatment of endometriosis. There are so many symptomatic similarities to endometriosis and other medical conditions affecting pelvis that they are overlooked and end up being misdiagnosed or delay the diagnosis process. Hence need of the hour is for definite diagnosis of endometriosis. The following are the steps in early diagnosis of endometriosis.

1. Clinical history:

   Preliminary diagnosis of endometriosis starts with clinical history. Since most women are asymptomatic in the early stages of the disease, by indulging in the symptom characteristics, pattern of menstrual cycle and menstrual related pain, an idea of the size and severity of the disease is obtained.

   Diagnosis of endometriosis based on only clinical presentation is difficult because of its wide range of symptoms and significant overlap with other gynaecological and other medical conditions, such as chronic pelvic inflammatory disease (PID) and IBS[34]. Proper diagnosis of endometriosis will assist in excluding other differential diagnosis such as polycystic ovary syndrome, fibroid tumours, and interstitial cystitis[34].

History taking involves comprehensive review of systems[34]:

a. Menstrual and reproductive history:
    i. Age at menarche/menopause
    ii. Cycle lengths, menstrual flow, premenstrual and other menstrual related pain
b. Sexual history:
    1. Contraception methods used
    2. Pain during intercourse(dyspareunia)/defecation(dyschezia)/or micturition(dysuria)
    3. Prior history of STD's
    4. Abnormal vaginal bleeding or discharge.
c. Obstetric history:
    i. Number of pregnancies, miscarriages & abortions.
d. Family history of similar problems
e. Social history:
    i. Nutrition, exercise, and stress.
f. Past treatment history:
    i. History of taking oral contraceptive pills (Non contraceptive use)

ii. Progestins

iii. NSAID's for pain relief.

g. Past surgical history:

i. History of surgery for endometrioma/diagnostic laparoscopy/any other abdominal surgeries.

History taking should be in such a way that it decreases the time delay in diagnosis of endometriosis.

1. Examination:

The next important step is examination of the patient, keeping the symptoms of the patient in mind, particular areas are looked for their involvement[33]. For example palpation for uterine or adnexal tenderness, retroverted uterus with restrictive mobility, nodules in uterosacral ligament, and for any pelvic masses[33]. A tenderness on palpation of posterior fornix is the most common finding, indicating presence of endometriotic nodule involving the uterosacral ligaments[33]. Single digit per vaginal examination followed by bimanual per vaginal examination and then by per rectal examination covers the pelvic examination[33].

2. Diagnostic tests:

Other causes of pelvic pain should also be evaluated like urinalysis, pap smear, pregnancy test, vaginal and endocervical swabs[33]. Women with endometriosis show altered levels of CA-125, cytokines, angiogenic and growth factors compared to normal women, but none of the markers have been statistically proven to be definitive clinical tool for diagnosis of endometriosis.

3. Diagnostic imaging:

Imaging is done prior to staging of endometriosis and prior to planning surgical treatment of pain relief[35]. Usually there is involvement of different adjacent organs due to growing endometriomas which causes compression of adjacent pelvic structures and increase in endometriosis related chronic pain requiring surgical treatment[35]. Endometrioma has a characteristic ground-glass appearance on ultrasound[35]. It is known that endometriomas vary widely in their sonographic appearance- homogeneous, hypoechoic, thick-walled round mass, highly suggestive of endometrioma[35].

Pelvic ultrasound scans are used to facilitate diagnosis of site, size and type ovarian cysts and endometrioma. Pelvic masses are visualized by the use of transvaginal and transabdominal ultrasound[35]. Transvaginal ultrasound is used to better visualize endometrium and uterine cavity and detect ovarian endometriotic cysts but does not rule out peritoneal endometriosis, endometriosis-associated adhesions and deep infiltrating endometriosis[35]. Occasionally, a magnetic resonance imaging and computed tomography scans

are conducted to characterize the pelvic masses[35]. In case of extra pelvic endometriosis like in gastro intestinal tract barium meal studies done to see for irregularities in the tract walls denoting endometrial implants[35].

4. Diagnostic surgery and histology evaluation:

   Recent guidelines recommends that the histological examination of specimens collected from the suspicious areas during the visual inspection of the pelvis at diagnostic laparoscopy is the gold standard for diagnosis of endometriosis[35]. Laparoscopy may not be appropriate for all women with a history and physical examination suggestive of endometriosis[35]. Therefore, proper care has to be taken to identify the patients who require diagnostic laparoscopy.

   On the other hand, endometriomas may have other sonographic appearances including echoic or heterogeneous echoes, internal septation, and fluid-fluid level[35]. Diagnosing endometrioma before surgery, has a good clinical implication especially in patients for whom surgery is contraindicated or for prescribing preoperative medications to them to decrease the size of the endometrioma[35].

CLASSIFICATIONS OF ENDOMETRIOSIS

The clinical manifestations of endometriosis are diverse, and the relationship between symptoms and disease severity are ambiguous. The ideal classification system should be able to explain the extent of disease, predict pain and fertility, provide accurate information to patients, and reflect the anatomical features[36].

The four standard classification systems, widely known and used are:

1. Revised American Society for Reproductive Medicine (rASRM) classification,
2. ENZIAN classification,
3. Endometriosis fertility index (EFI),
4. American Association of Gynecological Laparoscopists (AAGL) classification.

1. **Revised ASRM classification of endometriosis**

   This classification was proposed in 1979, the stage of endometriosis depends on a cumulative score[36]. It took into consideration size of the endometriotic lesions in the ovaries, peritoneum, and fallopian tubes, and the severity of adhesions[36].

The staging system was divided into four stages[36]:

Stage		points
I	Mild	1 to 5
II	Moderate	6 to 15
III	Severe	16 to 30
IV	Extensive	31 to 54

However, there was lack of a relationship between the disease stage and the clinical symptoms of pain and infertility.

Therefore, revised AFS (1985)/ American society for Reproductive Medicine(rASRM) (1996) defined the stages of endometriosis as[36]

Stage		Points
I	Minimal	1 to 5
II	Mild	6 to 15
III	Moderate	16 to 40
IV	Severe	>40

Tubal endometriosis was omitted from the revised classification.[36]Lesions of endometriosis were classified as superficial and deep lesions. The size of deep ovarian endometriosis > 3 cm scored 20-point, Dense ovarian adhesion and dense tubal blockage scored 16 points. Single finding of complete cul-de-sac obliteration scored 40 points.

Advantages:

a. It's easy to use
b. Easy to interpret

Disadvantages:

a. Difference between histologically diagnosed endometriosis and visually diagnosed stage.
b. There is inter-observer difference hence reproducibility of the rASRM score is poor
c. Severities of pain and infertility are not correlated with rASRM stage.
d. rASRM classification does not consider the presence of deeply infiltrating endometriosis (DIE) in different sites such as the uterosacral ligaments, bladder, vagina, and bowel[36].

2. ENZIAN Classification:

This classification was introduced in Austria in 2005[36]. The ENZIAN score, like the rASRM classification, is determined by the extent of endometriosis during surgery. It was used to supplement rASRM classification with addition of its description of DIE. However, two revisions of the ENZIAN classification system were carried out in 2010 and 2011 to correct the overlap between rASRM and ENZIAN systems and to make it easy for use. The revised ENZIAN classification was simplified by dividing retroperitoneal structures into three compartments.

Compartment	
A	Rectovaginal septum and vagina
B	Uterosacral ligaments and pelvic walls.
C	Sigmoid colon and rectum.

Severity	Invasiveness
Grade 1	<1cm
Grade 2	1 to 3cm
Grade 3	>3cm

Nomenclature in classification[36]:

a. The prefix “E” indicates the presence of a tumour of endometriosis
b. The number that follows the prefix indicates the size of the lesion
c. the lowercase English letter that follows the letter indicates the affected compartment.
d. Two lowercase English letters mean bilateral disease.
e. The invasion of endometriosis to other organs in the pelvic cavity and to distant organs is expressed as follows:

Terminology	Site of invasion
FA	Adenomyosis
FB	Bladder
FU	Intrinsic ureter
FO	Other locations
FI	Intestine

2. **Endometriosis fertility index**:

The goal of EFI is to predict the pregnancy rate in patients with surgically documented endometriosis who have not yet conceived by IVF[36]. In 2010, Adamson and Pasta proposed an EFI system. This system reflects on age, duration of infertility, and previous pregnancies. For pregnancy to occur proper functioning of the fallopian tube, fimbria, and ovary is required. The functional score is used to denote the embryo is well implanted into the uterus, whether the uterus can withstand normal pregnancy[36].

The least function score is calculated by evaluating the function of the ovary, fallopian tube, and fimbria for each side, and adding the lowest score on the left and the lowest score on the right[36].

Functional scores	
0	Non-functional
1	Severe dysfunction
2	Moderate dysfunction
3	Mild dysfunction
4	Normal

The EFI score is calculated by summing the historical and surgical scores, and ranges from 0 to 10 points, with 10 indicating the best prognosis and 0 the worst prognosis[36].

Advantages[36]:

a. Superior in predicting pregnancy outcome.
b. More reliable system to predict IVF outcomes in endometriosis patients than the rASRM classification.

Diadvantages[36]:

a. Does not correlate with pain.
b. The least function score is judged subjectively and varies with interobserver.
c. More complicated to use than the rASRM classification and ENZIAN score.

4. **American Association of Gynaecological Laparoscopists classification**:

In 2007, the AAGL initiated a project to develop a new classification of endometriosis. This score predicts the fertility outcome for women who attempt non-in vitro fertilization conception, following surgically documented endometriosis extent of disease in a patient[36].

Surgical difficulties	
Level 1	Excision or desiccation of superficial implants and simple thin avascular adhesions
Level 2	stripping of ovarian endometriomas; appendectomy; deep endometriosis not involving the vagina, bladder (not requiring sutures), bowel, or ureter; dense adhesions not involving the bowel and/or the ureter
Level 3	dense adhesions involving the bowel and/or ureter; bladder surgery requiring sutures; ureterolysis; bowel surgery without resection (shaving)
Level 4	bowel resection with end-to-end anastomosis; ureteral reimplantation or anastomosis.

For validation of the score system, visual analogue scale scores and infertility history were collected from the patients before surgery[36]. In 2012, the AAGL Special Interest Group reported that the AAGL classification for endometriosis was shown to be related to pain, infertility, and surgical difficulty. However, the AAGL classification is yet to be validated and published, even though 10 years have passed[36].

MEDICAL MANAGEMENT OF ENDOMETRIOSIS

The management of endometriosis mainly involves treatment for cyclical pain, non-cyclical deep pelvic pains and infertility treatment[1]. Pain associated with endometriosis is associated with active secretion from the endometrial implant site, leading to irritation, inflammation, release of inflammatory cytokines like interleukin 1 and interleukin 6, increase in size of the implant, formation of endometrioma and adhesions in the peritoneum[1]. The other major debilitating condition as direct complication of endometriosis is infertility[1]. There is major loss of healthy ovarian tissue and ovarian reserve due to growing endometriotic implants and endometriomas. There is also significant irreparable loss of ovarian reserve during surgical management of endometriomas[1].

The main principles of medical management of endometriosis consists of the following[1]:

1. Suppression of intrinsic oestrogen production in the endometrial implant by aromatase enzyme.
2. Suppression of oestrogen from ovarian synthesis.
3. Increased production of inflammatory markers.
4. Decrease progesterone resistance.

The first line treatment for pain relief for minor complaints like dysmenorrhea is NSAIDs[1]. It decreases production of inflammatory markers leading to decrease in

inflammation and adhesion which gives pain relief for the patients with earlier stages of endometriosis[1].

However, the main stay management widely used are hormonal suppression of the endometriotic tissues by using combined oral contraceptive pills, progesterone only pills, gonadotropin releasing hormone agonists, aromatase inhibitors and androgens like danazol[1]. Although each method of medical management has their own advantages, there are also quite significant side effects for hormonal suppression[1].

The following are the various pharmacological options available for medical management of endometriosis[1]:

	Medical managements	Examples
1.	NSAIDs	Mefenamic acid, Ibuprofen and Diclofenac
2.	Combined oral contraceptive pills	Ovral-L, Ovral-G and Loette
3.	Progesterone containing contraceptives	Oral or injectables, implants and LNG IUS
4.	Selective progesterone receptor modulator	Mifepristone Ulipristal acetate Onapristone
4.	Gonadotrophin releasing hormone agonists	Leuprolide acetate Nafarelin Goserelin
5.	Gonadotrophin releasing hormone antagonists	Cetrorelix
6.	Aromatase inhibitors	Letrozole, Anastrozole
7.	Androgen	Danazol

NSAIDs:

Mostly commonly used for treatment of dysmenorrhea and other endometriosis related pain. Usually, the pain occurs mostly secondary to elevated levels of prostaglandins and inflammatory cytokines[1]. The mechanism of action of NSAIDs is by reversible blockade of COX-1 and COX-2 which is required in the pathway for production of prostaglandin E2[1]. Recent studies have shown a new drug rofecoxib, a selective COX-2 inhibitor NSAID which can also be used as effective first line treatment in early stages of endometriosis. It has been found inhibit the growth of endometrial tissue[1]. The maximum duration of treatment with NSAIDs was 3 to 4 months, following which continuing the treatment has no significant benefit as it indicates the progress of disease needing further medical management[1]. Common side effects noticed while using NSAIDs are gastrointestinal abdominal pain. However there has been no significant results regarding the effectiveness of NSAIDs.[1]

Combined oral contraceptives (COCs):

The management of endometriosis with COCs is suppression of ovaries and hence the suppression of endometriosis[1]. COCs are the most common hormonal therapy used as first line in treatment of endometriosis. Estrogen and progesterone combinations or progesterone only pills cause decidualization of the endometriotic

tissue and decreases the progress of endometriosis[1]. They have been used with varying degree of success in women with endometriosis[1]. It is of low cost, easily administered and available easily hence it has been used widely for treatment of endometriosis. In comparison to cyclical COCs, long term administration has more suppression of ovary and better pain relief [1]. However, there are also various risk factors along with long term administration of COCs like risk for thromboembolism, decreased ovarian reserve and decreased fertility due to effect of long-term ovarian suppression[1]. It also causes decreased bone density and increased risk for bone fractures[1].

GnRH agonists:

The mechanism of action of GnRH agonists is by creating hypoestrogenism by blocking ovarian estrogen production which leads to regression of endometriotic implants[1,37]. Initially there is stimulation of the anterior pituitary which releases FSH and LH. Then further administration leads to inhibition of pituitary GnRH receptors and suppression of the hypothalamic pituitary ovarian axis leading to anovulation[1,37]. This eventually leads to hypoestrogenism, amenorrhea, and regression of the endometriotic implants by depriving the implants of estrogen which is important for their growth[1]. They are usually used in women with previous failed therapy with oral contraceptive pills or women who have medical comorbidity against for COCs[1]. They are available in both nasal and injectable

forms and offer high rates of pain relief and longer symptom free period for up to 12 months.[1]

Leuprolide acetate is available as 3.75 mg injection monthly and 11.25 mg injection available 3 monthly[37]. Others like goserelin and nafarelin are also commonly used as GnRH agonists[37]. GnRH agonists can cause significant reduction in pelvic pain in women with endometriosis and they have been approved for continuous use for only up to 6 months due to concerns of side effects secondary to hypoestrogenism - vaginal atrophy , hot flashes, dryness, bone loss and abnormalities in lipid profile[1,37]. Currently add-back therapy provides symptomatic relief and decreases the rate of bone loss[1]. Norethindrone acetate, low-dose estrogen and a combination of estrogen and progesterone have also been used for adding back therapy to decrease the risk factors of using only GnRH agonists. Hence can also be used for longer periods of up to 12 months[37]. Beyond 12 months no studies have been done. The commonest addback therapy in combination with GnRH agonists is norethindrone acetate[37]. Women wanting to conceive cannot be eligible for GnRH agonists as there will be prolonged suppression of ovulation leading to contraceptive action and hence cannot be used in women who have not completed family and desiring to conceive[37].

GnRH antagonists:

GnRH antagonists have been used on patients who are prone for hypoestrogenism effects of using GnRH agonists[37]. Long term use of GnRH agonists has caused hypoestrogenism on long term use, this has not been found in GnRH antagonists treatment[37]. There is a proven decrease in estrogen level, better compliance and tolerance among women who were changed over to GnRH antagonists from prior GnRH agonists[37].

In a study done by, Kupker et al [37] concluded that treatment with GnRH antagonist cetrorelix showed significant symptomatic relief and decrease in size of the endometriotic implants visualized under laparoscopy.

Progesterone Containing Contraceptives:

There are many modes of action of progesterone in the treatment of endometriosis[38].

1. It causes decidualization of the endometrial implant,
2. Inhibits the mitosis induced by oestrogen by blocking and altering the estrogen receptors[38],
3. Prevents vascularisation and decrease growth of the endometriotic implants.

Progesterones are available in different forms such as oral tablets, parenteral injections and implants and IUDs[1,38].

1. ***Medroxyprogesterone****:*

   It is available as oral and parenteral injectable preparations. It has better compliance when administered as 150 mg intramuscular injection once every 3 months[39]. There is added advantage, it by passes the first pass mechanism. Hence it also avoids common side effects like gastritis which is commonly seen in oral medications.[39]

2. ***Norethindrone acetate:***

   It is a progestin, 19- nortestosterone derivative, used in pain relief for dysmenorrhea, dyspareunia, dyschezia and deep pelvic pain[31]. It had better tolerance and compliance with patients when used in low doses. A study done by Vercellini et al [31] had used norethindrone acetate 2.5 mg/d for 12 months and was found to have similar pain relief as compared with combined oral contraceptive pills[31]. Hence used as alternative to COC's in women with contraindications to their use.

3. *Dienogest:*

It is also a 19-nortestosterone derivative, progestin used for treatment of endometriosis. It acts on progesterone receptors with more specificity[40].

Its mode of actions:

a. Decidualization of endometrial implants,
b. Atrophy of the endometrial lesions,
c. Anti-inflammatory and anti-angiogenic properties,
d. Inhibits mitosis of the endometrial tissue.

It is available as oral tablets in doses of 2mg/d or 4 mg/d. It has easy compliance and less antiandrogenic side effects[40]. There has been overall improvement in the endometriosis-related symptoms and in quality of living for the patient [40]. There is however associated side effect of moderate irregular menstruation.

4. *Levonorgestrel Containing Intrauterine Systems (LNG-IUS):*

LNG-IUS is a T-shaped intrauterine device containing levonorgestrel 52mg. It releases 20mg of hormone for about 5 years. Direct delivery of the

progesterone bypasses all systemic side effects[41]. The compliance for LNG-IUS increases with longer use, mainly to women not keen to conceive.

The mechanism of action of LNG-IUS:

a. Atrophy of the endometrial tissues, decreases endometrial shedding, leading to less retrograde menstruation,
b. Anti-inflammatory on the endometriotic implants.

In a study by Vercellini et al [42] LNG-IUS usage helped in suppressing endometriosis related pelvic pain and improved patient compliance with associated pain relief in patients who were found to have deep rectovaginal endometriosis.

5. ***Etonogestrel Implant:***

It is a progestin. This is administered by subdermal route of administration. It is available commonly as Implanon and Nexplanon and inserted intradermally in the arm. It is used as contraceptives for 3 years[1]. This implant has been found to have similar efficacy in endometriosis related pain relief as DMPA injections[1]. Also has similar side effects such as irregular menstrual bleeding, weight gain, nausea, acne, headache and breast tenderness, similar to DMPA injections[1]. It is also used as contraceptives on women who are not keen to conceive.

Aromatase Inhibitors:

Aromatase enzyme acts by the conversion of steroid precursors into oestrogen. Ovaries and adipocytes are the main source of the enzyme[43]. Aromatase enzyme activity is absent in the endometrium inside the uterus, but it is over expressed in endometriotic implants.[43] Aromatase enzyme causes estrogen synthesis, then growth of the endometrial implants, COX expression and the prostaglandin secretion[43].

Aromatase inhibitors are used to stop the synthesis of estrogen in the periphery as well as in the ovaries[43]. This mechanism is particularly used in postmenopausal women with endometriosis where the peripheral fat is the predominant source of estrogen. Anastrazole, letrozole, and exemestane are third generation aromatase inhibitors that can be administered orally[43].

The combination of COC's+ GnRH agonists/ COC's+ progesterone's/ COC's+ aromatase inhibitors can significantly decrease the endometriosis related pain, if used minimum for 3 months of duration[43]. There has been improvement in quality of living and reduction in size of the endometriomas. Disadvantage involves formation of ovarian follicular cyst and bone loss with long term use for more than 6 months[43]. Combination with GnRH agonists or COC's have been known to prevent follicular development and add back oral contraceptives progestins can decrease the

bone loss[1]. They are more compliant to use, reversible, more effective and has faster onset of action[43].

Danazol:

It is a 17 alpha-ethinyl testosterone, it is an androgen that blocks LH surge and decreases ovarian steroidogenesis by direct inhibition of the ovarian enzymes. It has been effective in controlling endometriosis related pain[44]. However, due to its androgenic side effects like acne, hirsutism, deepening of voice, weight gain, muscle cramps, liver dysfunction, and an abnormal lipid profile its usage got decreased[44].

As the side effects are mostly associated with oral administration, alternative routes of like danazol vaginal ring and intrauterine devices are currently in research[44].

MANAGEMENT OF OVARIAN ENDOMETRIOMA

Endometriomas larger than 3 cm are not known to respond well to medical therapy. Management of endometrioma includes medical and surgical treatment[2].

Medical management is offered first for 3 to 6 months in women with:

1. Endometrioma with size less than 5cm,
2. Reproductive age group wanting to conserve ovarian reserve,
3. Preoperative reduction in endometrioma size.

Surgical management is offered in women with:

1. Endometriomas of size more than 5cm,
2. Resistant to medical management,
3. Recurring cases even on medical management.

A study[45] showed 25% decrease in endometriotic cyst diameter after administrating GnRH-a when compared with placebo[2]. Medical therapy may improve symptoms of pelvic pain and dyspareunia, but it interferes with fertility treatment[46]. On the other hand, there is a small risk of cancer in those endometriomas which are left[2]. Despite a 57% reduction in the size of endometriomas after medical therapy in the management of ovarian endometrioma the most effective way to treat these patients is the surgical method[2].

[47]A study compared the effects of danazol and GnRH-a on endometriomas and found a decrease of size of about 40% to 57% in endometriotic cysts and did not find any difference between these 2 drugs in decreasing the size of endometriomas.

Very few studies have shown the effect of only medical therapies on endometriomas size reduction[47]. This study aims to compare the efficacy of the various medical management for decrease in size of endometriomas and to evaluate the need for change in medical management among patients on treatment for ovarian endometriomas in CMCH Vellore Gynaecology OPD for a period of 3year.

MEDICAL MANAGEMENT OF ENDOMETRIOMA:

Commonly used medical modalities are:

1. Extended spectrum oral contraceptive pills,
2. Progestins,
3. Anti-GnRHs,
4. Aromatase inhibitors,
5. Danazol.

Extended cycle OCPs:

Estrogen and progesterone combinations pills cause decidualization of the endometriotic tissue and delays the growth of endometriosis. As compared to cyclic administration, Extended therapy with COC's[48] has been shown to reduce and delay the recurrence rate of endometriomas by 8.2%, compared to 14.7% in cyclical OCPs.

Progestins:

It causes suppression of GnRH, leading to suppression of FSH and LH leading to decrease in estrogen, anovulation, decidualization of the endometrium, inhibits estrogen induced mitosis, inhibits angiogenesis [46]and expression of matrix metalloproteinase [49]needed for the growth of the endometriotic implants.

Available in different forms oral, injectable, vaginal, or intra-uterine device. Progestins have shown to have resistance of treatment in endometriosis due to decreased PRB gene in endometriotic tissues, caused by PRB promoter methylation due to increased inflammatory cytokines in endometriosis.[50]

1) **Injection-Depot Medroxyprogesterone acetate (Injection DMPA):**

   Derivative of C21 progesterone. An RCT study in Thailand [51]July 2018 on 28 women with endometriosis treated with DMPA injection in 3months resulted in suppression of cell proliferation and enhanced apoptosis of ectopic endometrium of women with endometriosis.

2) **Dienogest:**

   Suppresses the hypothalamo-pituitary-ovarian axis, reduces GnRh secretion, causes hypoestrogenic state but estrogen production is not suppressed. Hence hypoestrogenic side effects are less. An RCT study [52], on effect of Dienogest therapy on size of endometrioma in Feb 2020, showed reduction in mean volume of endometrioma after 6months treatment was 66.71% and 76.19% after therapy for 12 months.

3) **LNG-IUS:**

Derivatives of C19progesterone. In a multicenter retrospective cohort study, in Gangnam Medical center over 2007 to 2014 [53]. LNG IUS had not much effect on decreasing size of endometriomas but relatively decreased recurrence after postoperative long term maintenance therapy[54].

The medical management of endometriosis suppresses the ovulation and menstruation hence suppresses hormonally active endometriotic tissue and its other symptoms. In this study, we would like to find the effectiveness of various medical management of endometriomas mostly by Extended cycle oral contraceptive pills and with different types of Progestins and the need for change in medical management. To find the most effective way for treatment of decrease in size of endometrioma and to improve tolerance and compliance among patients in our hospital, this study has been done.

METHODOLOGY

AIMS:

To assess the efficacy of various types of medical management of ovarian endometrioma and the need for change in management-medical or surgical, among women of reproductive age group 18 to 45 years who were treated in CMC Vellore gynecology OPD for at least 6months.

OBJECTIVES:

Primary objective:

-To assess the efficacy of medical management in terms of regression of size of ovarian endometrioma retrospectively by viewing online OPD chart data from 2017 to 2019 over a period of 3years among women of reproductive age group 18 to 45years on medical management – extended cycle oral contraceptive pills or progestins for at least 6months in CMC Vellore Gynaecology OPD.

Secondary objective:

-To assess the need for change in management -medical or surgical in regression of size of endometrioma retrospectively by viewing online OPD chart data from 2017 to 2019 over a period of 3years among women of reproductive age group 18 to 45years on medical management for at least 6months in CMC Vellore Obstetrics and Gynaecology OPD.

Introduction:

Endometriosis is a well-known debilitating disease affecting reproductive age women, there are various medical managements for women with this condition. Each woman is treated individually, depending on their symptomatology. There are not many studies available, comparing the efficacy of multiple medical managements in treatment of endometriosis and their effect on ovarian endometrioma. This study will compare the efficacy of various medical managements on endometriosis, on endometrioma and on menstrual cycle. We hope to get an idea on various medical management in use for treatment of endometriosis in CMC Vellore, gynecology OPD and to understand the existing pattern of medical managements along the way for the long-term follow-up in women with endometriosis.

Research design:

Ours was a retrospective observational study on the efficacy of medical management and the need for change in management -medical or surgical, in terms of regression of size of ovarian endometrioma. Charts of 300 patients who consulted CMC Vellore Gynecology OPD from 2017 to 2019 and on medical management for ovarian endometrioma for at least 6 months were reviewed.

This study was done as a chart review, on women in reproductive age group of 18 to 45years. These women were on a medical management for endometrioma for at least 6 months during the years 2017 to 2019. Most frequently used medical managements in our hospital for treatment of endometrioma were extended cycle OCP's or progestins like Injection DMPA, Dienogest and Mirena. Following which recruitment of patients according to the inclusion and exclusion criteria was done. The total women selected in the study were 300. Through online charts of patients, the change in size of endometriomas and menstrual regulation were collected and analyzed. Size of endometrioma after the new treatment was noted following repeat ultrasound scan. The details were obtained from the online chart retrospectively. The efficacy of various medical managements on the endometrioma were compared from the time of starting medical management to after the change in management.

Study setting:

The study was conducted retrospectively by reviewing the charts available online. This study was done as a chart review of 300 women, starting from 2017 to 2019 over a period of 3 years within reproductive age group of 18 to 45years. These women were on a prior medical management for endometrioma for at least 6 months with follow up in CMC Vellore gynecology OPD.

Pilot Study:

To assess the efficacy of medical management of ovarian endometrioma and need for change in management-medical or surgical, in terms of regression of size of endometrioma retrospectively, a pilot study was done retrospectively of the month – January of 2017, to calculate the sample size required for 3 years, 2017 to 2019 among women of age group 18 to 45years, in CMC Vellore gynecology OPD.

Based on the pilot data in one month (January 2017), the prevalence of Loette (extended cycle OCP) use in endometriosis was 87.5% (95% CI: 47% - 99%). The sample size calculation was based on provided estimate of 87.5% with 4% precision level and 95% confidence limit, the required number would be around 272.

Inclusion and exclusion criteria:

Inclusion criteria:

We included patients who were on regular medical management for endometriosis in CMC Gynaecology OPD for at least 6months, from 2017 to 2019.

Exclusion criteria:

Women who attained menopause, with pregnancy and lactating mothers were excluded from the study.

Sample size:

The objective of the study was to assess the efficacy of medical management of ovarian endometrioma and need for change of that management-medical or surgical, in terms of regression of size of endometrioma retrospectively by OPD view chart online data from 2017 to 2019 over a period of 3 years in CMC Vellore Gynaecology OPD among women of reproductive age group 18 to 45years on medical management - Oral contraceptive pills or progestins for at least 6 months.

Based on the pilot data in one month (January 2017), the prevalence of Tablet Loette use in endometriosis was 87.5% (95% CI: 47% - 99%). The sample size calculation

was based on provided estimate of 87.5% with 4% precision level and 95% confidence limit, the required number would be around 272.

Single Proportion - Absolute Precision

Expected Proportion	0.875	0.875	0.875	0.875	**0.87**
Precision (%)	2	2.5	3	3.5	**4**
Desired confidence level (1- alpha) %	95	95	95	95	**95**
Required sample size	1050	672	467	343	**272**

Formula

$$n = \frac{Z^2_{1-\alpha/2}\, p(1-p)}{d^2}$$

Where,

p : Expected proportion

d : Absolute precision

1- α/2 : Desired Confidence level

Data collection method:

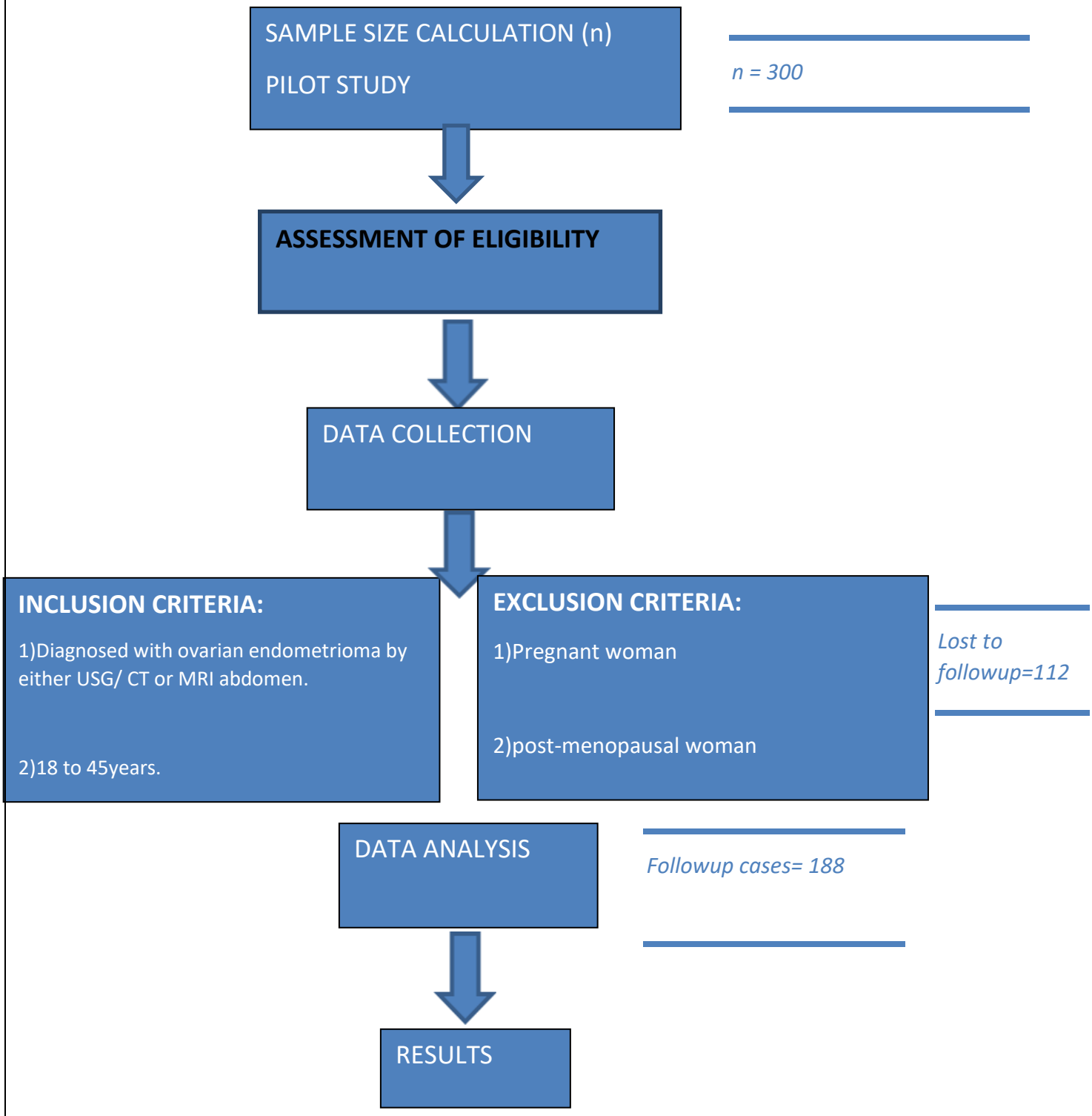
Data was directly collected from CMC Vellore online patient outpatient charts. Sample selection was done, such that at least 300 women from year 2017 to 2019 were chosen, in which about 100 women per year were selected. Information obtained was entered on proforma sheets. Thereafter the data collected were entered on an excel sheet using EPIDATA software. The raw data was analyzed by statisticians to come to conclusion on the efficacy of various medical management in the treatment of endometriosis, specifically ovarian endometriosis.

Statistical methods:

Data was entered using EPIDATA software and screened for outliers and extreme values using Box-Cox plot and histogram (for shape of the distribution). Summary statistics was used for reporting demographic and clinical characteristics. ANOVA was used for the analysis of continuous data with Normal distribution and the Kruskal walis test for data with non- Normal distribution with groups. Chi-square test was performed for categorical variables with groups. Pearson correlation done between two continuous variables. Differences were considered significant at $p<0.05$. All the statistical analyses were performed using Excel and SPSS 25.0.

RESULTS

CONSORT STATEMENT OF THE STUDY



A total of 300 women were recruited for this study. There were 188 women who came for follow-up and 112 were lost for follow-up in this study. initially data was analysed for 300 women , but after followup were analysed for 188.

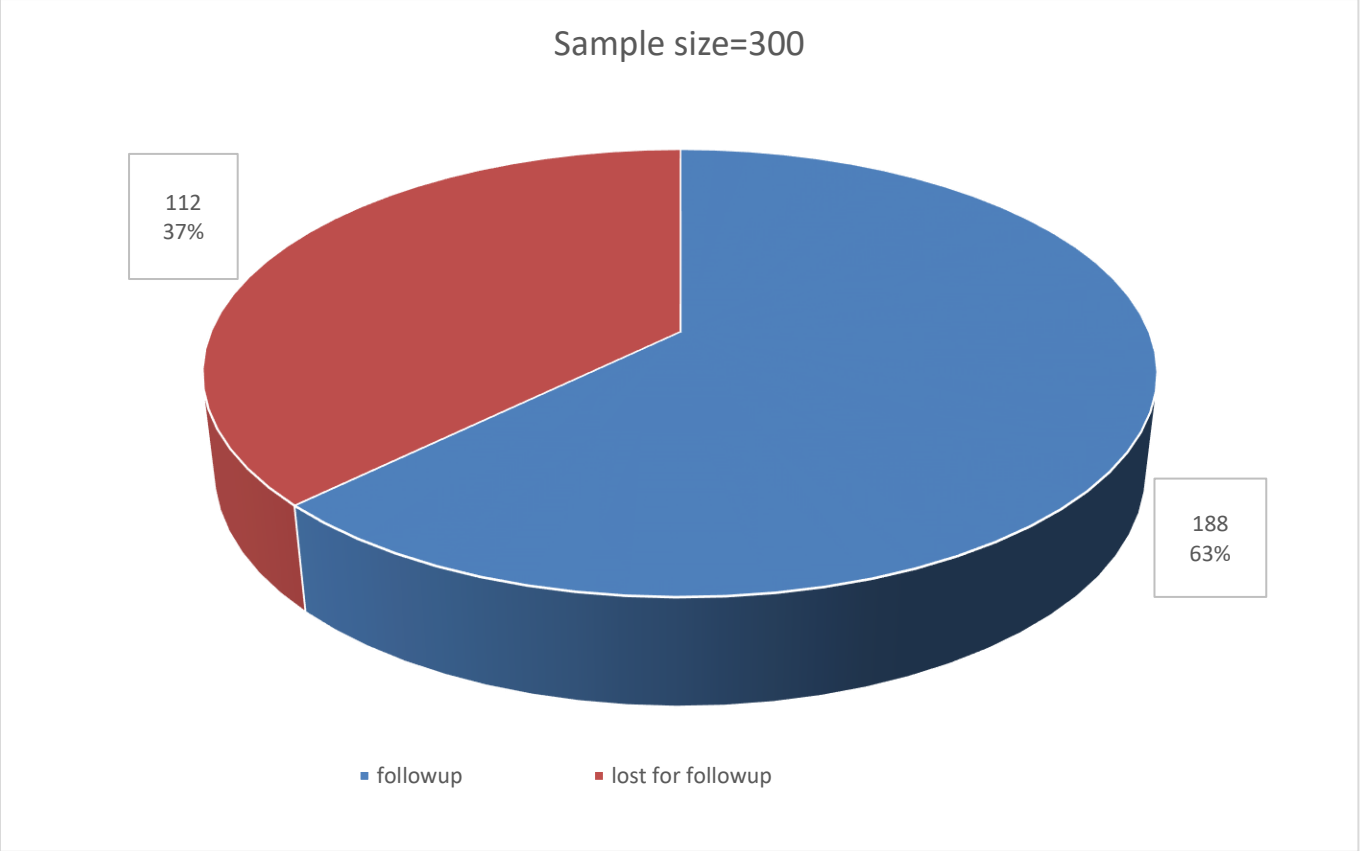


FIG. 1: SAMPLE SIZE OF STUDY

The demographics of the study samples were analysed. Primary and secondary outcome was calculated.

DEMOGRAPHICS:

1.Age:

Out of the 300 sample patients, 47% (139)belonged to between 29 to 39 age group. The mean age of study population was 32.78 years with standard deviation 7.69.

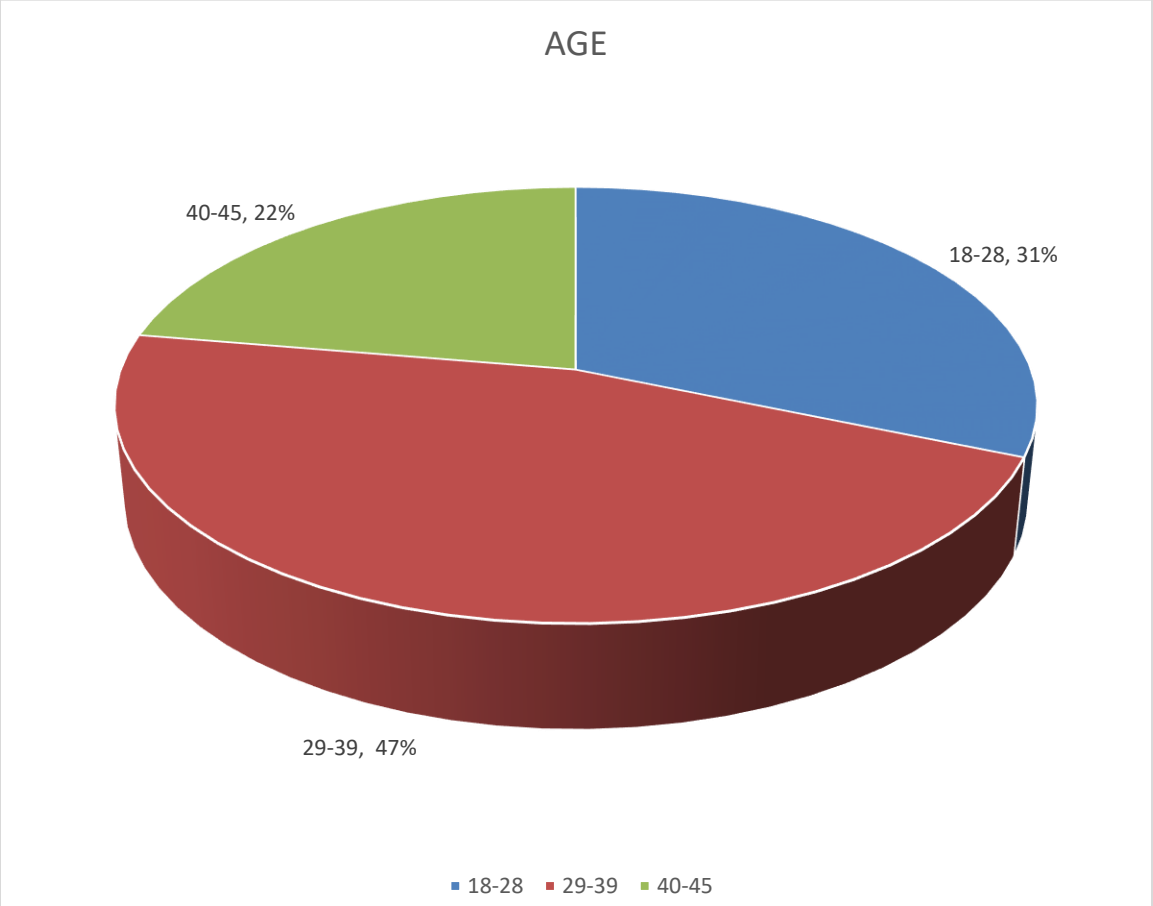


FIG. 2: AGE DISTRIBUTION IN THE STUDY POPULATION

2.BMI:

Out of 300 sample population, 52% (156) of the women belonged to the BMI range of 21 to 26 kg/m2, 32% (95) of the women belonged to the BMI group of range 27 to 32 kg/m2, while 13% (39) of the women belonged to BMI 14 to 20 kg/m2 and only 3% (10) of the women belonged to the category of BMI above 33kg/m2. The mean BMI of the women in the study was 24.5 Kg/m2 with standard deviation 4.2

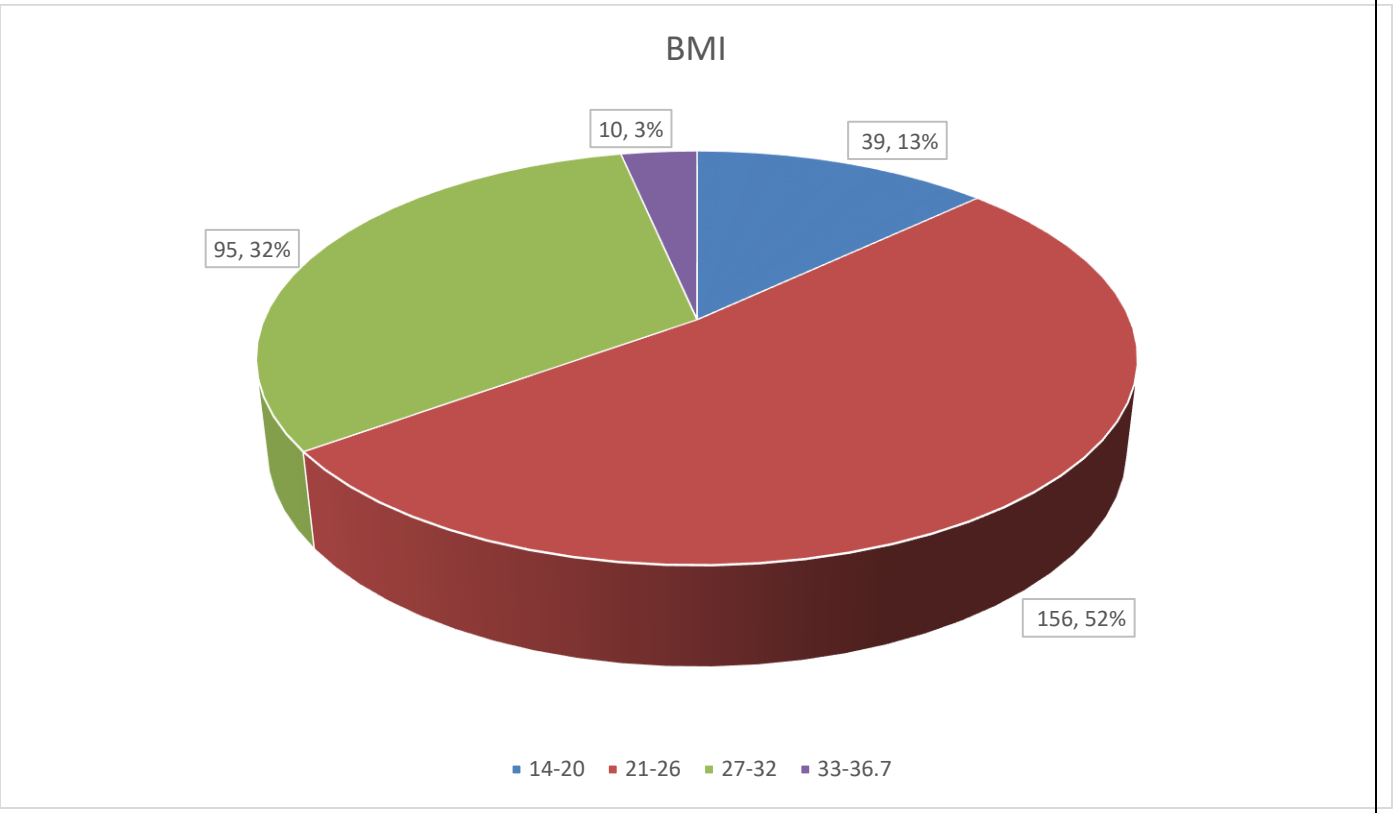


FIG.3: BMI DISTRIBUTION AMONG THE STUDY POPULATION

3. Menarche:

The majority of women, 61%(182) attained menarche around 13 to 15 years of age, 38%(115) of the women attained menarche at 10 to 12 years of age and around 1% (3) attained menarche at 16 to 18 years of age. The mean age of attaining menarche in this distribution of sample patients was 12.8 with a standard deviation of 1.1. There were 5 patients who had attained menarche at 10 years of age.

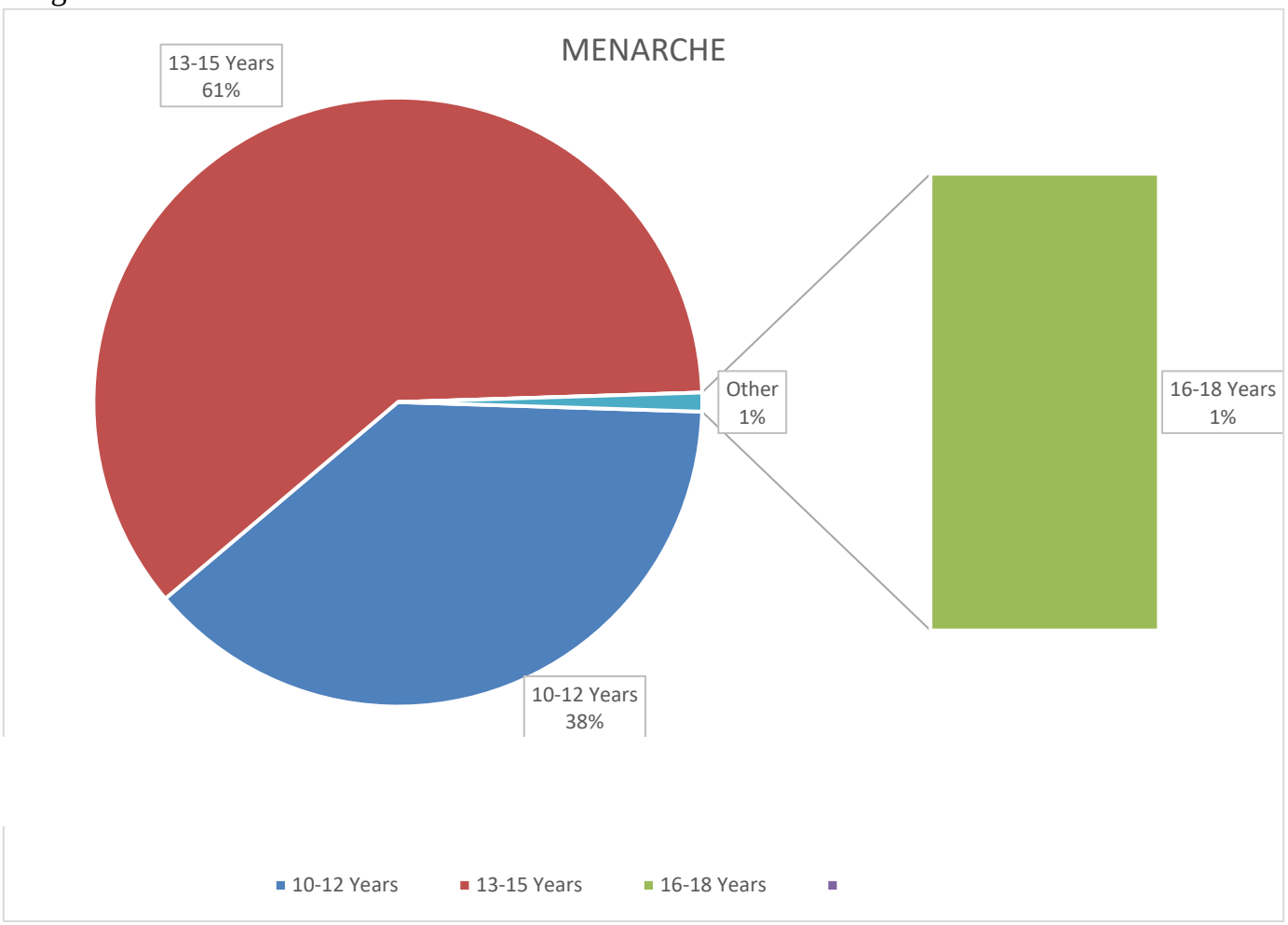


FIG. 4: ONSET OF MENARCHE DISTRIBUTION AMONG THE SAMPLE POPULATION

4.Marital status and sexual activity:

Out of the 300 women participating in the study, 73% (219) of them are married and sexually active. 27% (81) are single.

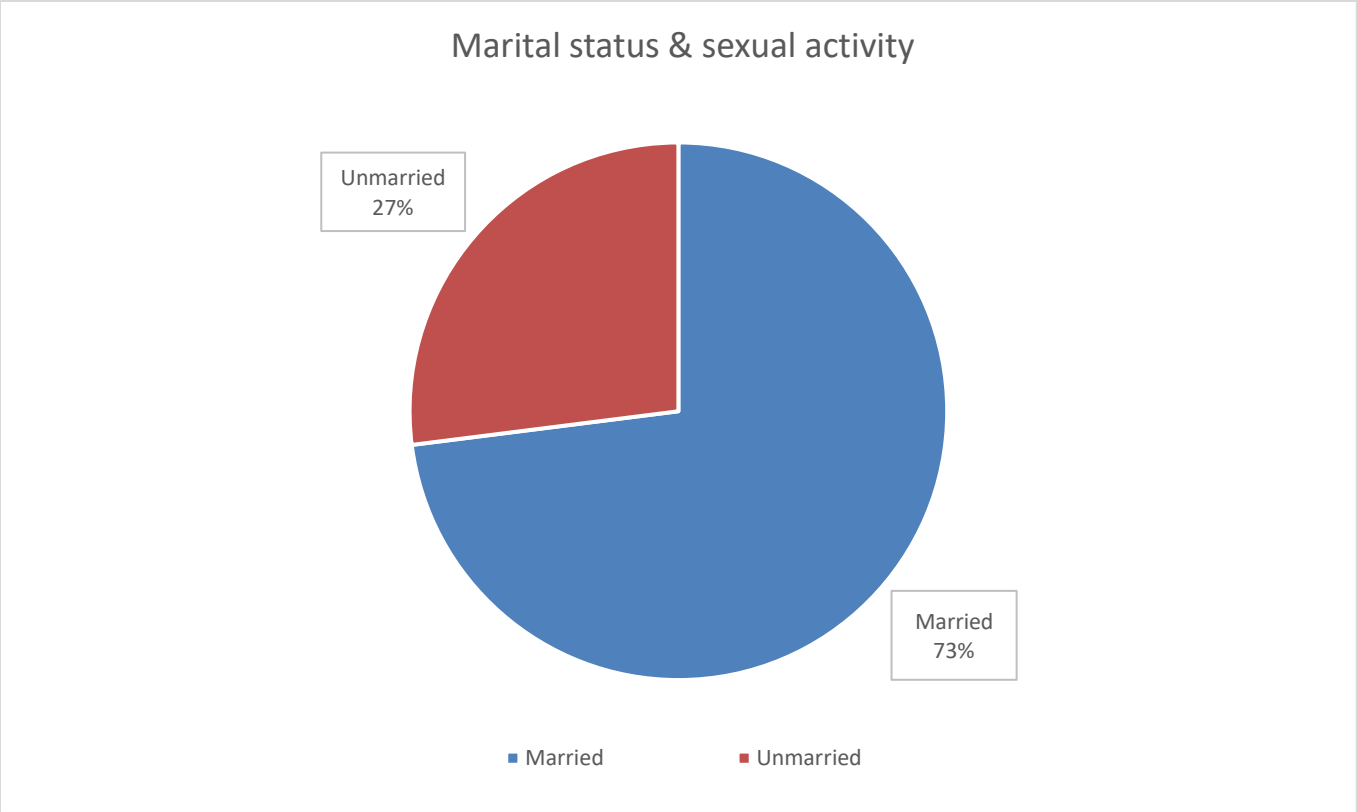


FIG. 5: DISTRIBUTION OF MARITAL STATUS AND SEXUAL ACTIVITY AMONG THE SAMPLE SIZE

5.Parity:

Women with parity 1, consist the majority, 41%(123) of the study population, followed by 32.3% (97) being without children. 81 out of the 97 women with 0 parity were single.

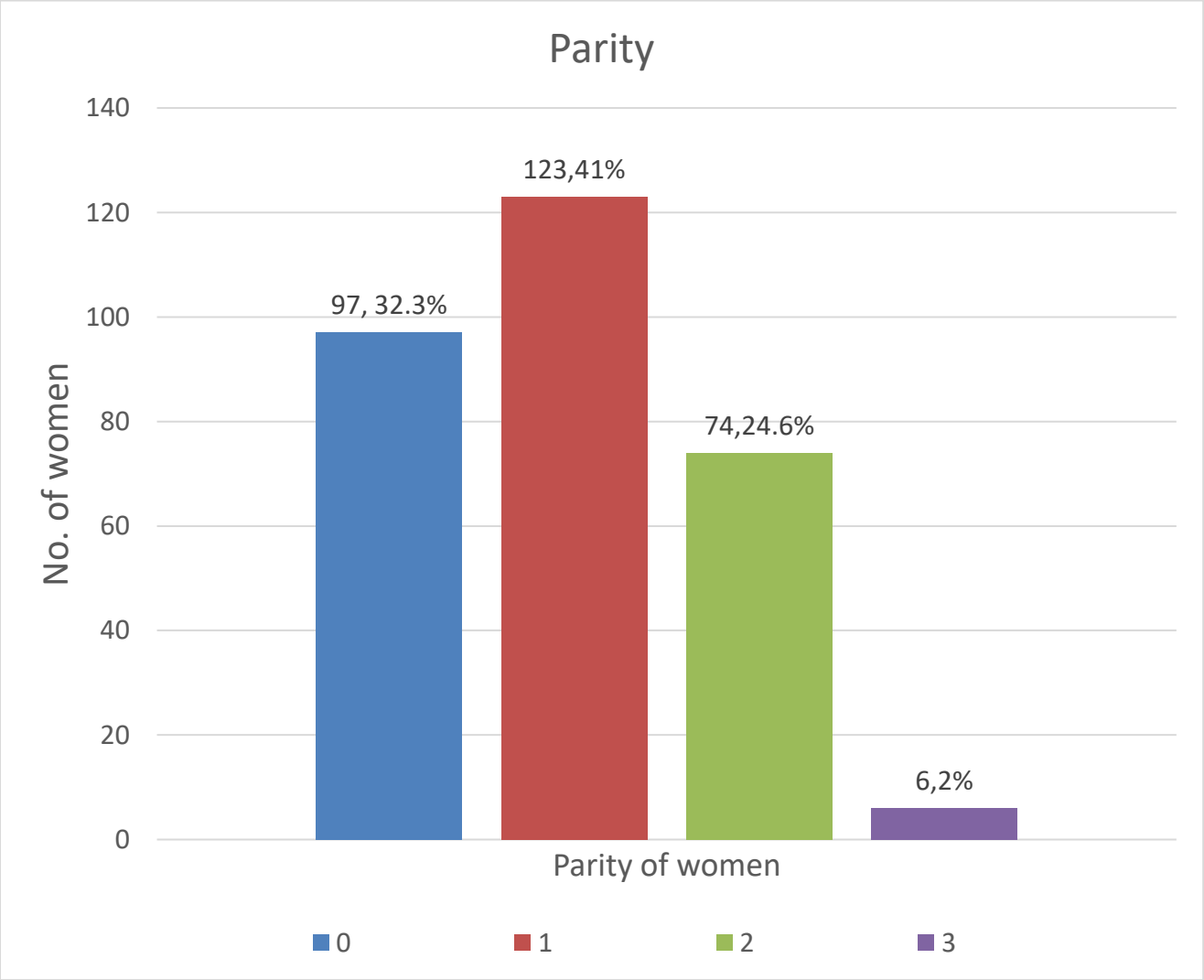


FIG.6: PARITY DISTRIBUTION OF WOMEN IN THE STUDY POPULATION

6. Menstrual cycle before first line medical management:

91.3% (267) of the women in the sample size of 300 had regular menstrual cycle, while 4.3% (13) had frequent cycles and 4.3% (13) had infrequent menstrual cycles.

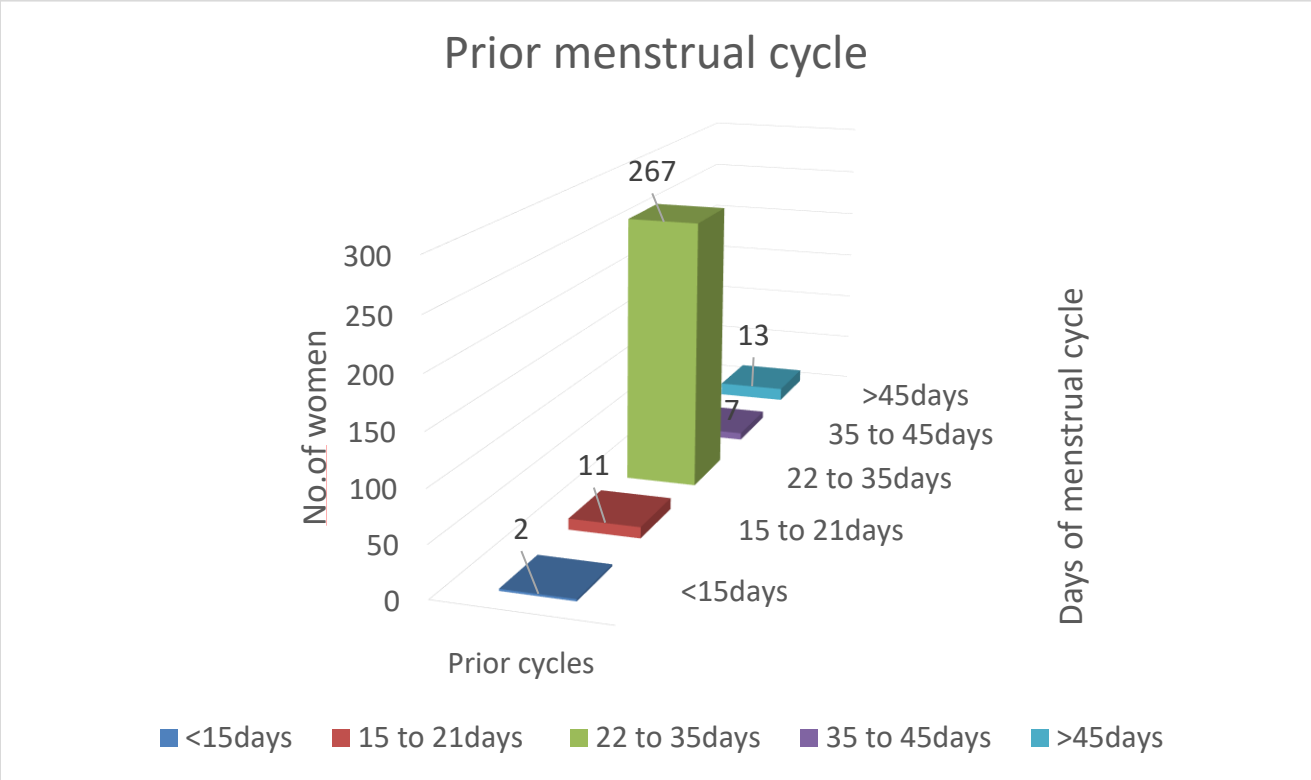


Fig.7: Pattern of menstrual cycle before first line medical management:

7. Menstrual cycle after first line medical management:

84.5% (159) of the women in the followup group of size of 188 had regular menstrual cycle, while 2% (4) had frequent cycles and 13.2% (25) had infrequent menstrual cycles.

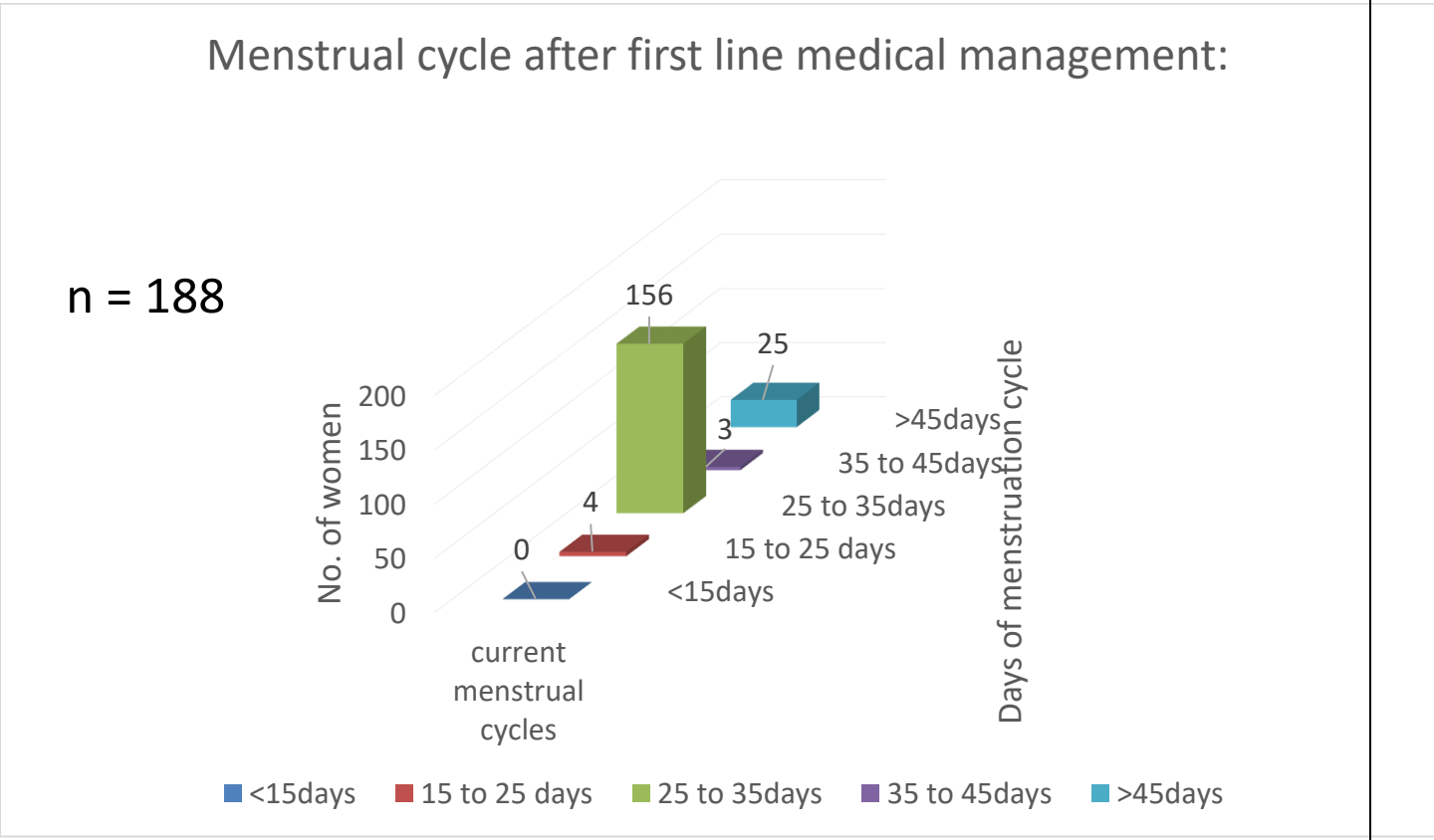


FIG.8: MENSTRUAL CYCLE AFTER FIRST LINE MEDICAL MANAGEMENT

8.Pattern of bleeding after medical management:

Out of the 300 women who were started on first line medical management, only 188 came for followup and 112 were lost for followup. 82% (154) on followup had breakthrough bleeding, 12% (23) of them had withdrawal bleeding and 6% (11) of them had amenorrhoea.

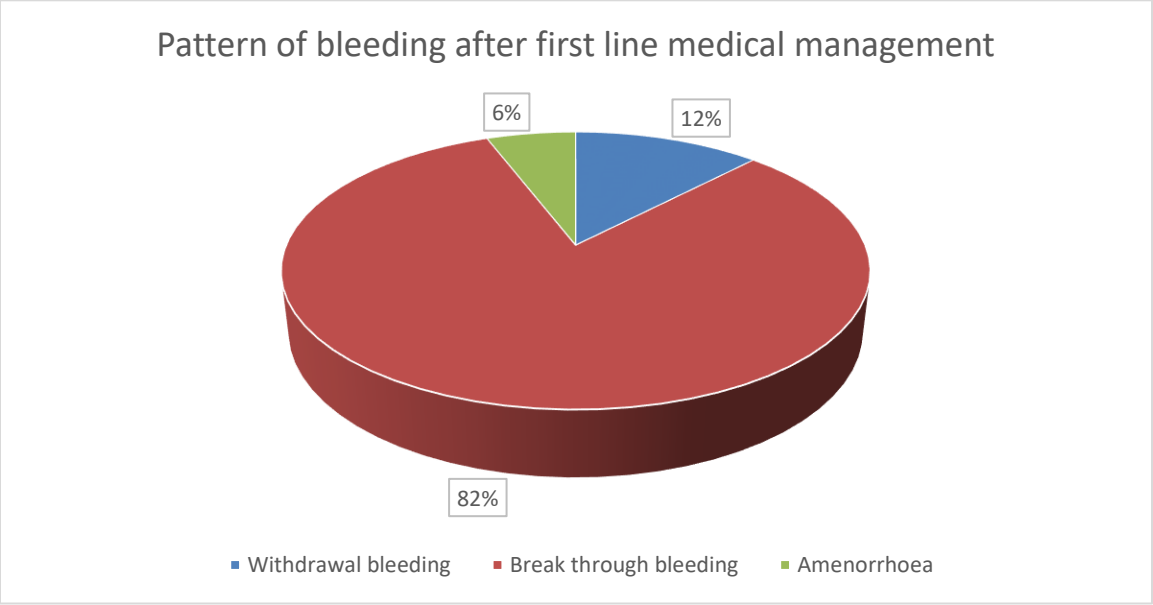


FIG.9: PATTERN OF BLEEDING AFTER FIRST LINE MEDICAL MANAGEMENT.

9. Break through bleeding:

Out of the 154 people with breakthrough bleeding 133 (86%) patients were on extended cycle OCPs, 8% (12) were on inj.DMPA and 3% each were on Mirena and Dienogest.

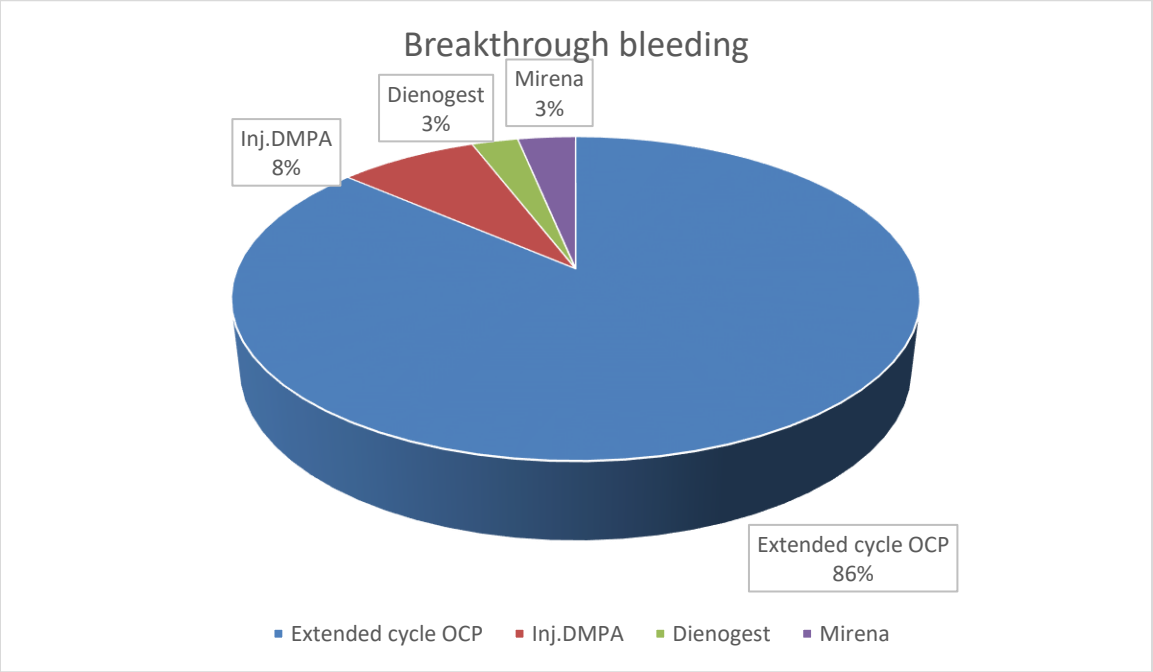
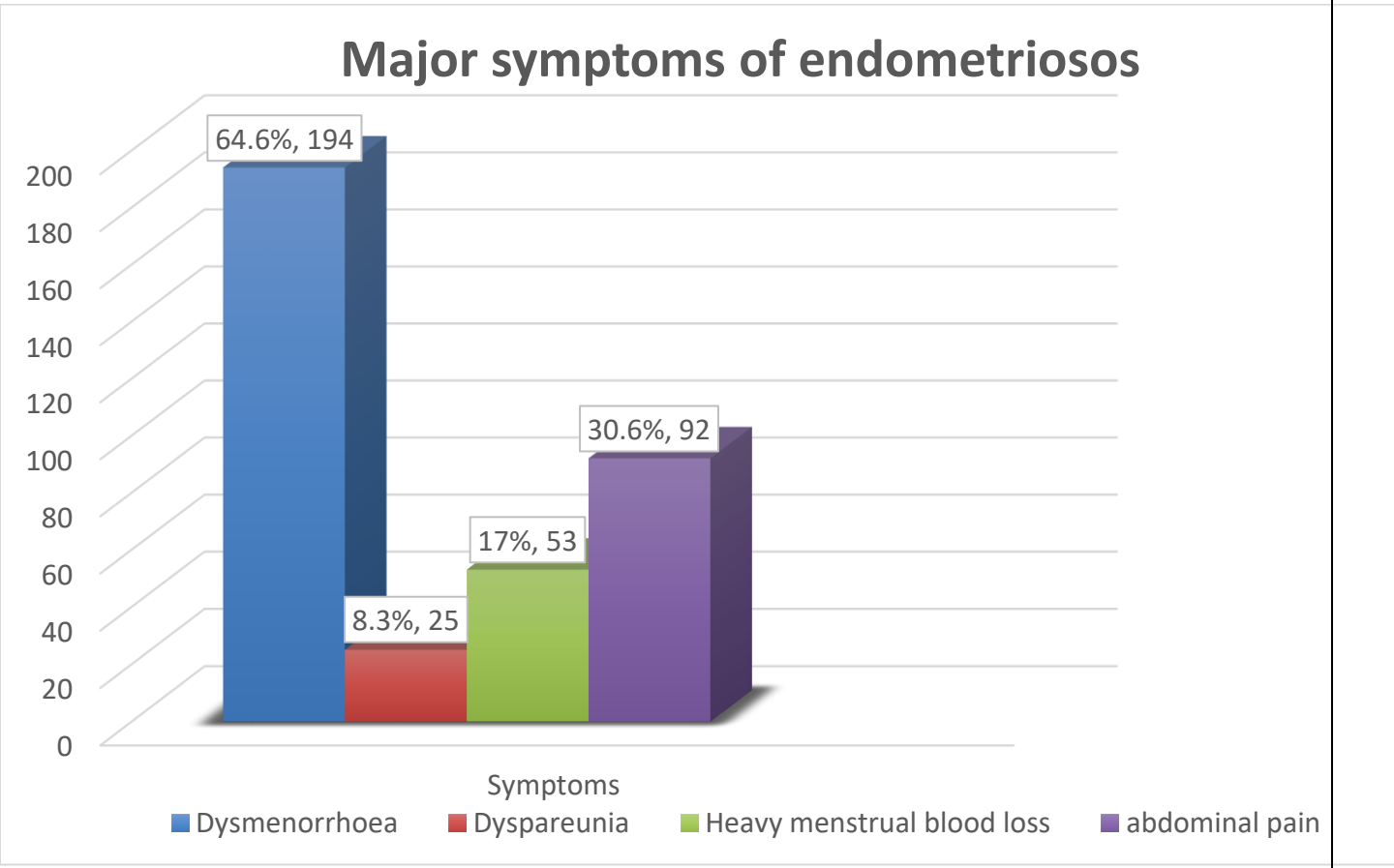


Fig.10: Break through bleeding with various medical management in the study population

10. Symptoms prior to first line of medical management:

The major symptoms reported by sample size of 300 patients during their first visit to OPD were 1)Dysmenorrhoea,-64.6% 2)Non cyclical abdominal pain-30.6%, 3)Heavy menstrual blood loss-17.% and 4) Dyspareunia-8.3%.



PRIMARY OUTCOME:

1) Out of the 300 women with endometrioma , 260 (86.6%) women received extended cycle oral contraceptive pill as the first line of medical management. While 24 (8%) people received inj.DMPA, 6 people (2%) received dienogest and 10 people (3%) received LNG IUS as first line medical management in the study population.

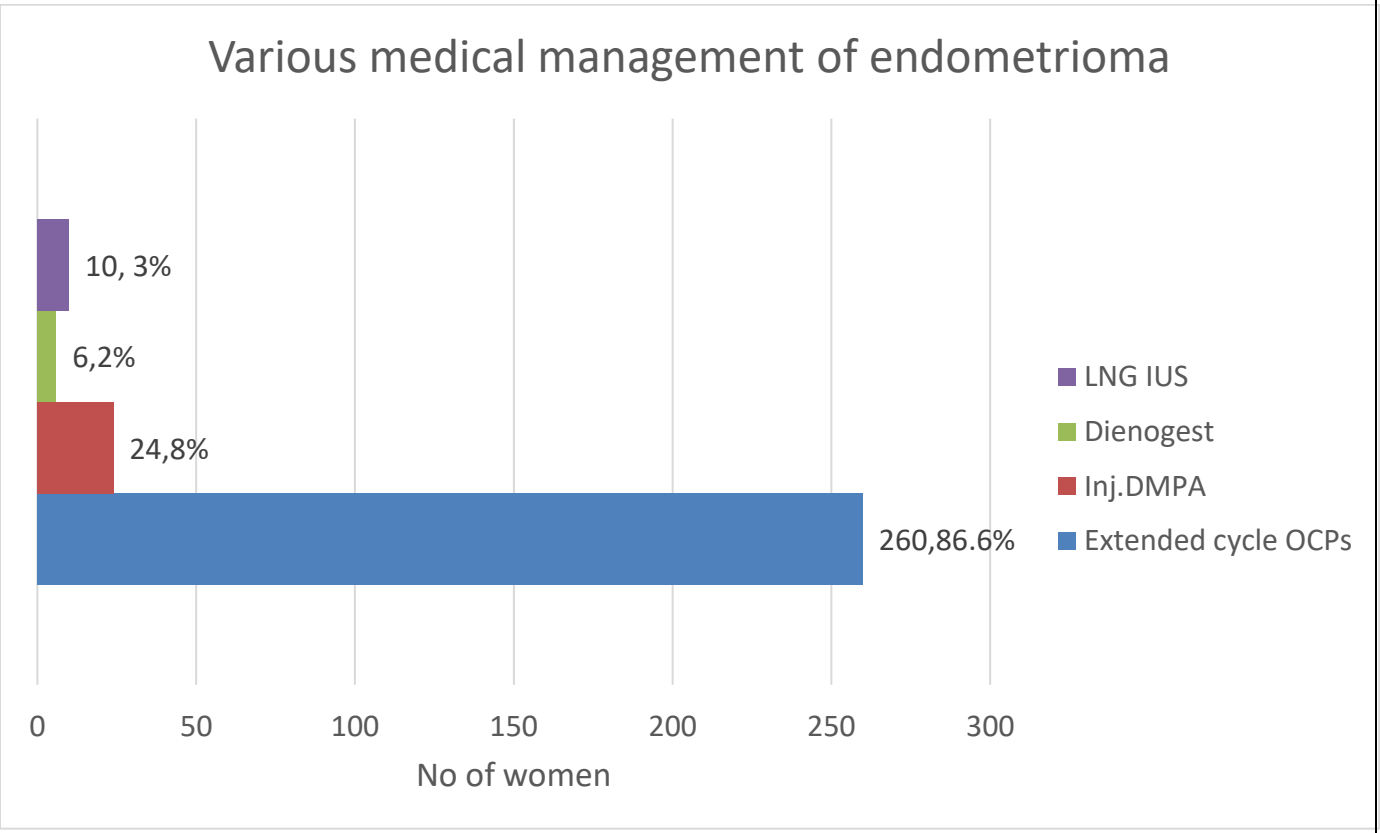


Fig. 11: Various medical management distribution in study population

2)In the study population of 300 women with endometrioma, 96% (288) were diagnosed with endometrioma by ultrasound abdomen, followed by 2% (6) by MRI, 1.3% (4) by CT abdomen and 0.6% (2) intra operatively.

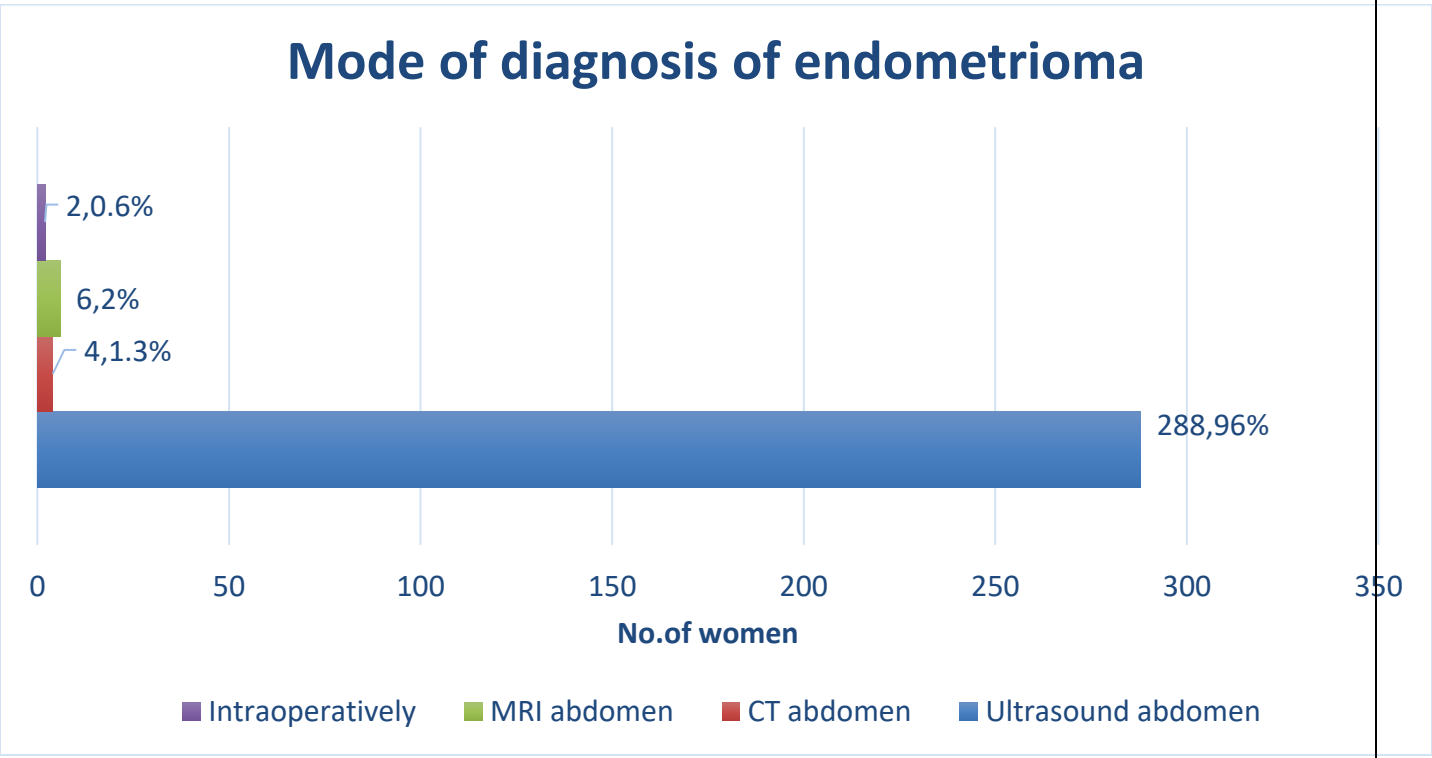


FIG.12: MODE OF DIAGNOSIS OF ENDOMETRIOMA IN STUDY POPULATION

3)Side of endometrioma:

69% (113) of the endometrioma were found on left side among the total sample size of 300 patients.

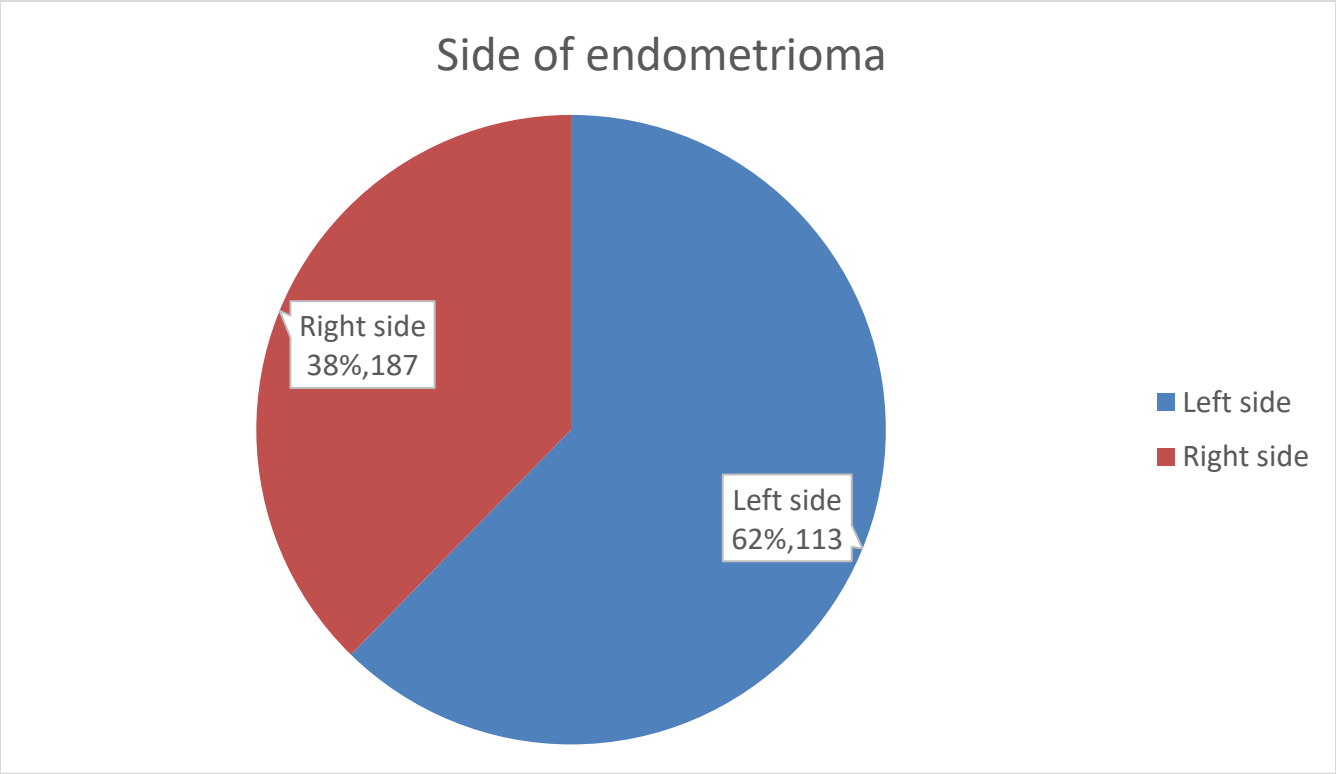


FIG.13: SIDE OF ENDOMETRIOMA AT DIAGNOSIS AMONG THE STUDY POPULATION

4)Changes in size of endometrioma after first line medical management:

Out of the 188 patients who were on followup, 14 had not repeated ultrasound, hence out of 174 people 79% (138) had reduction in size of the endometrioma, 19% (32) had increase in size of endometrioma, 2% (4) had no change in size of endometrioma. Out of the 138 people who had reduction in size of endometrioma, 13 (6.9%) had complete resolving of the cyst.

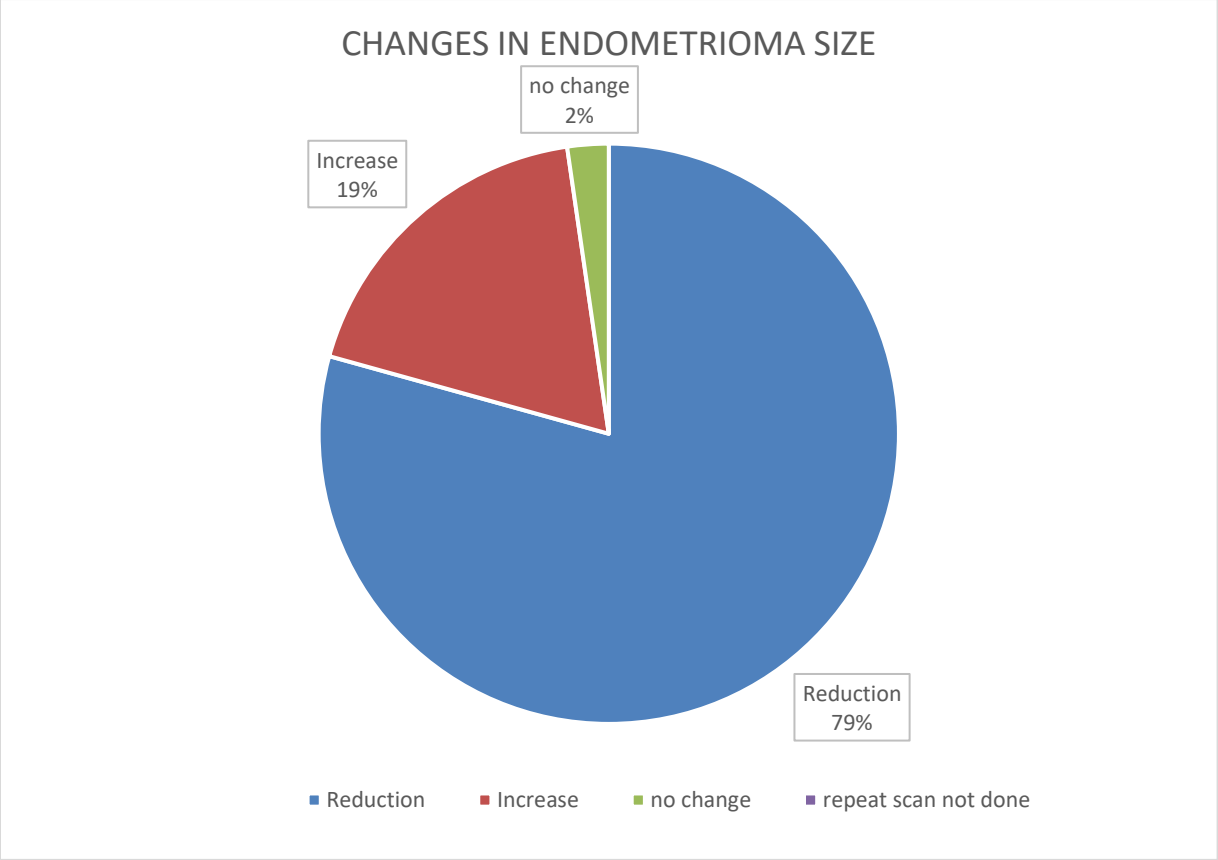


FIG.14: CHANGES IN ENDOMETRIOMA SIZE

5)Reduction in size of endometrioma by various medical management:

a) Extended cycle OCPs- out of the 174 patients who came for followup with ultrasound abdomen 154 received OCP as their first line of treatment, out of which 106 of them had reduction in cyst size. It is 68.8% reduction. The mean reduction in size of endometrioma in using Extended cycle OCP was 1.9cm.
b) Inj. DMPA- out of the 174 patients who came for followup with ultrasound abdomen, 21 received inj.DMPA as their first line of treatment, out of which 13 of them had reduction in cyst size. It is 61.9% reduction. The mean reduction in size of endometrioma in using inj.DMPA was 1.62cm.
c) Dienogest- out of the 174 patients who came for followup with ultrasound abdomen, 5 received dienogest as their first line of treatment, out of which 3 of them had reduction in cyst size. It is 60% reduction. The mean reduction in size of endometrioma in using dienogest was 2.33cm.
d) Mirena - out of the 174 patients who came for followup with ultrasound abdomen 4 received mirena as their first line of treatment, out of which 3 of them had reduction in cyst size. It is 75% reduction. The mean reduction in size of endometrioma in using Extended cycle OCP was 1.6cm

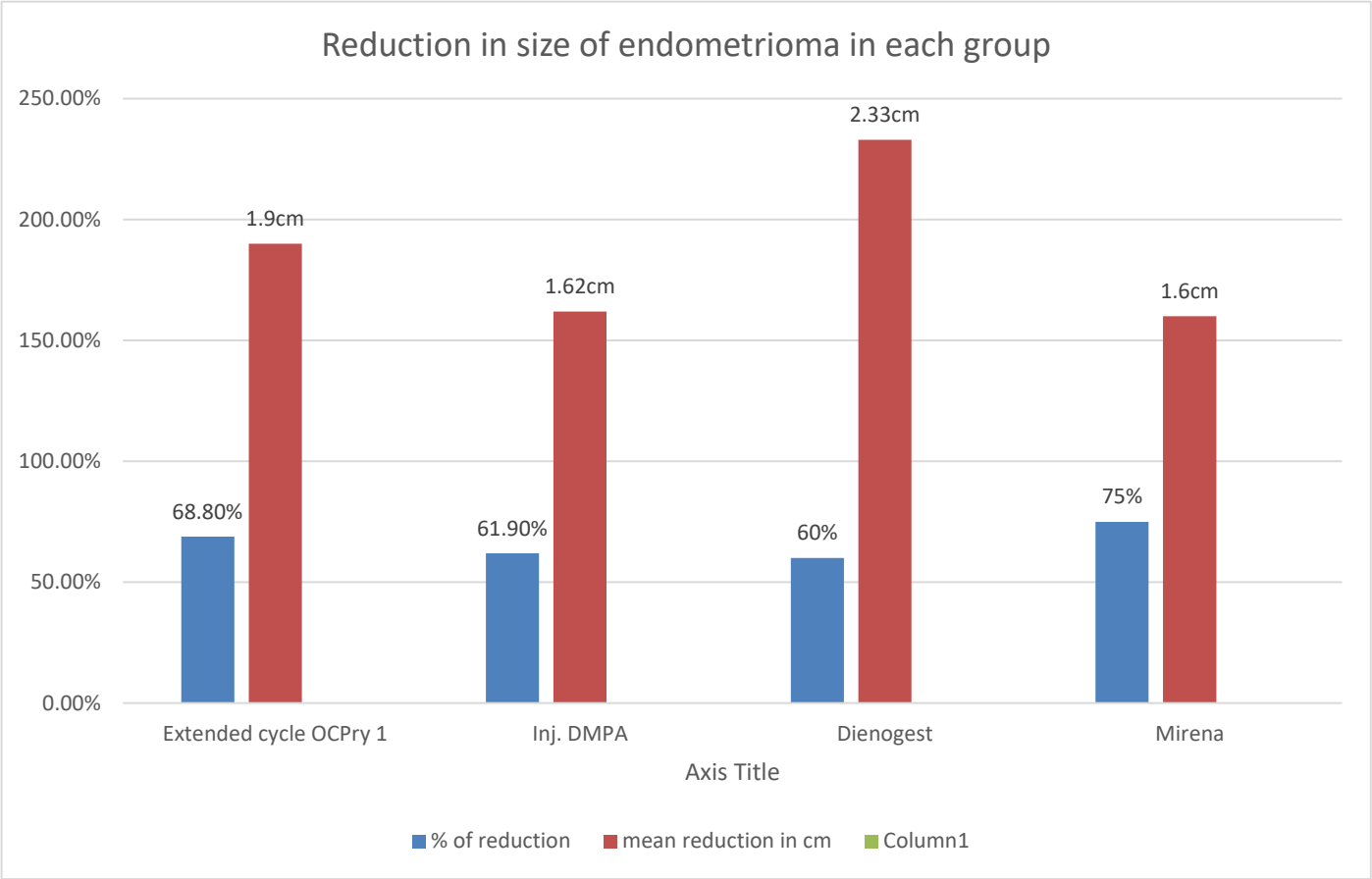


FIG. 15: REDUCTION IN SIZE OF ENDOMETRIOMA IN EACH GROUP IN THE SAMPLE SIZE

Chi-Square Test applied:

Comparing reduction in size of endometrioma:

1) Extended cycle OCP vs Inj.DMPA

	Reduction	Not reduced	Row total
Extended cycle OCP	106	48	154
Inj. DMPA	13	8	21
	119	56	175
	P value 0.77, not significant.		

2) Extended cycle OCP vs Dienogest

	Reduction	Not reduced	Row total
Extended cycle OCP	106	48	154
Dinogest	3	2	5
	109	50	159
	P value 0.67, not significant.		

3)Extended cycle OCP vs Mirena

	Reduction	Not reduced	Row total
Extended cycle OCP	106	48	154
Inj. DMPA	3	1	4
	109	49	158
	P value 0.79, not significant.		

e)Combined therapy:

Out of total 300 people, 25 had taken combined treatment

The combination therapy included two or three drugs including extended cycle OCPs group, inj. DMPA group, Novelon, danazol, cyclical loette, novex and cyclical ovral-L.

Reasons for combination therapy:

S.NO	Reasons for combined therapy in the study sample
1)	Heavy menstrual blood loss,
2)	Severe congestive dysmenorrhea and
3)	Dyspareunia.

SECONDARY OUTCOMES:

1)Change in management:

	% change in management
Change in management 98/188	52.1%
Changed to medical management 37/98	37.7%
Changed to surgical management 61/98	62.2%

Out of the 188 women who were on followup, 98(52.1%) had change in management.

-37 (37.7%)had change over to other medical management and 61(62.2%) had underwent surgical m

Among those who had change in medical management – 78% (29) were from the Extended cycle OCPs group, 24% (9) belonged to Inj.DMPA, 2.7% (1) belonged to Dienogest and 5.4% (2) belongs to Mirena.

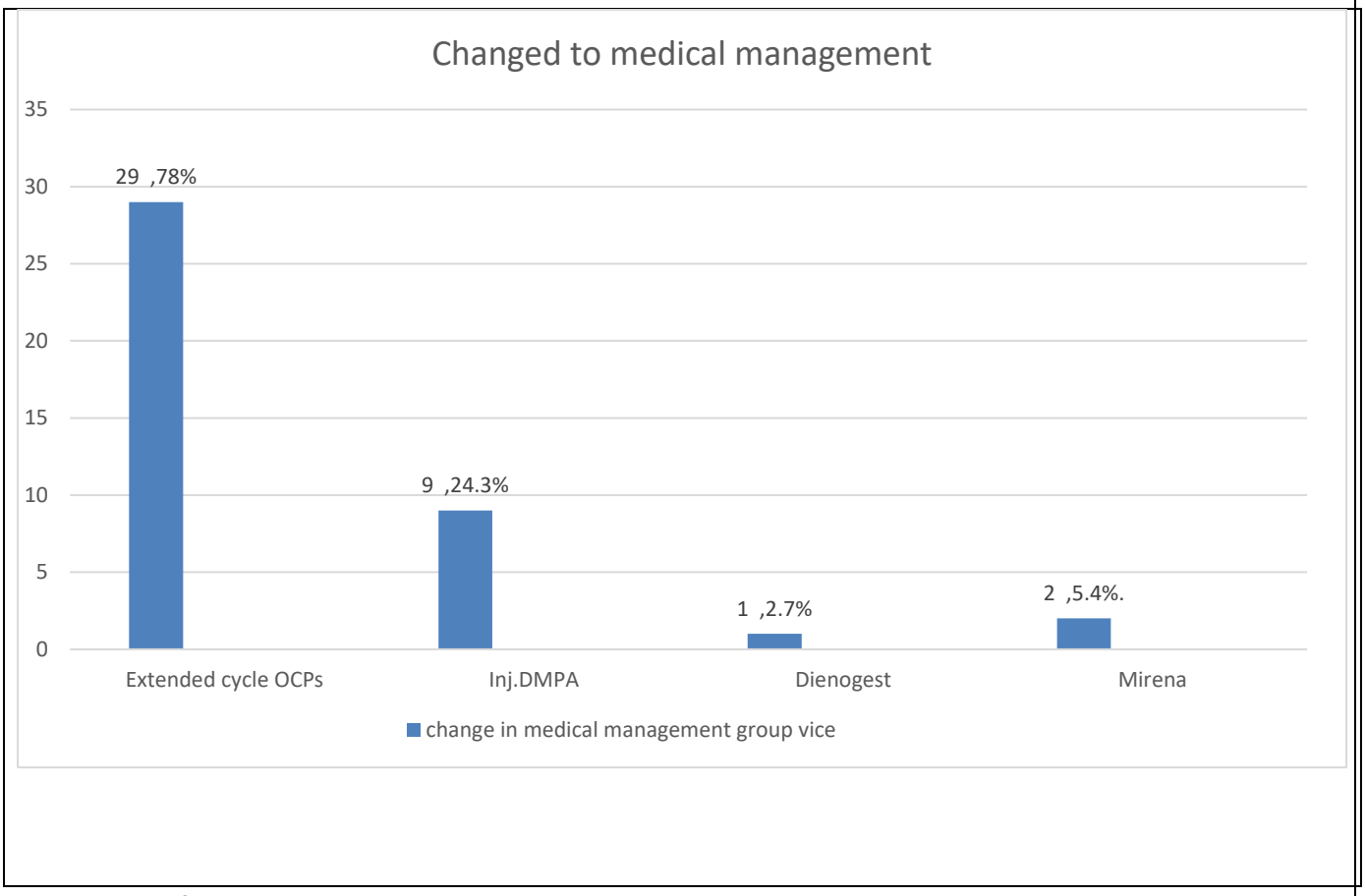


Fig.16: No of women who had change in medical management

Out of the 61 women who underwent surgery, 42 belonged to the extended cycle OCPs, 12 belonged to Inj.DMPA group, 4 belonged to dienogest and 2 belonged to Mirena group.

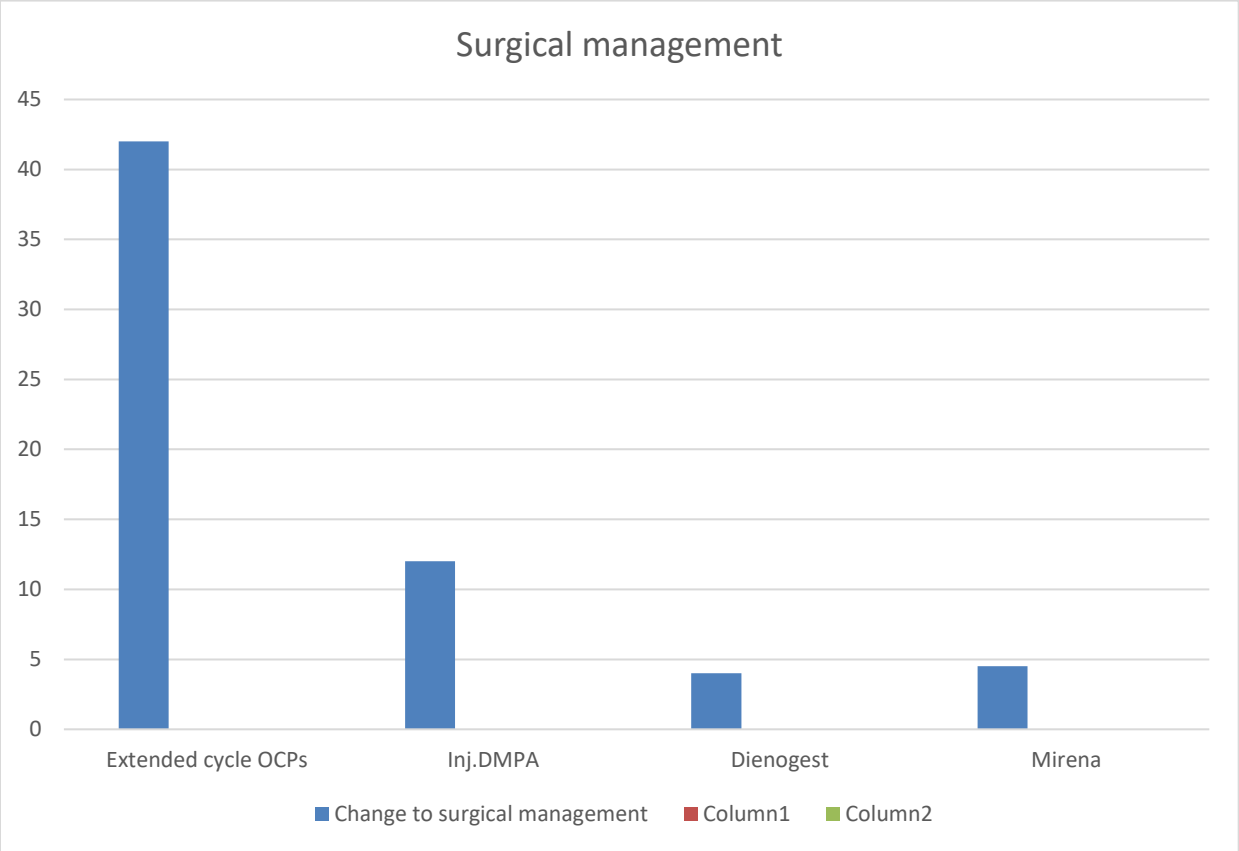


FIG.17: NO OF WOMEN WHO HAD CHANGE IN SURGICAL MANAGEMENT

2)Need for change in medical therapy:

Out of the 37women who had change to medical management, 30 of them had dysmenorrhoea, 5 others had non cyclical abdominal pain and 2 had dyspareunia.

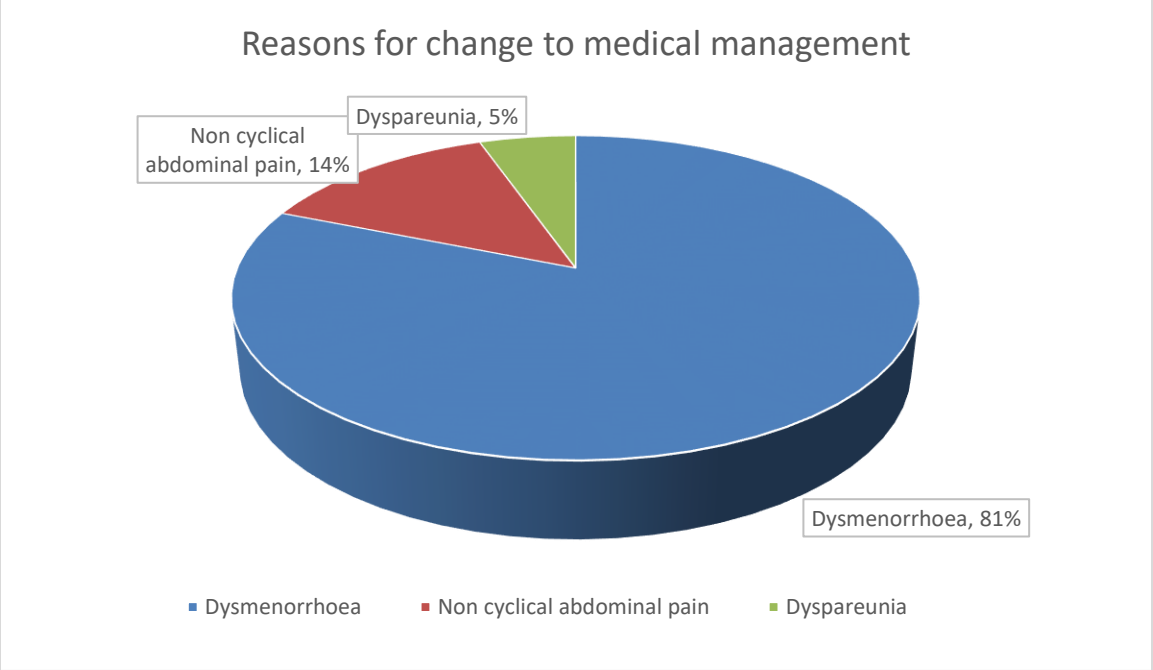


FIG.18: NEED FOR CHANGE IN MEDICAL MANAGEMENT

3)Need for change in surgical therapy:

Out of the 61 women who had change to surgical management 25 of them had dysmenorrhea, 9 of them had non cyclical abdominal pain, 3 of them had dyspareunia, while 24 others had complaints of combined dysmenorrhea and non cyclical abdominal pain.

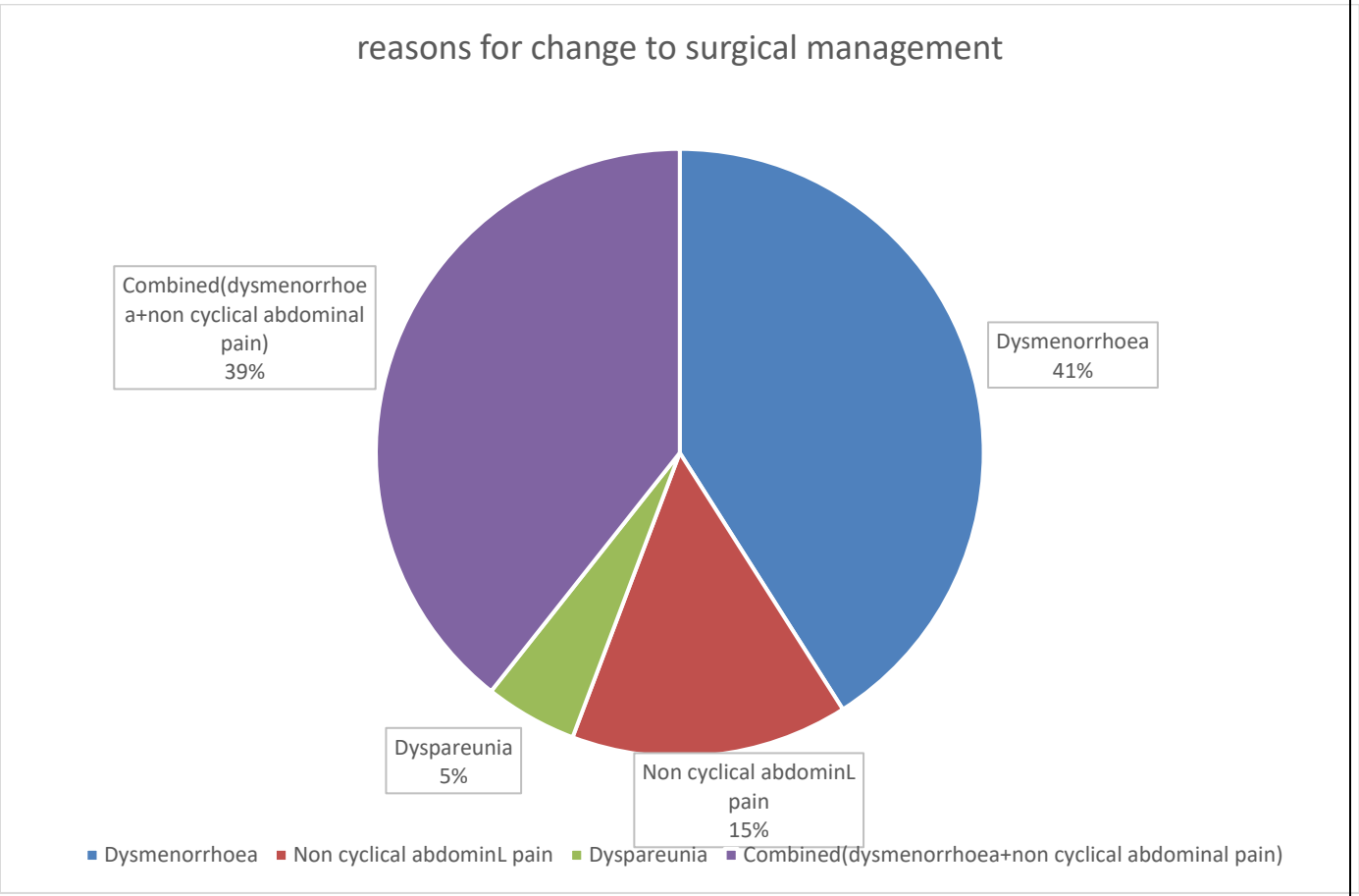


FIG.19: NEED FOR CHANGE IN SURGICAL MANAGEMENT

DISCUSSION

MEND study- Medical management in endometrioma study was done retrospectively to review the various medical managements of ovarian endometrioma practiced commonly in CMCH Vellore from the year 2017 to 2019. The various demographic prevalences and symptomatology of the patients were reviewed and compared with studies done elsewhere to see any variations.

The various demographics of our patients are discussed below:

In our study sample the mean age group of the women diagnosed with endometriosis ranged from 29 to 39 years with an average age of 32.78years. In a study done in U.S.A [55] the mean age of diagnosis of endometrioma was 25 to 29 years, which is similar to our study. Ovarian endometrioma affects about 17 to 44 % of women with endometriosis[26].

A study done in Iran[6] on association of risk factors with diagnosis of endometrioma states that early menarche, late menopause, women with symptoms of dysmenorrhoea, dyspareunia and chronic pelvic pain, increasing age, low socio economic status, nulliparous, underweight, family history of endometriosis, prolonged menstrual flow and short cycle interval are more prone for endometrioma. Moreover in another study done by hemmings et al[7] on the risk

factors associated with maximum prevalence for endometrioma was noticed in patients with infertility 55%, pelvic mass 47% and chronic pelvic pain 46%. According to our sample size population of 300, 64.6% had complaints of dysmenorrhoea, 30.6% had complaints of non-cyclical abdominal pain, 17.6 % had heavy menstrual blood loss and 8.33% had history of dyspareunia.

In our study the mean range of body mass index(BMI) of the women with endometrioma was 21 to 26 kg/m2 and the mean BMI was 24.5kg/m2. In similar comparison to our study, another study done by ferrero et al[56] in 2015 shows the mean body mass index of the study group with endometrioma was 18.55 to 24.99kg/m2.

Women with early menarche and late menopause are prone for endometrioma. Early menarche refers to onset of menstrual cycle at age less than or equal to 11 years of age, in some studies the onset is less than or equal to 12 years of age[57]. In a study done by Nnoahom et al in 2015[57] there was slightly increased risk for endometrioma with early menarche. However it was not statistically significant. Based on the principle of retrograde menstruation early menarche is explained by the prolonged exposure to menstrual cycle and retrograde transfer of endometrial glands and stroma leading to endometrioma. Out of the 300 sample size in our study about 61% of the sample size had attained menarche between 13 to 15 years

of age, the mean age of attaining menarche was 12.8 years. Only 1% of the sample population underwent early menarche around 10 to 12 years of age.

The other factors with association with endometrioma noticed in the study are 73% of the study population were married and sexually active. 41% of the population in this study had 1 child and 32.3% of the population were barren. In a meta-analysis study done by wei et al in 2015[58] showed that menstrual cycle length smaller than or equal to 27days had increase in risk for endometriosis and similarly menstrual cycle length larger than or equal to 29 decreases the risk for endometriosis. Out of 300 women in our sample size, 91.3% (267) of the women had regular menstrual cycle, while 4.3% (13) had frequent menstrual cycles and 4.3% (13) had infrequent menstrual cycles. Menstrual irregularity of frequent small cycles and prolonged days of menstruations' can be explained similarly by retrograde menstruation theory[58].

Primary objective of our study was to assess the efficacy of medical management in terms of regression of size of ovarian endometrioma. Various medical management in use are 1)Extended cycle Oral contraceptive pills- loette, ovral-L and ovral-G, 2)Injection. DMPA, 3)Dienogest and 4)LNG- IUS.

Out of the 300 women with endometrioma in our study, 260 (86.6%) women received extended cycle oral contraceptive pill as the first line of medical management. Followed by 24 (8%) received inj.DMPA, 6 people (2%) received dienogest and 10 people (3%) received LNG IUS as first line medical management in the study population. There has been no similar studies done to assess the efficacy of different medical management. Total of 300 women who received medical management in CMCH vellore only 188 had come for followup.

Among the followup patients, variations for any change in menstrual cyclicity and bleeding patterns were looked for and compared to prior menstrual cycle.

A)84.5% (159) of the women in the followup group of size of 188 had regular menstrual cycle, 2% (4) had frequent cycles, 13.2% (25) had infrequent menstrual cycles.

B)82% (154) of the women presented on followup with breakthrough bleeding, 12% (23) of them presented with withdrawal bleeding and 6% (11) of them presented with amenorrhoea.

Patients with breakthrough bleeding were compared with their first line medical management. [59]A study done on patients on extended spectrum OCP's but not diagnosed for endometrioma, reported breakthrough bleeding in 24 % of women

which gradually decreased to 4% during the next 3months of extended cycle OCPs. There are not many similar studies done on the prevalence of breakthrough bleeding on using various medical managements for endometrioma. In our study out of the 154 people with breakthrough bleeding 133 (86%) patients were on extended cycle OCPs, 12 (8%) were on inj.DMPA and 3% were on Mirena and Dienogest respectively.

In our study patients, 96% (288) were diagnosed with endometrioma by ultrasound abdomen, followed by 2% (6) by MRI, 1.3% (4) by CT abdomen and 0.6% (2) Intra operatively. However intraoperative diagnosis is the gold standard for diagnosis if endometrioma, but invasive procedure can lead to spread of the disease.

In case of unilateral endometrioma there is left sided predominance, it can be explained by the difference in the anatomy of body which includes sigmoid colon in the left side leading to deposition of the retrograde endometrial implants. In a study done by sznurkowski 2003 among women with one sided endometrioma in a population of 180 women, 62.8% were on left side and only 37.2% on the right side. Similarly in our study 69% (113) of the endometrioma were found on left side among the total sample size of 300 patients.

Among 188 patients who were on followup, 14 had not repeated ultrasound during followup, hence totally 174 people with repeat ultrasound imaging out of which

79% (138) had reduction in size of the endometrioma, 19% (32) had increase in size of endometrioma, 2% (4) had no change in size of endometrioma. Out of the 138 people who had reduction in size of endometrioma, 13 (6.9%) had complete resolving of the cyst.

1) Extended cycle OCP's- 154 received it as their first line of treatment, out of which 106 of them had reduction in cyst size of 68.8%. The mean reduction in size of endometrioma in using Extended cycle OCP was 1.9cm.

2)Inj. DMPA- 21 received it as their first line of treatment, out of which 13 of them had reduction in cyst size of 61.9%. The mean reduction in size of endometrioma in using inj.DMPA was 1.62cm.

3)Dienogest- 5 received it as their first line of treatment, out of which 3 of them had reduction in cyst size of about 60%. The mean reduction in size of endometrioma in using dienogest was 2.33cm.

4)Mirena - 4 received it as their first line of treatment, out of which 3 of them had reduction in cyst size of 75%. The mean reduction in size of endometrioma in using Extended cycle OCP was 1.6cm

5)Combined therapy: 25 people had taken combined treatment, it included two or three drugs including extended cycle OCPs group, inj.DMPA group, Novelon, danazol, cyclical loette, novex and cyclical ovral-L.

Secondary objective of our study is to assess the need for change in management - medical or surgical in regression of size of endometrioma. Out of the 188 women who were on followup, 98 had change in management. 37 had change over to other medical management and 61 had underwent surgical management.

	No of women with change in management	% of change in management
Change in management combined:	98/188	52.1%
Changed to medical management:	37/98	37.7%
Changed to surgical management:	61/98	62.2%

Among those who change in medical management –

Medical managements	% change to medical managements Total= 37 women
Extended cycle OCPs group	78% (29)
Inj.DMPA	24% (9)
Dienogest	2.7% (1)
Mirena	5.4% (2)

Among those who underwent surgery-

Medical managements	% change to surgical managements Total= 61women
Extended cycle OCPs group	68.8% (42)
Inj.DMPA	19.6% (12)
Dienogest	6.5% (4)
Mirena	3.3% (2)

Reason for change over to medical management among the 98 women was 81% due to dysmenorrhea, followed by 13.5% due to non cyclical abdominal pain and

5.4% due to dyspareunia. Among the 61 women who underwent surgical management 40.98% had dysmenorrhea, 14.75% of them had non cyclical abdominal pain, 4.9% of them had dyspareunia, while 39.3% others had complaints of combined dysmenorrhea and non cyclical abdominal pain. Further mean size of endometrioma for change in management was calculated, for change in medical management it was 3.89cm and for change over to surgery it was 7.15cm.

CONCLUSION:

1. Extended cycle OCP's had 68.8% reduction in size of endometrioma with mean reduced size of endometrioma being 1.9cm.
2. Inj. DMPA had 61.9% reduction in size of endometrioma with a mean reduced size of endometrioma being 1.62cm.
3. Dienogest had 60% reduction in size of endometrioma with a mean reduced size of endometrioma being 2.33cm.
4. Mirena had 75% reduction in size of endometrioma with a mean reduced size of endometrioma being 1.6cm
5. All medical managements compared for efficacy in terms of reduction of endometrioma size came as similar to each other.
6. Need for change over to medical management - 81% due to dysmenorrhea, followed by 13.5% due to non cyclical abdominal pain and 5.4% due to dyspareunia.
7. Need for change over to surgical management - 40.98% due to dysmenorrhea, 14.75% had non cyclical abdominal pain, 4.9% had dyspareunia, while 39.3% others had complaints of combined dysmenorrhea and non cyclical abdominal pain

LIMITATIONS:

1)Necessary information for Performa was not available from some charts of patients due to incomplete documentation.

2)122 people were lost for follow-up due to which sample size reduced.

3)14 patients who were on follow-up did not have ultrasound done, hence were excluded from final analysis.

4)Similar study should have been performed prospectively for better significant findings due to COVID pandemic -time constraints was not able to do so.

ANNEXURE -I

PERFORMA – MEND Study

1. **S.No.** :
2. **Patients name** :
3. **H No** :
4. **Age** :
5. **Address & Contact No:**
6. **BMI** :
7. **Menarche** :
8. **Marital status** :
9. **Sexually active** :
10. **Parity** :
11. **Prior cycles**

a.Cyclical (range<15days, 15-21days, 22-35days, 35-45days, more than 45days)
b. Days of bleeding
c. Material used(pads, cloth)
d. Number of pads/day

12. **Current menstrual cycles, Do you have monthly and regular cycles?**

a. Cyclical (range<15days, 15-21days, 22-35days, 35-45days, more than 45days)
b. Days of bleeding
c. Material used(pads, cloth)
d. Number of pads/day

13. Did you have endometrioma(endometrioctic cyst) ? (Diagnosed by USG abdomen/CT abdomen/MRI abdomen)

- No
- Yes

What was the:

Size of endometrioma at diagnosis	Menstrual pattern and cycle at diagnosis	Pain Score in NRS pain scale at diagnosis

13. **How was it diagnosed? (Diagnosed by USG abdomen/CT abdomen/MRI abdomen/Surgery)**

14. **What were your symptoms?**

15. **Name the medication prescribed in first?**

Extended cycle OCPs	
Inj.DMPA	
Dinogest	
LNG IUS	

16. **How long was that treatment given?**

17. **What was the:**

Size of endometrioma after treatment	Menstrual pattern and cycle after treatment	Pain Score in NRS pain scale after treatment

18. **What was the next prescribed medication?**

19. **How was it effective than before?**

Size of endometrioma after treatment	Menstrual pattern and cycle after treatment	Pain Score in NRS pain scale after treatment

20. Did you need surgical management of endometriosis?

- No
- Yes

if Yes, Why? How was it effective than before?

Size of endometrioma after treatment	Menstrual pattern and cycle after treatment	Pain Score in NRS pain scale after treatment

ANNEXURE-II

MASTERSHEETS

s1no	TYPBLE	name	hospno	age	address	bmi	menar	marita	sexact	parity	pcycle	pbleed	pmate	ppads	ccycle	cbleed	cmate	cpads	ovaren	endosi	Diff in	endosi
1	2	Samima aktar	759920G	25	shibganj, wes	25	10	1	1	1	3	5	1	4	3	5	1	4	1	1	0.2	4.1
2	2	Sarbani pal	748928G	22	west bengal,	17.2	13	1	1	1	3	5	1	3	3	5	1	3	1	2	0.7	2.5
3	1	Dorathi.J	809322C	23	Vellore, 9843	22.2	12	2	2	0	3	3	1	3	3	3	1	3	1	1	-2.1	6.7
4	2	Farhana khan	364591G	27	West bengal	27.1	14	1	1	0	3	4	1	3	3	4	1	3	3	1	-1.8	9
5	1	Rupa kedia	334698G	36	West bengal,	27.3	13	1	1	1	3	4	1	3	3	4	1	3	2	1	-3.3	6.3
6		MST.Fatima k	779615G	23	Bangladesh, 1	20.3	12	1	1	0	3	3	1	3	3	3	1	3	1	1		4.4
7	2	Papiya khatur	297133G	23	West bengal,	23.2	14	1	1	1	3	3	1	4	3	4	1	4	4	1	-1.2	4.5
8	1	Kaberi mukhe	726335G	32	West bengal,	23.5	13	1	1	0	3	3	1	2	3	3	1	2	1	1	-2.4	5.6
9	2	Priyanka das	761243G	23	West bengal,	28.4	14	2	2	0	3	4	1	4	3	4	1	4	1	1	-1.3	6.2
10	2	Gouri ghosh	041638F	38	West bengal,	30	12	1	1	1	5	3	1	4	5	3	1	4	1	2	-1	10
11	1	Anita keshri	624153G	37	Jharkhand 98	28	13	1	1	1	3	3	1	4	3	3	1	4	1	1	0.2	4.2
12	2	Chandra perv	332347G	32	Ranchi,jharkh	21.9	13	1	2	0	3	3	2	1	3	3	2	1	1	1	0	6
13	1	Archana mait	747665G	37	West bengal	26	14	1	1	2	1	2	1	3	1	2	1	3	1	1	-3.1	7.6
14	2	Ruma rani bas	632346G	35	West bengal,	22.3	13	1	1	1	2	6	1	3	2	6	1	3	1	1	2	8
15	2	ruchi kumari	774840G	22	Jharkhand, 80	18.48	12	2	2	0	3	5	1	4	3	5	1	4	1	1	8.9	6
16	1	Sarita devi	727475F	35	Jharkhand, 88	28.6	13	1	1	2	3	2	1	2	3	2	1	2	1	1	-1.3	5.6
17	2	Moumita gole	737032G	20	West bengal,	17.4	15	2	2	0	3	4	1	3	3	4	1	3	1	2	-1.4	6.2
18	2	Archana mani	754445G	45	West bengal	20.3	13	1	1	2	3	4	1	4	3	10	1	4	1	1	-1.9	5.1
19	1	Gouri kumari	765466G	26	Jharkhand, 96	23	13	2	2	0	3	5	1	3	3	5	1	3	1	2	2	3.2
20	1	Sonchita sark	766893G	32	Bangladesh, 9	24.5	14	1	1	1	3	5	1	4	3	5	1	4	1	2	-2.4	7.2
21	2	Saraswati san	767235G	28	West bengal	20.4	13	1	1	1	3	4	1	4	1	10	1	4	2	1	-1.5	6
22	2	Kalpana.M	542982G	20	Ambur, 95665	19.7	13	2	2	0	3	4	1	4	3	4	1	4	1	1	-1.6	7.4
23	2	Kavitha.A	178856G	36	Chandragiri, A	27.9	12	1	1	1	3	4	1	3	2	7	1	6	1	1	-1	9
24	2	rajeswari	203471C	40	Chetpet, tami	23.7	15	1	1	3	3	4	1	3	3	4	1	3	1	2	0.4	4.8
25	2	Sangita devi	774831G	42	Bihar, 943062	23.1	14	1	1	2	3	6	1	4	3	6	1	4	1	2	-0.6	5.8
26	2	Susan Jehang	213220B	24	Bangladesh	20	12	1	1	1	2	3	1	3	3	3	1	3	1	1	-1.2	5.2
27	2	Anju manara	162385G	31	Bangladesh, f	22.3	14	2	2	0	3	6	1	3	3	6	1	3	1	2	1	4
28	2	Nasrat Parvee	479379f	25	Bangladesh	28.1	14	1	1	1	3	5	1	3	3	5	1	5	1	1	-0.8	4.4
29	2	Kajol mukhar	767764G	31	Bangladesh	28.3	14	1	1	1	3	5	1	4	3	5	1	8	1	1	-5.7	7.8
30	2	gurusha kuma	437500G	28	jharkhand, 91	18.6	13	2	2	0	3	5	1	3	3	5	1	3	1	2	-2.1	7.3
31		prabhati deba	768503g	41	west bengal	24.2	13	1	1	2	3	3	1	3	3	3	1	3	1	1		5.4
32		shreeja shree	119365g	23	jharkhand, 70	21	13	1	1	2	3	3	1	3	3	3	1	2	1	1		5.7
33	2	agnes anna m	119365g	40	west bengal, 9	28	13	1	1	1	3	3	1	3	3	3	1	3	1	1	-1.3	4.3
34		manashi sark	751479g	41	west bengal, 9	24	13	1	1	1	3	5	1	6	3	5	1	6	1	1		2.4
35	2	SAARFUNNES	084629H	21	West Bengal _	18	12	2	2	0	3	7	1	2	3	7	1	2	1	2	-3.2	5.3
36	2	KAMALIKA JA	044186H	36	WEST BENGA	28.1	13	1	1	2	3	4	1	3	3	6	1	5	1	2	0.3	3.7
37	2	TANUSRI SAU	649104F	35	West Bengal _	24.4	13	1	1	0	3	6	1	4	3	6	1	4	1	1	0.6	4
38	2	urmila kumar	237021F	20	jharkhand _9	21.6	15	2	2	0	3	6	1	2	3	10	1	2	1	1	8	3.6
39	2	SARASWATI D	909013D	33	Bihar _94313	22.6	12	1	1	2	3	3	1	3	3	3	1	3	1	1	-4	4
40	2	SOMA SEN	905830G	36	West Bengal _	31.6	12	1	1	2	3	7	1	4	3	7	1	4	1	1	2	6.4
41	2	SPANDITA M	052311C	26	Tamil Nadu _9	28	10	2	2	0	3	10	1	4	4	10	1	4	1	1	-0.2	4
42		RATNA BHAT	014308H	31	Bihar _89869	25.3	12	1	1	1	2	8	1	5	5	8	1	5	1	1		2.9
43	1	SAMIMA AKT	759920G	24	Bangladesh _9	29.1	10	1	1	1	3	5	1	4	3	5	1	4	1	1	-0.9	5
44	2	ANJU MAN AI	162385G	31	Bangladesh	22.3	14	2	2	0	3	7	1	3	3	7	1	3	1	1	2	3
45	2	PAPIYA KHAT	2971335	34	West Bengal	22.9	14	1	1	1	3	6	1	5	3	6	1	5	1	1	3.1	4.7
46	2	ANITHA. S	773726G	36	TIRUPATTUR	27	12	2	2	0	3	3	1	2	3	3	1	2	1	1	3	5.6
47	2	ARUNA RANI	410224G	38	Bangladesh	25.11	13	1	1	1	3	5	1	6	3	5	1	6	1	1	-1.6	4.3
48	1	JAYANTI KUM	646012G	42	NEPAL	23	15	1	1	2	3	5	1	4	3	5	1	4	1	2	-0.3	5.1
49	1	SALMA PARVI	936467F	38	Bangladesh	23.9	12	1	1	1	3	3	1	3	3	3	1	3	1	2	1.1	4.5
50	2	NITU KUMAR	775817G	21	West Bengal	21.9	12	2	2	0	5	3	1	1	5	3	1	1	1	1	1.2	3.9
51	1	NANDITA BIS	795506G	40	West Bengal	23.8	12	1	1	1	5	4	1	4	3	4	1	3	1	2	2.1	4.6
52	1	PREETHI GIRI	792315G	40	Andhra Prade	26	12	1	1	1	3	5	1	3	3	5	1	3	1	2	-1.6	4.8
53	2	SAMPIKA DAS	765686G	41	West bengal	20.2	13	1	1	1	3	5	1	4	3	3	1	4	1	2	3.1	4.2
54	2	BANDANA NA	256367F	39	TAMIL NADU	23.2	13	1	1	1	4	5	1	4	4	5	1	4	1	2	-0.7	4.3
55	2	SWARNALATA	803618G	30	TRIPURA	15.1	14	2	1	0	2	2	1	2	2	2	1	2	1	2	2.8	7.6
56	2	MITALI PATRA	284270G	41	West bengal	28	14	1	1	0	3	4	1	2	3	4	1	2	1	2	6	4.2
57	2	MITALI CHAK	776468G	40	West Bengal	31	12	1	1	1	3	3	1	3	3	3	1	3	1	1	-0.6	3.6
58	2	ASHRU ADAK	807402G	31	West Bengal	28.15	12	1	1	2	3	6	1	2	3	6	1	2	1	1	0.5	4.5
59	2	CHHANDA KO	185642C	37	West Bengal	28.1	12	1	1	0	3	8	1	5	3	8	1	5	1	1	6	5
60		SWETA MAHA	796134G	30	West Bengal	25.4	12	1	1	1	3	3	1	5	3	5	1	5	1	1		3.1
61	1	MIDYA KRISH	800351G	42	West Bengal	27.4	12	1	1	2	3	6	1	3	3	10	1	3	1	1	-1.5	5
62	2	SUMA RANI S	208219G	27	BANGLADESH	28	13	1	1	0	3	4	1	3	3	4	1	3	1	1	-0.7	3.3
63		SAHALI SAHA	806390G	18	WEST BENGA	20	12	2	2	0	3	6	1	3	3	6	1	3	1	1		4.5
64	2	CHITRA DAS	960721D	35	JHARKHAND	23.8	12	1	1	1	3	3	1	3	3	3	1	3	1	2	1.2	5.6
65		NURZAHAN P	867918G	49	BANGLADESH	36.7	13	1	1	2	4	4	1	3	4	4	1	3	1	1		6.6
66		ANJALI DEY	676337D	45	WEST BENGA	26.4	13	1	1	2	2	4	1	2	2	4	1	2	1	2		6
67	2	AMANDA DEE	875430G	26	MAHARASTRA	17	11	2	2	0	3	4	1	2	3	4	1	2	1	2	0.7	8.9
68		TASLIMA	858817G	31	BANGLADESH	23.6	12	1	1	2	3	3	1	3	3	3	1	3	1	1		3.2
69		RUMI AKTAR	874147G	29	BANGLADESH	27.5	15	1	1	2	3	3	1	3	3	3	1	3	1	1		4.5
70		ANITHA ROSE	895901G	35	TAMIL NADU	28.6	12	1	1	2	3	4	1	3	3	10	1	3	1	1		3.4
71	2	MAMATA SIN	830177G	39	WEST BENGA	27.2	12	1	1	2	3	3	1	4	3	3	1	4	1	1	3	3.2
72	2	HARI PRIYAM	302296G	30	WEST BENGA	34.9	12	1	1	1	3	3	1	2	3	10	1	2	1	2	-0.7	5.7
73	2	RINKU PAL	214880G	26	WEST BENGA	21.5	14	1	1	2	3	3	1	2	3	6	1	5	1	1	1	5.2
74		TAKRIMA HAI	904487G	38	BANGLADESH	17.8	13	1	1	1	3	3	1	2	3	5	1	2	1	1		4.3
75	2	ANTARA MON	911008G	23	WEST BENGA	20.2	12	2	2	0	3	3	1	3	3	5	1	5	1	1	-1.2	6
76	2	KEYA DAS GU	445237C	32	BANGLADESH	14.5	14	1	1	0	2	3	1	2	2	3	1	2	1	1	-0.3	3.3
77	2	SHILA MISHR	905754G	30	WEST BENGA	22	13	1	1	1	3	3	1	2	3	6	1	4	1	1	1.9	3.4
78	2	KAJALI MAND	267639G	30	WEST BENGA	26.4	13	1	1	1	3	3	1	3	3	3	1	3	1	1	-1.8	5.2
79		SOMASREE M	917721G	32	WEST BENGA	32.8	12	1	1	1	3	3	1	3	3	3	1	3	1	2		6.8
80	2	SOMA SEN	905830G	36	WEST BENGA	31.6	12	1	1	2	3	3	1	2	3	7	1	4	1	1	1.3	5.1
81	2	SIMA DUTTA	908529G	42	WEST BENGA	25	12	1	1	1	3	3	1	2	3	5	1	3	1	2	0.7	6.3
82	2	REKHA DEVI	919355G	39	BIHAR	32	14	1	1	3	3	4	1	3	5	5	1	5	1	2	0.8	5.5
83	2	RADHA	505580C	38	TAMIL NADU	27	12	1	1	0	3	3	1	3	5	3	1	4	1	1	0.9	5
84	2	MUSALI ANUS	922551G	27	ANDHRA PRA	21	12	2	2	0	3	2	1	2	3	2	1	2	1	2	-0.9	5.5
85		GEETHA V	586641B	26	KANCHIPURA	28	13	2	2	0	3	3	1	2	3	9	1	3	1	1		2.5
86		ASIMA NAND	874667G	34	WEST BENGA	28	12	1	1	2	5	6	1	5	5	6	1	5	1	1		3
87	2	ARCHANA SA	912492G	28	WEST BENGA	22	10	1	1	1	3	3	1	2	3	10	1	2	1	2	0.6	4.6
88	2	HERSCHEL RE	917105G	18	KARNATAKA	21	12	2	2	0	3	4	1	3	3	4	1	3	1	1	-1.2	11.2
89	2	VIMALA T	193843C	40	TAMIL NADU	23	12	1	1	2	3	4	1	2	5	4	1	5	1	1	-0.8	3
90		THENMOZHI	903734G	39	TIRUVANAMA	20	13	1	1	1	3	4	1	2	3	15	1	3	1	1		3.5
91	2	SOWMIA	836530B	25	TIRUVANAMA	21	14	2	2	0	3	2	1	3	3	2	1	3	1	1	-4.6	4.6
92		ARCHANA D	942758G	25	VELLORE	22	13	2	2	0	3	5	1	3	3	5	1	3	1	1		6.1
93		FARHANA KH	364591G	28	BANGLADESH	24	14	1	1	0	3	4	1	3	3	4	1	3	1	2		3.7
94		BITHI PAUL	877116G	37	WEST BENGA	23.5	12	1	1	0	3	5	1	2	2	9	1	5	1	2		6.6
95	2	PIYALI DEY SA	920669G	32	WEST BENGA	24	11	1	1	1	3	3	1	2	3	3	1	2	1	1	-7.8	7.8
96	2	BANDANA NA	256367F	39	VELLLORE	24	12	1	1	1	3	3	1	3	3	3	1	3	1	2	-4.3	4.3
97		VANITHA	196456C	18	VELLORE	18	12	1	1	2	3	5	1	2	3	5	1	2	1	1		4
98		ALPANA SANT	965840G	24	WEST BENGA	20	12	1	1	2	3	3	1	3	3	5	1	3	1	2		5.8
99	2	Dr. Shabnam	137351D	39	Hospital Anne	29.8	12	1	1	2	5	4	1	4	5	10	1	4	1	1	-0.7	4.2

mcycle	mbleed	dysme	hmbl	abdom	dyspar	dysche	othsyn	cycleo	ecocpy	injdmp	dinoge	lngius	othdru	durme	endosi	endosi	matcyc	matble	treatch	medm	meddu	surgma	manage
3	5	1	2	2	2	2		1	3	2	2	2		9	1	4.3	5	5	2				ECO-L
3	5	1	2	2	2	2		2		2	2	2	Novelo	3	2	3.2	3	5	1	1	9	2	EC0-L
3	3	1	2	2	2	2		1	3	2	2	2		9	1	4.6	5	5	2				ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		3	1	7.2	3	4	1			1	Lap.cystec
3	4	1	2	2	2	2		1	3	2	2	2		9	1	3	5	4	2				ECO-L
3	3	1	2	1	2	2		1	3	2	2	2		9									
3	3	2	1	2	2	2		1	3	2	2	2		24	1	3.3	4	5	1	1			Mirena
3	3	1	2	2	2	2		1	1	2	2	2		12	1	3.2	5	3	2				ECO-L
3	4	1	1	2	2	2		1	3	2	2	2		36	1	4.9	3	4	2				ECO-L
5	3	1	1	2	2	2		1	3	2	2	2		3	2	9	4	4	1	1	12		DMPA
3	3	1	1	2	2	2		1	3	2	2	2		9	1	4.4	5	5	2				ECO-L
3	3	2	2	2	2	2	mass P	2		1	2	2	Leupro	12	1	6	2	3	1			1	Lap.cystec
1	2	1	1	2	2	2		1	1	2	2	2		12	1	4.5	5	2	1	1			Mirena
2	6	1	2	2	2	2		2		2	1	2		3	1	10	3	3	1			1	Lap.SO
3	5	2	2	1	2	2		2		2	1	2		12	1	14.9	3	5	1			1	Lap.cystec
3	2	1	2	2	2	2		1	3	2	2	2		12	1	4.3	3	3	2				ECO-L
3	4	1	2	2	2	2		2		2	2	2	Novelo	3	2	4.8	3	4	1	1			ECO-L
3	10	2	1	1	2	2		1	3	2	2	2		12	1	3.2	5	6	2				ECO-L
3	5	1	2	1	2	2		1	3	2	2	2		24	2	5.2	5	5	2				ECO-L
3	5	2	2	1	2	2		1	3	2	2	2		9	2	4.8	5	5	2				ECO-L
2	10	1	1	2	2	2		1	3	2	2	2		3	1	4.5	3	5	1	1			DMPA
3	4	2	2	1	2	2		1	3	2	2	2		9	1	5.8	3	4	2				ECO-L
2	7	1	1	2	1	2		1	1	2	2	2	Leupro	12	1	8	3	3	1			1	TAH+BSO
3	4	1	2	1	2	2		1	3	2	2	2		3	2	5.2	3	4	2				ECO-O
3	6	2	2	1	2	2		1	3	2	2	2		6	2	5.2	3	6	2				ECO-L
3	5	1	2	2	2	2		1	3	2	2	2		9	1	4	3	5	2				ECO-L
3	6	1	2	2	2	2		1	3	1	2	2		24	2	5	3	4	1			1	Lap.cystec
3	5	2	1	2	2	2		1	3	2	2	2		12	1	3.6	3	5	2				ECO-L
3	5	1	2	1	2	2		1	3	2	2	2		9	2	2.1	3	5	2				ECO-L
3	5	2	2	1	2	2		1	3	2	2	2		12	2	5.2	3	5	2				ECO-L
3	3	1	2	2	2	2		1	3	2	2	2		9									
3	3	2	2	1	2	2		1	3	2	2	2	Leupro	12									
3	3	2	2	1	2	2		1	3	2	2	2		12	1	3	3	3	2				ECO-L
3	5	2	1	2	2	2		2		2	2	1		60									
3	7	1	2	2	2	2		1	3	2	2	2		9	2	2.1	3	7	2				ECO-L
3	6	2	1	2	2	2		1	3	2	2	2		4	2	4	3	6	1			1	TAH+USO
3	6	1	2	2	2	2		1	3	2	2	2		9	1	4.6	3	6	2				ECO-L
3	10	1	2	1	2	2		2		2	2	2	Danazo	24	1	11.6	3	6	1			1	Lap.cystec
3	3	1	2	1	2	2		1	3	2	2	2		9	1	0	3	3	2				ECO-L
3	7	1	1	2	2	2		1	3	2	2	2		2	1	8.4	3	7	1			1	Lap.cystec
4	10	1	1	2	2	2		1	3	2	2	2		9	1	3.8	4	4	2				ECO-L
5	8	1	1	2	1	2		1	3	2	2	2		9									
3	5	1	2	2	2	2		1	3	2	2	2		9	1	4.1	5	5	2				ECO-L
3	7	1	2	2	2	2	Clot	1	3	1	2	2		12	1	5	3	7	1			1	Lap.cystec
3	6	2	1	2	2	2		1	3	2	2	2		9	1	7.8	3	3	1	1			mirena
3	3	1	2	2	2	2		1	1	2	2	2		3	1	8.6	3	3	1			1	Lap.cystec
3	5	1	2	2	2	2		1	3	2	2	2		9	2	2.7	5	5	1	1			mirena
3	5	1	2	2	2	2	INTER	1	3	2	2	2		9	2	4.8	5	5	1	1			Mirena
3	3	1	2	2	2	2		1	3	2	2	2		9	2	5.6	5	3	2				ECO-L
5	3	1	2	1	2	2		1	3	2	2	2		6	1	5.1	3	1	2				ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		6	2	6.7	5	3	1			1	Lap.cystec
3	3	1	2	2	2	2	Clots	1	3	2	2	2		6	2	3.2	5	5	2				ECO-L
3	3	1	2	2	2	2		2		2	2	2	Cyclica	6	2	7.3	3	3	1			1	TAH+BSO
3	5	2	2	1	2	2		1	3	2	2	2		12	2	3.6	3	5	2				ECO-L
2	2	2	2	1	2	2		2		2	2	2	Cyclica	6	2	10.4	2	2	1			1	Lap.cystec
3	4	2	2	1	2	2		2		2	2	2	Cyclica	6	2	10.2	3	4	1			1	TAH+BSO
3	3	1	2	1	2	2		1	3	2	2	2		12	1	3	3	3	1	1			mirena
3	6	2	2	1	2	2		2		2	2	2	Cyclica	6	1	5	3	6	1			1	Lap.cystec
3	8	1	2	2	2	2		1	3	2	2	2		6	1	11	3	8	1			1	TAH+BSO
3	5	2	1	2	2	2		1	3	2	2	2		6									
3	10	2	1	2	2	2		1	3	2	2	2		8	1	3.5	5	6	2				ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		6	1	2.6	3	4	2				ECO-L
3	6	1	2	2	2	2		1	3	2	2	2		6									
3	3	1	2	2	1	2	POST C	1	3	2	2	2		9	1	6.8	3	3	2				ECO-L
4	4	1	2	2	2	2		2		1	2	2		3									
2	4	1	2	1	2	2	WHITE	2		2	2	2	Novex	6									
3	4	1	2	1	2	2		1	3	2	2	2		8	2	9.6	3	4	2				ECO-L
3	3	1	2	1	1	2		1	3	2	2	2		6									
3	3	2	1	1	2	2		1	3	2	2	2		6									
3	10	2	1	2	2	2		1	3	2	2	2		6									
3	10	1	2	2	1	2		2		1	2	2	Novex	6	1	6.2	3	10	1			1	TAH+BSO
3	3	1	1	2	2	2		1	3	2	2	2		6	2	5	3	3	1	1			Mirena
3	6	1	1	2	2	2	WHITE	1	3	2	2	2		3	1	6.2	3	10	1			1	Lap.cystec
3	5	1	2	1	2	2		1	3	2	2	2		6									
3	5	1	2	2	2	2		1	3	2	2	2		6	1	4.8	3	3	2				ECO-L
2	3	1	2	1	2	2		1	3	2	2	2		3	1	3	3	4	2				ECO-L
3	6	1	2	1	2	2		1	3	2	2	2		6	1	5.3	3	6	2				ECO-L
3	3	1	2	2	2	2		1		2	2	2	ECO-O	8	1	3.4	3	3	2				ECO-Ovral
3	4	2	2	1	2	2		1	3	2	2	2		6									
3	7	1	1	1	2	2	Abdom	1	3	2	2	2		6	1	6.4	3	7	1	2		1	Lap.cystec
3	5	1	2	2	2	2		2		2	2	2	Cyclica	3	2	7	3	5	1	2		1	TAH+BSO
5	5	2	1	2	2	2		1	3	2	2	2		6	2	6.3	3	5	2				ECO-L
5	3	1	2	2	2	2		1	3	2	2	2		6	1	5.9	3	4	2				ECO-L
3	3	1	2	2	2	2		1	3	2	2	2		6	2	4.6	3	3	2				ECO-L
5	6	1	2	2	2	2		1	3	2	2	2		6									
5	6	1	2	2	2	2		1	3	2	2	2		6									
3	10	2	2	1	2	2		1	3	2	2	2		24	2	5.2	3	5	1	1			Mirena
3	4	2	2	1	2	2		1	3	2	2	2		9	1	10	3	4	1	2		1	Lap.cystec
4	4	2	1	2	2	2		1	3	2	2	2		9	1	2.2	3	4	1	1		2	Mirena
3	15	2	2	2	2	2		2		2	2	1		6									
3	20	1	2	2	2	2		1	3	2	2	2		9	1	0	3	3	2				resolved
3	5	1	2	2	2	2		1	3	2	2	2		6									
3	4	1	2	2	2	2		1	3	2	2	2		6									
2	9	1	2	2	2	2		1	3	2	2	2		6									
3	3	1	2	2	2	2		1	3	2	2	2		6	1	0	3	3	2				resolved
3	3	2	2	1	2	2		1	3	2	2	2		12	2	0	3	3	2				resolved
3	5	1	2	2	2	2		1	3	2	2	2		6									
3	5	1	2	2	2	2		1	3	2	2	2		2									
5	10	1	1	2	2	2		1	3	2	2	2		9	1	3.5	3	6	2				ECO-L

100	2	SALMA PARV	936467F	37	BANGLADESH	23.9	12	1	1	1	3	3	1	3	3	3	1	3	1	1	-2.4	4.5
101	2	PAYAL DEY	955427G	23	WEST BENGA	18	13	2	2	0	3	6	1	4	3	6	1	4	1	2	-0.4	5.4
102	2	Nirma nidhi x	921698g	29	Jharkhand	27.46	11	2	2	0	3	5	1	5	3	5	1	5	3	2	-0.5	4.7
103	2	Sangita ghosh	719159g	23	west bengal	18	13	2	2	0	3	3	1	2	3	3	1	2	1	1	-0.8	3.4
104	2	preethi giridh	792315g	40	Andhra pradh	26	14	1	1	1	3	5	1	3	3	5	1	3	1	2	-1.6	4.8
105		Adwitya kar	971508g	27	west bengal	26	11	2	2	0	3	3	1	3	3	3	1	3	1	1		3.7
106	2	sree vithya M	673401c	30	vellore	31.1	12	1	1	2	3	2	1	4	3	2	1	4	1	1	-3.3	3.3
107	2	martha das	968064g	37	jharkand	30.5	13	2	2	0	3	5	1	3	3	5	1	3	1	1	-4.9	4.9
108	2	chhaya das	977959g	29	west bengal	17.8	13	1	1	2	3	3	1	2	3	3	1	2	1	1	-1.3	4.7
109	2	ritu parna	659808f	22	west bengal	16	11	2	2	0	3	3	1	3	3	3	1	3	1	1	2.3	4.1
110	2	priyanka mait	311926f	34	west bengal	21.3	13	1	1	0	3	3	1	3	3	3	1	3	1	2	0.2	4.2
111	2	Menaka maha	924940g	37	west bengal	25.8	12	1	1	1	3	4	1	5	3	4	1	5	1	1	-0.3	3.6
112	2	tapasi Mahat	651095g	41	west bengal	25.7	13	1	1	1	3	3	1	3	3	3	1	3	1	2	-3.3	3.3
113		Parul dolai	173335f	43	west bengal	26.5	14	1	1	2	4	5	1	4	4	5	1	4	1	1		4.6
114		mitali patra	284270g	41	west bengal	28	14	1	1	0	3	4	1	3	3	4	1	3	1	1		4
115	1	chaitali bera	957027g	44	west bengal	30	11	2	2	0	3	3	1	3	3	3	1	3	1	1	-1	5
116	3	susmita saha	001838h	35	west bengal	24.7	13	1	1	1	3	3	1	4	2	5	1	4	1	2		6.4
117	1	nitu kumari	775817g	21	west bengal	21.9	12	2	2	0	3	3	1	2	5	3	1	1	1	1	1.2	3.9
118	2	pampa dey	612262g	37	west bengal	24.3	13	1	1	1	3	4	1	3	3	4	1	3	1	2	-0.2	7.2
119	2	kasturi kundu	960359g	37	west bengal	29.4	11	1	1	1	3	3	1	3	4	3	1	3	1	2	2.8	7
120		sanubba naaz	135601g	26	west bengal	24.6	13	1	1	2	3	2	1	3	3	4	1	3	1	2		3.3
121		fatema johor	013387h	33	uttara, bangla	24.8	13	1	1	1	4	5	1	4	4	5	1	4	1	2		4.2
122		farhana islam	628162d	37	bangladesh	21.5	13	1	1	1	2	10	1	2	2	10	1	2	1	2		6.9
123		solema bibi	016241h	41	west bengal	24.8	13	1	1	2	3	3	1	3	3	3	1	3	1	2		2.2
124		rakpi angu	086465h	21	arunachal pra	25.8	13	2	2	0	3	2	1	2	3	2	1	2	1	1		3.5
125		nitu singh	054171h	43	bihar	27.4	17	1	1	1	3	3	1	4	3	3	1	4	1	1		8.4
126		hari priyaman	302296g	30	west bengal	34.9	13	2	1	1	3	10	2	6	3	10	2	6	1	1		5
127	2	sora rani mon	025275h	41	west bengal	27	14	1	1	2	3	3	1	3	3	3	1	3	1	1	-0.5	4.7
128	1	seema tirkey	079742h	34	chattisgarh	21	14	2	2	0	3	3	1	3	3	3	1	3	1	1	-2	4
129		chamon afrog	115074h	39	bangladesh	31.4	13	1	1	2	3	3	1	2	3	3	1	2	1	1		5.7
130		gomathi.v	108006c	40	tamil nadu	30.6	13	1	1	2	3	5	1	2	3	5	1	2	4	1		6.7
131		krishna bera	126879h	35	west bengal	30.2	13	1	1	1	3	3	1	3	3	3	1	3	1	1		3.5
132	2	antara manda	059705h	24	west bengal	22	13	2	2	0	3	3	1	3	3	3	1	3	1	1	-1.9	4.9
133	2	manisha das	073086h	38	west bengal	27.1	14	1	1	1	3	4	1	3	3	4	1	3	1	2	-2	8
134		suraiya khana	145771h	28	chittagong, ba	21	14	1	1	1	3	3	1	2	3	3	1	2	1	2		5.5
135	2	siuli dutta	593827g	38	west bengal	30.3	13	1	1	1	3	3	1	3	3	3	1	3	1	2	-3	3
136		piyali das	149513h	35	assam	21	13	1	1	1	2	12	1	3	2	12	1	3	1	1		4.5
137		mahuya patha	101592h	38	west bengal	28.3	11	1	1	1	3	3	1	2	3	3	1	5	1	1		5.8
138		sandipa sen	116448h	37	west bengal	28.1	18	1	1	2	3	5	1	3	3	5	1	3	1	1		5
139		monika. k	712526d	22	andhra prade	30.7	12	2	1	1	3	5	1	3	3	5	1	4	2	1		5
140	2	payal dey	955427g	23	west bengal	22	13	2	2	0	3	3	1	3	3	3	1	3	1	2	-2.5	5.4
141		ragasudha	134234h	27	tamilnadu	22.4	13	1	1	2	3	4	1	3	3	4	1	3	1	1		8
142		saarfunnessa	084629h	21	west bengal	24	12	2	2	0	3	3	1	3	3	3	1	3	1	2		2.1
143		sharmistha ch	173062h	39	west bengal	20	13	2	2	0	3	3	1	3	3	3	1	3	1	1		6.2
144		lovely rani ha	162019h	32	bangladesh	24.5	12	1	1	1	3	3	1	3	3	3	1	3	1	1		2.9
145	2	selima khatur	142570h	34	west bengal	30	10	2	2	0	3	3	1	3	3	3	1	5	2	1	-3.5	7.5
146	2	kajali mandal	267639g	29	west bengal	28	13	1	1	1	3	3	1	2	3	3	1	2	1	1	-2	5.4
147		debjani maha	194351h	36	west bengal	28.1	14	1	1	1	3	3	1	4	3	3	1	4	1	2		3.8
148	2	rinku pal	214880g	37	west bengal	20.2	13	1	1	2	3	5	1	8	4	15	1	8	1	1	-2	6
149	2	tripti biswas	108652h	35	west bengal	27.3	13	1	1	2	3	4	1	5	3	4	1	5	1	1	-1	4
150	2	rinku pandit	117952h	40	west bengal	23.7	13	1	1	1	3	3	1	3	3	3	1	3	1	1	-1	4
151	2	jyoti kumari	154482h	26	bihar	18.9	13	2	2	0	3	4	1	3	3	4	1	3	1	1	-1.9	4.2
152	1	arpita roy	636137d	31	west bengal	25	13	2	2	0	5	4	1	3	3	4	1	3	1	2	-0.3	3.5
153		midya krishna	800351g	42	west bengal	27.5	13	1	1	2	3	5	1	3	3	5	1	3	1	1		3.2
154	3	sudhanthira p	906983d	37	vellore, tamil	23.7	13	1	1	1	3	3	1	3	3	3	1	3	1	2		8
155		nirmala kiran	148496h	44	tamilnadu	25	13	1	1	2	3	3	1	4	3	3	1	4	1	2		7.6
156	2	champa nath	187162h	43	west bengal	27.1	12	2	1	1	3	4	1	4	3	4	1	4	1	1	-2	5
157	3	sumitra ghosh	035347f	40	west bengal	24	14	1	1	2	2	5	1	2	3	4	1	4	1	2		2.1
158	3	hari priyaman	302296g	27	tamil nadu	32	13	1	1	2	4	5	1	4	4	5	1	4	1	2		4.6
159	3	priya chakrab	320078h	23	west bengal	24.4	14	1	1	1	3	6	1	4	3	6	1	4	1	2		8.5
160	2	supriya dey	997647d	33	west bengal	25.1	13	1	1	2	3	4	1	4	3	4	1	4	1	2	1.5	4.8
161		manisha das	324958h	29	assam	19.4	13	2	2	0	3	3	1	4	3	3	1	4	1	1		6.2
162	2	bhavana m	323052h	26	tamil nadu	24	14	2	2	0	3	5	1	6	3	5	1	6	3	1	-3	5
163		sarita shaw	685898f	34	west bengal	25.4	14	1	1	1	3	2	1	2	3	2	1	2	1	2		3.3
164	2	abeda bibi	324961h	32	west bengal	14	14	1	1	1	3	4	1	4	3	4	1	4	1	2	-1.2	3.4
165	2	soma das	185126f	34	west bengal	24	14	1	1	0	3	4	1	5	3	4	1	5	1	2	-1.9	3.1
166		megala m	935587d	35	tamilnadu	18.1	13	2	2	0	3	5	1	3	3	5	1	3	1	2		5.2
167	2	mousumi sah	309956h	23	west bengal	17.8	12	2	2	0	3	4	1	2	3	4	1	2	1	1	-2.1	8.3
168	2	chung dukpa	211680h	31	west bengal	24.3	14	2	2	0	3	4	1	2	3	4	1	2	1	1	-3.2	7.3
169	2	dharani k	438381f	41	tamilnadu	30.1	14	2	2	0	3	5	1	2	3	5	1	2	1	1	0.8	5.2
170		babita devi	344890h	32	jharkhand	18.9	14	1	1	1	2	1	1	2	2	2	1	2	1	1		2.7
171		bhabani pacc	943124g	24	west bengal	22.9	13	1	1	0	3	3	1	1	3	2	1	1	1	2		5.6
172	2	chitra	326647h	38	vellore, tamil	30.4	13	1	1	2	3	4	1	3	3	4	1	3	1	2	-2	7
173	2	farhana dipti	374674g	32	bangladesh	26.3	13	1	1	1	2	2	1	2	2	2	1	2	1	2	-1.2	3.5
174		sampa monda	295586h	26	west bengal	25	11	1	1	1	3	5	1	3	3	5	1	3	1	1		3.6
175		basanti saha	048495g	23	bihar	21.4	13	1	1	1	3	4	1	2	3	4	1	2	1	2		3.5
176	2	lakshmi chatt	729125f	21	west bengal	33.7	13	1	1	1	3	4	1	5	3	4	1	5	1	2	-0.7	13.7
177	1	chamon afrog	115074h	40	bangladesh	30.5	11	1	1	1	3	3	1	5	3	3	1	5	1	1	-2.9	5.9
178	2	susmita akthe	373428h	28	bangladesh	20.7	13	1	2	0	3	4	1	4	3	4	1	4	1	1	0	10
179	1	sudhanthira p	906983d	37	tamilnadu	23.7	11	1	1	1	3	3	1	3	3	3	1	3	1	1	-3.4	5.4
180	3	sova rani mor	025275h	41	west bengal	26	14	1	1	2	3	3	1	2	1	3	1	2	1	1		4.7
181	2	rehana sultan	363868h	39	bangladesh	34.9	13	1	1	1	3	5	1	5	3	5	1	5	1	2	-1.9	4.8
182	2	swarnali paul	396194h	18	west bengal	19	13	2	1	0	3	4	1	3	3	4	1	3	1	1	-0.4	12.4
183		manisha patr	308872h	33	west bengal	33.1	13	1	1	1	3	5	1	2	3	5	1	2	1	2		6.6
184		sharifa yasmi	402409h	35	khulna, bangl	24.2	13	2	2	0	3	3	1	4	3	3	1	4	1	1		7.5
185		silpi chowdhu	361267h	42	nagaland	25.9	12	2	2	0	3	7	1	2	3	7	1	2	1	2		16.8
186	2	afroja bibi mo	399457h	40	west bengal	24.7	15	1	1	1	3	4	1	5	3	4	1	5	1	2	-1.7	5.7
187	2	alpana podda	404740h	38	west bengal	23	11	1	1	1	3	3	1	3	3	3	1	3	1	1	-2	4.7
188		rajashree maj	396020h	23	west bengal	23	12	2	2	0	5	7	1	4	5	7	1	4	1	1		7.8
189	2	lovely rani ha	162019h	32	westbengal	21	12	1	1	1	3	4	1	2	3	4	1	2	1	1	-4.5	7.4
190	2	menaka maha	924940g	37	west bengal	25.6	12	1	1	1	3	4	1	5	3	4	1	5	1	2	-0.3	3.6
191	2	sova sharma	685261g	43	kolkata	21.2	13	1	1	1	3	3	1	1	3	3	1	1	1	1	-1.5	5
192	2	rajashree maj	396020h	23	west bengal	22.4	13	2	2	0	5	7	1	4	5	7	1	4	1	1	-0.8	7.8
193	2	mithu monda	527400c	40	west bengal	27.2	13	1	1	1	5	4	1	2	5	4	1	2	1	1	-4.1	4.1
194	2	rubi samanta	403684c	44	west bengal	18	12	1	1	1	3	3	1	2	3	3	1	2	1	2	-6.1	6.1
195	2	jabeen m	899171f	33	tamilnadu	19.9	14	1	1	1	3	4	1	5	3	4	1	5	1	2		5
196	2	rimpa manda	216214h	18	west bengal	15.1	15	2	2	0	3	7	1	2	3	7	1	2	1	1	0	3.5
197	2	esther rai	377395h	35	west bengal	28	14	1	1	2	3	4	1	6	3	4	1	6	3	1	0	2.9
198		nursama khat	513940h	35	west bengal	25.1	12	2	2	0	3	7	1	4	5	4	1	4	1	2		9.5
199	2	salma akter	510234h	33	bangladesh	24.8	13	1	2	1	3	7	1	5	3	7	1	5	1	1		4.8

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3	6	2	2	1	2	2		1	3	2	2	2		6	2	5	3	6	2				ECO-L
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3	5	2	2	2	2	2	backac	1	3	2	2	2		9	1	0	3	5	2				ECO-L
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4	5	2	2	2	2	2		1	3	2	2	2		9									
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3	3	1	2	2	2	2		1	3	2	2	2		24	2	7	3	3	1	2		1	TAH+BSO
3	3	2	2	1	2	2		1	3	2	2	2		6	2	9.8	3	3	1	2		1	Lap.cystec
3	3	2	2	1	1	2		1	3	2	2	2		12									
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3	3	2	2	1	2	2		1	3	2	2	2		9									
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3	4	1	2	1	2	2		1	3	2	2	1		9									
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3	3	2	2	1	2	2		1	3	2	2	2		9									
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3	4	1	2	1	1	2		2		1	2	2	Novex	9	1	3	3	3	1	1			Mirena
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3	4	1	2	2	2	2		2		1	2	2	Novex	3	1	2.3	3	3	1	1	6		inj. dmpa
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3	3	2	2	2	2	2		1	3	2	2	2		9									
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3	3	1	2	2	2	2		1	3	2	2	2		9									
3	4	2	2	1	2	2		1	1	2	2	2		9	2	2.2	3	3	2				ECO-OL
3	4	1	2	2	2	2		1	3	2	2	1		9	2	1.2	3	4	1	1			mirena
3	5	1	2	1	2	2		2	2	1	2	2		9									
3	4	1	2	2	2	2		1	3	2	2	2		3	1	6.2	3	4	1			1	Lap.cystec
3	4	1	2	2	2	2		1	3	2	2	2		9	1	4.1	3	4	2				ECO-L
3	5	1	2	1	2	2		2	3	2	2	2		3	1	6	3	3	1			1	TAH+BSO
2	2	2	1	1	2	2		1	3	2	2	2		9									
3	2	2	2	2	2	2		1	3	2	2	2		9									
3	4	1	1	2	2	2		1	3	2	2	2		3	2	5	3	3	1	1			mirena
2	2	2	2	1	2	2		1	3	2	2	2		12	2	2.3	3	4	2				ECO-L
3	5	1	2	2	2	2		1	3	2	2	2		9									
3	4	1	2	2	2	2		1	3	2	2	2		9									
3	4	1	2	2	2	2		2	3	2	2	1		3	1	13	3	4	1			1	Lap.cystec
3	3	2	2	1	2	2		1	3	2	2	2		12	1	3	5	3	2				ECO-L
3	4	2	1	2	2	2		1	3	2	2	2		3	1	10	3	3	1			1	Lap.cystec
3	3	1	2	2	2	2		1	3	2	2	2		12	1	2	5	3	2				ECO-L
1	3	2	1	2	2	2		1	3	2	2	2		3	1	4.2		0	2				ECO-L
3	5	2	2	2	1	2		2		1	2	2		9	2	2.9	3	3	2				ECO-L
3	4	2	2	1	2	2		2		1	2	2		3	1	12	3	4	1			1	lap.cystect
3	5	2	2	2	2	2		1	3	2	2	2		9									
3	3	1	2	2	2	2		1	3	2	2	2		9									
3	7	2	2	1	2	2		1	3	2	2	2		9									
3	4	2	2	2	2	2		1	3	2	2	2		12	1	4	2	3	1	1			Mirena
3	3	1	2	1	1	2		1	3	2	2	2		9	1	2.7	3	3	1	1	12		mirena + ir
5	7	1	2	2	2	2		1	3	2	2	2		9									
3	4	1	2	2	2	2		1	3	2	2	2		9	1	2.9	3	4	2				ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		9	2	3.3	3	4	1	1			Mirena
3	3	1	2	2	2	2		1	3	2	2	2		9	1	3.5	3	4	2				ECO-L
5	7	1	2	2	2	2		2		2	1	2		3	1	7	5	5	1			1	Lap.cystec
5	4	2	2	2	1	2		2		1	2	2	Novex	3	1	0	3	3	1	1	12		Inj.DMPA
3	3	1	2	2	2	2		1		1	1	2		6	2	0	5	5	1	1	6	2	ECO-L
3	4	1	2	2	2	2		1	3	1	2	2		9									
3	7	1	2	2	2	2		1	3	2	2	2		9	1	3.5	3	7	2				ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		12	1	2.9	3	4	1			1	TAH+BSO
5	4	1	2	2	2	2		1	3	2	2	2		9									
3	7	1	2	2	2	2		1	3	2	2	2		12									

200	2	sumitra ghosh	035347f	39	west bengal	31.2	14	1	1	2	3	4	1	4	3	4	1	4	1	2	-0.5	3.6
201		chandra kala	665862h	36	bhutan	26.9	13	1	1	2	3	8	1	4	3	8	1	4	1	2		4.2
202	2	Tina	816184h	27	Tamil nadu	28.6	13	1	1	1	3	3	1	2	3	3	1	1	1	1	-1.8	4.2
203	3	hamlin i	633708c	40	kanyakumari	27.3	13	1	1	1	3	3	1	3	3	3	1	3	1	1		4.1
204	2	shahnaz parve	473364h	31	west bengal	25.8	12	1	1	2	3	3	1	2	3	3	1	2	1	1	-3.5	3.5
205	2	sangeeta kum	544275h	22	bihar	22.4	13	1	1	1	3	20	1	2	3	3	1	2	1	1	-1.4	11.4
206	2	ramak bano	438725h	22	west bengal	22.1	14	2	2	0	3	5	1	5	3	5	1	5	1	1	0	10.9
207		dona addya	243635d	21	west bengal	24.7	10	2	2	0	3	3	1	5	3	3	1	5	1	2		4.1
208		ananika saha	493928h	43	west bengal	27.2	13	1	1	1	3	3	1	3	3	3	1	3	1	1		2.4
209	2	sampa banerj	160709h	39	jharkhand	28.7	13	1	1	1	4	3	1	4	4	3	1	4	1	1	-2.5	6.1
210		jenitha angela	382240h	27	madurai, tam	21	13	2	2	0	3	3	1	2	4	3	1	2	1	2		4.7
211	2	payel dey	955427g	23	west bengal	18.6	12	2	2	0	3	6	1	4	3	6	1	4	1	1	-1	5
212	2	akshar banu k	333685g	42	tamil nadu	28.1	13	1	1	2	3	10	1	6	3	10	1	6	1	1	0.2	6.3
213		rita viswas	545692h	33	west bengal	27.5	13	1	1	2	3	6	1	1	3	6	1	1	1	1		10
214		pratima das	4375214	45	west bengal	24	13	1	1	1	3	5	1	5	3	5	1	5	1	2		5
215	2	alpana podda	404740h	38	west bengal	23	11	1	1	1	3	3	1	3	3	3	1	3	1	1	-2	4.7
216		rina ghosh	539261h	38	west bengal	23	11	1	1	1	3	3	1	3	3	3	1	3	1	1		4.7
217		Anita	773647h	28	tamil nadu	27	13	1	1	1	3	3	1	1	3	3	1	1	1	1		4
218	2	Pushpa chettr	30529D	31	sikkim	24	14	1	1	2	3	5	1	2	3	5	1	1	1	2	-0.6	3.6
219		rinku bangal	4950414	30	west bengal	28	13	1	1	2	3	3	1	2	3	3	1	2	1	1		4.8
220		annwesha mo	586888h	25	west bengal	23.4	11	2	2	0	3	5	1	9	3	5	1	9	1	1		9.4
221		Faiza banu	681523c	30	Bangladesh	26	14	1	1	2	3	5	1	1	3	3	1	1	1	2		3.2
222	3	bharti tekwar	551709h	38	jharkhand	22	13	1	1	2	3	5	1	5	3	5	1	5	1	2		2.6
223		moumita das	575886h	43	west bengal	33.1	13	1	1	1	3	4	1	3	3	4	1	3	1	1		3.8
224	1	chung dukpa	211680h	28	west bengal	24.3	14	2	2	0	3	4	1	2	3	4	1	2	1	1	-7.3	7.3
225		thenindra the	626846g	28	tamil nadu	23	14	1	1	2	3	3	1	2	3	3	1	2	1	1		2.3
226		Sulka biswas	731077h	28	west bengal	24	13	1	1	2	3	4	1	2	3	4	1	2	1	2		4.2
227	2	arutchelvi p	188159f	32	cuddalore, tar	31.1	13	1	1	1	3	6	1	2	3	6	1	2	1	1	-0.4	8.4
228	2	nitu kumari	902714g	22	bihar	18.7	10	2	2	0	3	3	1	2	4	3	1	2	1	1	-1	3.8
229	2	biva rani guha	546677h	45	west bengal	26.8	13	1	1	1	3	4	1	3	3	5	1	5	1	1	-1.3	5.5
230	2	tamalama	436049h	36	west bengal	21	14	1	1	1	3	5	1	2	3	5	1	2	1	1	-3.4	6.8
231	2	jabeen m	899171f	33	arni thiruvana	19.9	14	1	1	1	3	15	1	5	3	15	1	5	1	2	-0.3	9.3
232		NUR NAHAR A	870918h	27	Bangladesh	21	13	1	1	0	3	3	1	1	3	3	1	1	1	2		4.1
233		priyanka barn	611513h	21	west bengal	16.8	13	1	1	0	3	5	1	5	3	5	1	5	1	2		2.4
234		happy khanar	618677h	31	bangladesh	22.6	12	1	1	1	3	3	1	3	3	3	1	3	1	1		4.9
235		sampa dey	614306h	29	west bengal	22.1	12	1	1	1	3	5	1	2	3	5	1	2	1	1		3.6
236		anarkali s	604205h	34	krishnagiri	30.9	13	1	1	2	3	3	1	3	3	3	1	3	1	2		6.4
237		jobymol jame	562819h	36	kerala	26.5	14	1	1	2	3	6	1	4	3	6	1	4	1	2		6.3
238		MOBINA	104575C	29	bangladesh	22.4	14	1	1	3	3	3	1	1	3	3	1	1	3	2		3.6
239		mst. asma ul	145557h	39	meherpur	31.8	13	1	1	2	3	5	1	4	3	5	1	4	3	2		6.1
240	1	tapasi dutta	632577h	22	west bengal	15.84	11	2	2	0	3	4	1	2	3	4	1	2	1	1	-2.1	8.6
241		nilima ghosh	135700h	40	west bengal	29.4	11	1	1	1	3	5	1	2	3	5	1	2	1	2		3.6
242		priyanka biswa	392510h	30	west bengal	29	11	1	1	1	3	3	1	4	3	3	1	4	1	2		3.4
243	2	debjani kunda	633530h	38	west bengal	25.9	11	1	1	1	3	4	1	3	3	4	1	3	1	1	-0.6	8.6
244	2	aupriya a	382242h	23	madurai, tam	24	12	2	2	0	3	3	1	2	3	3	1	2	1	2	-1.6	5.6
245	2	shreyoshi bau	243272g	22	west bengal	21.4	12	2	2	0	3	3	1	2	3	3	1	2	1	1	-1	6
246	2	afroja bibi mo	399457h	40	west bengal	24.7	15	1	1	1	3	3	1	6	3	3	1	7	1	2	-0.7	5.7
247	2	mousumi sah	309956h	23	west bengal	22	12	2	2	0	3	2	1	2	3	2	1	2	1	1	-1	8.3
248	2	piyali das	149513h	35	assam	24	13	1	1	1	3	7	1	3	2	12	1	3	1	1	-0.5	4.5
249		sindhu s	657620h	20	tamilnadu	18.8	16	2	2	0	3	2	1	2	3	2	2	2	1	2		4.1
250		lipika sarkar	307513h	28	west bengal	24	12	1	1	1	3	3	1	3	3	3	1	3	1	2		4.8
251	2	sangeeta kum	544275h	22	bihar	22.4	13	2	2	0	3	3	1	2	3	3	1	2	1	1	-6.4	11.4
252		hasina mamta	240536h	45	bangladesh	28.3	11	1	1	1	3	4	1	2	3	4	1	2	1	1		4.3
253		rinku rajak	112722d	38	west bengal	22.4	13	1	1	1	3	4	1	3	3	4	1	3	1	1		3.9
254		pakhi begum	762456h	45	bangladesh	32.9	11	1	1	3	3	4	1	2	3	4	1	2	1	1		3.7
255	2	suprava sarka	341319h	35	west bengal	20	12	1	1	1	1	7	1	2	1	7	1	2	1	1	-1	4.8
256		bharti tekwar	551709h	38	jharkhand	24	13	1	1	2	3	5	1	5	3	5	1	5	1	2		2.6
257	2	menaka maha	924940g	39	west bengal	26.3	12	1	1	1	3	6	1	8	1	3	1	6	1	2	-0.3	3.6
258	2	lipika das	760125h	24	west bengal	20.2	12	2	2	0	3	5	1	2	3	3	1	2	1	2	-0.1	6.1
259	2	thenindia the	626846g	40	chennai	24	12	1	1	2	3	4	1	2	2	4	1	2	1	1	1	4
260		laden bhutia	345317h	44	sikkim	22.8	13	2	2	0	3	3	1	3	3	3	1	3	1	2		12.4
261		celin cherian	982693g	39	thirumalaikod	22.8	13	1	1	1	3	5	1	5	3	5	1	5	1	1		2.6
262		mochammat	448180h	42	bangladesh	28.8	12	1	1	2	3	4	1	2	3	4	1	2	1	2		5.1
263	2	lipika saha	122341h	26	west bengal	22	13	2	2	0	3	5	1	4	3	5	1	4	1	1	-0.2	6.2
264	3	sudha saha	391184h	37	west bengal	23.6	12	1	1	1	3	5	1	5	3	5	1	5	1	1		3
265		dolon mal	389829g	30	west bengal	31.1	12	1	1	1	5	3	1	2	5	3	1	2	1	1		3
266	2	pampa dey	612262g	35	west bengal	24.3	12	1	1	1	3	4	1	2	3	4	1	2	1	1	0.5	4.5
267	2	anupriya a	382242h	23	madurai	21	11	2	2	0	3	4	1	2	3	4	1	2	1	2	-1.6	5.6
268	2	mahmuda akt	745555g	45	jamalpur	21.8	12	1	1	3	3	3	1	5	3	3	1	5	1	1	-3.1	13.1
269		dharani	438381f	41	vellore	30.1	14	2	2	0	3	5	1	2	3	5	1	2	1	1		5.2
270		nafeela	100119b	26	vellore	25	12	1	1	1	3	5	1	5	3	5	1	5	1	1		6.6
271		jobymol jame	643385h	36	kerala	26.5	14	1	2	2	3	6	1	4	3	6	1	4	1	2		8.8
272		anjana sharm	646685h	45	west bengal	30.5	13	1	1	2	3	3	1	2	3	3	1	2	1	2		6.3
273	3	supriya dey	997647d	33	west bengal	23.5	13	2	2	2	3	7	1	2	3	7	1	2	1	1		7
274	2	chandona sar	509715h	43	bangladesh	28	13	1	1	2	5	3	1	2	5	3	1	2	1	1	-0.4	9.4
275	2	rimpa monda	216214h	17	west bengal	27	15	2	2	0	3	5	1	3	3	5	1	3	1	1	-1.1	4.6
276	2	mst rohina ak	720487h	40	dhaka	22.5	12	1	1	1	3	3	1	2	3	3	1	2	1	1	0.4	4.6
277		esther rai	377395h	35	sikkim	28	14	1	1	1	3	4	1	6	3	4	1	6	1	2		3.9
278		mast fahema	672722h	38	bangladesh	20.5	14	1	1	1	3	6	1	3	3	6	1	3	1	1		3.6
279		rita devi	924262b	45	bihar	35.37	12	1	1	3	3	20	1	4	3	20	1	4	1	2		1.8
280		keya mandal	696843h	30	jharkhand	20.8	12	1	1	1	3	4	1	2	3	4	1	2	1	2		4
281		anita devi	680646h	42	jharkhand	28.5	12	1	1	2	3	4	1	2	3	4	1	2	1	1		5.1
282	2	shahanara be	686733h	41	bangladesh	31.1	13	1	1	2	3	3	1	4	3	3	1	4	1	2	0.7	1.8
283		celia cherian	982693g	39	vellore	22.8	13	1	1	1	3	5	1	5	3	5	1	5	1	1		2.6
284		rubi samanta	403684c	44	west bengal	21	12	1	1	0	3	4	1	2	3	4	1	2	1	2		6.1
285	1	chandana sar	509715h	43	bangladesh	20.4	12	1	1	2	5	3	1	2	5	3	1	2	1	2	-1.4	9.4
286	2	rimpa monda	216214h	18	west bengal	15.1	15	2	2	0	3	5	1	2	3	5	1	2	1	1	-1.1	4.6
287	2	shreyoshi bha	243272g	22	west bengal	21.4	13	2	2	0	3	4	1	2	3	4	1	2	1	1	-1	6
288		manju garai	714804h	26	west bengal	23.7	12	1	1	1	3	4	1	2	4	4	1	2	1	2		7
289	2	neelima t.b	577838f	23	andhra prade	22.9	11	2	2	0	3	3	1	2	3	3	1	2	1	1	2.6	2.6
290		renu singh	264732f	48	jharkhand	20.4	13	1	1	1	4	5	1	4	4	5	1	4	1	1		3
291		yeasmeen Akt	385669c	47	bangladesh	26.3	12	1	1	1	3	4	1	5	3	12	1	5	1	1		6.2
292		vijayalakshmi	156700c	35	andhra prade	24	12	1	1	2	5	3	1	3	5	3	1	3	1	2		3.9
293	2	tapasi dutta	632577h	22	west bengal	15.8	11	2	2	0	3	4	1	2	3	4	1	2	1	1	-3.6	8.6
294	2	sharmin ferdo	477999h	32	bangladesh	21.4	13	1	1	2	3	3	1	2	3	3	1	2	1	1	-7.6	12.6
295	2	mochammat	448180h	42	bangladesh	28.8	14	1	1	1	5	4	1	2	5	4	1	2	1	2	0.4	5.1
296	2	thulasi. n	082067g	40	vellore	27.2	14	1	1	2	3	5	1	4	3	5	1	4	1	1	-2.1	8.1
297		swatidruw	421984g	29	chattisgarh	25	12	2	2	0	3	8	1	3	3	8	1	3	1	1		4
298	2	anarkali s	604205h	34	krishnagiri	30.9	14	1	1	2	3	3	1	3	3	3	1	3	1	1	-2	6
299	2	manika dey	307175g	40	west bengal	24	13	1	1	2	3	4	1	2	3	4	1	3	1	2	-1	5
300		basanti saha	048495g	23	west bengal	22	12	1	1	1	3	1	1	1	3	1	1	1	1	1		3

3	4	2	2	2	2	2		1	3	2	2	2		9	2	3.1	3	4	2					ECO-L
3	8	2	2	1	1	2		1	3	2	2	2		6										
3	3	1	1	2	2	2		1	1	2	2	2		9	1	2.4	3	3	2					ECO-L
3	3	1	2	2	1	2		2		1	2	2	Novex	3	1	5.3		0	1				1	TAH+BSO
3	3	1	2	2	2	2		1	3	2	2	2		9	1	0	3	3	2					ECO-L
3	3	1	2	2	2	2		1	3	2	2	2		3	2	10	3	4	1				1	Lap.cystec
3	5	2	2	2	2	2		1	3	2	2	2		6	1	10.9	3	4	1				1	Lap.cystec
3	3	2	2	1	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
4	3	2	2	2	2	2		1	3	2	2	2		3	1	3.6	3	4	1				1	TAH+BSO
5	3	1	2	2	2	2		2		1	2	2	Novex	9										
4	6	2	2	2	2	2		1	1	2	2	2		9	1	4	3	6	2					ECO-Ovral
3	10	1	2	2	2	2		1	3	2	2	2		3	1	6.5	3	10	1				1	TAH+BSO
3	6	1	2	2	2	2		1	3	2	2	2		9										
3	5	1	2	2	2	2		1	3	2	2	2		9										
3	3	1	2	1	2	2		1	3	2	2	2		9	1	2.7	3	3	2					ECO-L
3	3	1	2	2	1	2		1	3	1	2	2		9										
3	3	1	2	1	2	2		1	3	2	2	2		6										
3	3	1	1	2	2	2		1	1	2	2	2		9	2	3	3	3	1	1	24	2		Mirena
3	3	2	2	2	1	2		2		1	2	2		12					2					
3	5	1	1	2	2	2		1	3	2	2	2		9										
3	3	1	1	2	2	2		1	3	2	2	2		9										
3	5	1	2	2	2	2		1	3	2	1	2		12	2	1.8		0	2					ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		9	1	0	5	5	2					ECO-L
3	3	1	2	2	2	2		1	1	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	6	1	2	1	2	2		1	3	2	2	2		6	1	8	3	6	1				1	Lap.cystec
5	3	1	2	2	2	2		1	3	2	2	2		9	1	2.8	3	3	2					ECO-L
3	5	1	2	1	2	2		1	3	2	2	2		9	1	4.2	3	5	2					ECO-L
3	5	1	2	2	2	2		1	3	2	2	2		9	1	3.4	3	5	1	1				Mirena
3	15	1	2	2	2	2		1	3	1	2	2		3	1	9	3	4	1				1	TAH+BSO
3	3	1	1	1	1	2		1	3	1	2	2		6										
3	5	2	2	2	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	5	2	2	1	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	6	2	2	2	1	2		2		1	2	2		9										
3	3	1	2	1	2	2		1	3	2	2	2		9										
3	5	1	2	2	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2	Inj.Lup	9	1	6.5	3	4	1	2			1	Lap.cystec
3	5	2	2	2	1	2		2		1	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		3	1	8	3	5	1				1	Lap.cystec
3	3	1	2	2	2	2		1	3	2	2	2		3	2	4	3	3	1				1	Lap.cystec
3	3	1	2	2	2	2		1	3	1	2	2		9	1	5	3	3	2					ECO-L
1	7	2	2	2	1	2		1	3	1	2	2		3	1	5	3	4	1				1	Lap.cystec
3	3	2	2	2	2	2		1	3	2	2	2		12	1	7.3	3	4	1				1	Lap.cystec
2	12	1	2	2	2	2		1	3	1	2	1		9	1	4	2	5	1	1			2	Mirena
3	2	1	2	2	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		3	1	5	3	3	1				1	Lap.cystec
3	3	2	2	1	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		9										
3	4	2	2	1	2	2		1	3	2	2	2		9										
2	7	1	1	2	2	2		2		1	2	2	Novex	9	1	3.8	3	4	1	1				Mirena
3	5	1	2	2	2	2		1	3	2	2	2		9										
1	3	1	2	2	2	2		2		1	2	2		9	2	3.3	3	3	1	1				ECO-L
4	3	1	2	2	2	2		1	3	2	1	2		3	2	6	3	3	1				1	Lap.cystec
2	4	1	2	2	2	2		1	3	1	2	2		3	1	5	3	3	1				1	Lap.cystec
3	3	2	2	1	2	2		1	3	2	2	2		9										
3	5	1	2	2	1	2		1	3	1	2	2		9										
3	4	2	2	1	2	2		1	3	2	2	2		9										
3	5	1	2	2	2	2		1	1	2	2	2		9	1	6	3	3	2		24			ECO-L
3	5	1	2	2	1	2		2		1	2	2		2	1	5		0	1				1	TAH+BSO
5	3	2	2	1	2	2		1	3	1	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		6	1	5	3	4	1				1	TAH+BSO
3	4	1	2	2	2	2		1	3	2	2	2		3	2	4	3	4	1				1	Lap.cystec
3	3	1	2	2	1	2		1	3	1	2	2		9	1	10	3	3	1	1				Mirena
3	5	1	2	1	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	6	1	1	2	2	2		1	3	1	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9										
3	7	1	1	2	2	2		2		1	2	2	Novex	3	1	7		0	1				1	TAH+BSO
5	3	2	2	1	2	2		1	3	2	2	2		3	1	9	3	4	1				1	LAVH+BSO
3	5	1	2	2	2	2		1	3	2	2	2		9	1	3.5	3	4	2					ECO-L
3	3	2	2	1	2	2		1	3	2	2	2		3	1	5	3	4	1				1	Lap.oopho
3	4	1	2	2	1	2		1	3	1	2	2		12										
3	6	2	2	1	2	2		1	3	2	2	2		9										
3	20	2	2	2	1	2		2		1	2	2		9										
3	4	1	1	2	2	2		1	3	1	2	2		9										
3	4	2	2	1	2	2		1	3	2	2	2		9										
3	3	1	2	2	2	2		1	3	2	2	2		9	1	2.5	3	4	2					ECO-L
3	5	1	2	2	2	2		1	3	2	2	2		12										
3	4	2	2	1	2	2		1	3	2	2	2		9										
5	3	1	2	1	2	2		1	3	2	2	2		3	2	8	5	4	1				1	Lap.oopho
3	5	1	1	2	2	2		1	3	1	2	2		9	1	3.5	3	4	2					ECO-L
3	4	1	1	2	2	2		1	3	1	2	2		9	1	5	3	4	2					ECO-L
3	4	1	2	2	2	2		1	3	2	2	2		9										
3	3	2	2	1	2	2		1	3	2	2	2		9	1	5.2	3	4	2					ECO-L
4	5	2	1	2	1	2		2		1	2	2	Novex	9										
3	12	1	2	2	2	2		1	3	2	2	2		9										
5	3	1	1	2	2	2		1	3	2	2	2		9										
3	4	1	2	2	2	2		1	3	2	2	2		9	1	5	3	4	1				1	Lap.cystec
3	3	2	2	1	2	2		1	3	2	2	2		3	1	5	3	4	1				1	Lap.cystec
5	4	1	2	1	2	2		1	3	2	2	2		3	1	5.5	3	4	1				1	TAH+BSO
3	5	2	2	1	2	2		1	3	2	2	2		3	1	6	3	4	1				1	LAVH+BSO
3	8	1	2	2	2	2		1	3	2	2	2		9										
3	3	2	2	1	2	2		1	3	2	2	2		9	1	4	3	4	1	1				Mirena
3	4	1	2	2	2	2		1	3	2	2	2		9	2	4	3	4	1	1				Mirena
3	4	1	2	2	2	2		1	3	2	2	2		9										

ANNEXURE- III

Permission letters for IRB

PERMISSION LETTER FOR IRB

From,

Dr.Hana Christy.T

PG Registrar

MS.Obstetrics &Gynaecology

Christian Medical College&Hospital-Vellore.

To,

Head of Unit- OG-I

Dr.Elsy Thomas

Christian Medical College&Hospital-Veliore.

Subject-Permission for reviewing hospital numbers, outpatient, inpatient view charts and radiology scan of patiernts who used drugs-Extended cycle OCP'S namely Tablet Loette and Tablet Ovtal.L and projestins like injection.DMPA and LNG-IUS(Mirena) over the years 2017, 2018 and 2019 in OG-I department patients for my Retrospective IRB study.

Dear Madam,

Title of the study: Efficacy of various medical management of Ovarian Endometrioma- A Retrospective observationsl study.

Under Guide: Dr. Vaibhav Londhe

Aim of the study is to assess the efficacy of various types of medical management of ovarian ebdometrioma in reduction of size and need for change in management –medical or surgical, among women of reproductive age group 18 to 45years who were treated in CMC Vellore Gynaecology OPD for atleast 6months from 2017 to 2019.

Thanking You,

Yours sincerely,

Dr. Hana Christy.T

10/11/2020

Signature of Head of Unit OG-I

Elsy Thomas

10/11/2020

Dr. ELSY THOMAS, MD,DGO.,
Registration No. 57501
Professor & Ag. Head
Dept. of Gynaecology (New) Unit - I,
Christian Medical College,
Vellore - 632 004, Tamil Nadu, India.

PERMISSION LETTER FOR IRB

From,

Dr.Hana Christy.T

PG Registrar

MS.Obstetrics & Gynaecology

Christian Medical College&Hospital-Vellore.

To,

Head of Department- OG-II

Dr. Lilly Varghese

Christian Medical College& Hospital-Vellore.

Subject- Permission for reviewing Hospital numbers, Outpatient ,Inpatient view charts and Radiology scan findings of patients who used drugs - Extended cycle OCP'S namely Tablet .Loette and T.Ovral.L and Projestins like Injection. DMPA and LNG-IUS (Mirena) over the yearS 2017, 2018 and 2019 in OG-II department patients for my Retrospective IRB study Sample size collection.

Dear Madam,

Title of the Study: Efficacy of various medical management of Ovarian Endometrioma- A Retrospective observational study.

Under Guide: Dr.Vaibhav Londhe

Aim of the study is to assess the efficacy of various types of medical management of ovarian endometrioma in reduction of size and the need for change in management-medical or surgical, among women of reproductive age group 18 to 45years who were treated in CMC Vellore Gynaecology OPD for atleast 6months from 2017 to 2019..

Thanking You,

Yours sincerely,

Dr. Hana Christy. T,

10/11/2020

Signature of Head of Unit OG-II

Dr. Lilly Varghese, MD.
Professor & Head, OG II
Emp. No. 30645 Regn. No. 62658
CMCH, Vellore - 632 004,
Tamil Nadu, India

BIBLIOGRAPHY:

1. Rafique S. Medical Management of Endometriosis. :12.

2. Lee N, Min S, Won S, Cho YJ, Kim M, Kim MK, et al. The recurrence rate of ovarian endometrioma in women aged 40–49 years and impact of hormonal treatment after conservative surgery. Sci Rep. 2020 Oct 5;10(1):16461.

3. Brown J, Farquhar C. Endometriosis: an overview of Cochrane Reviews. Cochrane Gynaecology and Fertility Group, editor. Cochrane Database Syst Rev [Internet]. 2014 Mar 10 [cited 2021 Feb 14]; Available from: http://doi.wiley.com/10.1002/14651858.CD009590.pub2

4. Chapron C. Management of ovarian endometriomas. Hum Reprod Update. 2002 Nov 1;8(6):591–7.

5. Valson H, Kulkarni C, Teli B, T. N. Study of endometriosis in women of reproductive age, laparoscopic management and its outcome. Int J Reprod Contracept Obstet Gynecol. 2016;514–9.

6. Ashrafi M, Akhoond MR. Evaluation of Risk Factors Associated with Endometriosis in Infertile Women. :11.

7. Hemmings R, Rivard M, Olive DL, Poliquin-Fleury J, Gagné D, Hugo P, et al. Evaluation of risk factors associated with endometriosis. Fertil Steril. 2004 Jun;81(6):1513–21.

8. Calhaz-Jorge C. Clinical predictive factors for endometriosis in a Portuguese infertile population. Hum Reprod. 2004 Jun 24;19(9):2126–31.

9. Hillier SG, Whitelaw PF, Smyth CD. Follicular oestrogen synthesis: the 'two-cell, two-gonadotrophin' model revisited. Mol Cell Endocrinol. 1994 Apr 1;100(1):51–4.

10. Hill CJ, Fakhreldin M, Maclean A, Dobson L, Nancarrow L, Bradfield A, et al. Endometriosis and the Fallopian Tubes: Theories of Origin and Clinical Implications. J Clin Med. 2020 Jun 18;9(6):1905.

11. Ugur M, Turan C, Mungan T, Kuşçu E, Şenöz S, Ağis HT, et al. Endometriosis in Association with Müllerian Anomalies. Gynecol Obstet Invest. 1995;40(4):261–4.

12. Yan L, Zhao X, Qin X. MRKH syndrome with endometriosis: case report and literature review. Eur J Obstet Gynecol Reprod Biol. 2011 Nov;159(1):231–2.

13. Samani EN, Mamillapalli R, Li F, Mutlu L, Hufnagel D, Krikun G, et al. Micrometastasis of endometriosis to distant organs in a murine model. Oncotarget. 2019 Mar 19;10(23):2282–91.

14. Jabr FI, Mani V. An unusual cause of abdominal pain in a male patient: Endometriosis. Avicenna J Med. 2014 Oct;04(04):99–101.

15. Vinatier D, Orazi G, Cosson M, Dufour P. Theories of endometriosis. Eur J Obstet Gynecol Reprod Biol. 2001 May;96(1):21–34.

16. Anastasiu CV, Moga MA, Elena Neculau A, Bălan A, Scârneciu I, Dragomir RM, et al. Biomarkers for the Noninvasive Diagnosis of Endometriosis: State of the Art and Future Perspectives. Int J Mol Sci. 2020 Mar 4;21(5):1750.

17. Hansen KA, Eyster KM. Genetics and Genomics of Endometriosis. Clin Obstet Gynecol. 2010 Jun;53(2):403–12.

18. Wenzl R, Kiesel L, Huber JC, Wieser F. Endometriosis: A genetic disease. Drugs Today. 2003;39(12):961.

19. Logan PC, Yango P, Tran ND. Endometrial Stromal and Epithelial Cells Exhibit Unique Aberrant Molecular Defects in Patients With Endometriosis. Reprod Sci. 2018 Jan;25(1):140–59.

20. Ozel L, Sagiroglu J, Unal A, Unal E, Gunes P, Baskent E, et al. Abdominal wall endometriosis in the cesarean section surgical scar: A potential diagnostic pitfall. J Obstet Gynaecol Res. 2012;38(3):526–30.

21. Gunes M, Kayikcioglu F, Ozturkoglu E, Haberal A. Incisional endometriosis after cesarean section, episiotomy and other gynecologic procedures. J Obstet Gynaecol Res. 2005 Oct;31(5):471–5.

22. Rizk B, Fischer AS, Lotfy HA, Turki R, Zahed HA, Malik R, et al. Recurrence of endometriosis after hysterectomy. Facts Views Vis ObGyn. 2014;6(4):219–27.

23. Clement PB. The Pathology of Endometriosis: A Survey of the Many Faces of a Common Disease Emphasizing Diagnostic Pitfalls and Unusual and Newly Appreciated Aspects. Adv Anat Pathol. 2007 Jul;14(4):241–60.

24. Horne A, Daniels J, Hummelshoj L, Cox E, Cooper K. Surgical removal of superficial peritoneal endometriosis for managing women with chronic pelvic pain: time for a rethink? BJOG Int J Obstet Gynaecol. 2019;126(12):1414–6.

25. Reis FM, Santulli P, Marcellin L, Borghese B, Lafay-Pillet M-C, Chapron C. Superficial Peritoneal Endometriosis: Clinical Characteristics of 203 Confirmed Cases and 1292 Endometriosis-Free Controls. Reprod Sci. 2020 Jan;27(1):309–15.

26. Carnahan M, Fedor J, Agarwal A, Gupta S. Ovarian endometrioma: guidelines for selection of cases for surgical treatment or expectant management. Expert Rev Obstet Gynecol. 2013 Jan;8(1):29–55.

27. Hughesdon PE. THE STRUCTURE OF ENDOMETRIAL CYSTS OF THE OVARY. BJOG Int J Obstet Gynaecol. 1957 Aug;64(4):481–7.

28. Brosens IA, Puttemans PJ, Deprest J. The endoscopic localization of endometrial implants in the ovarian chocolate cyst. Fertil Steril. 1994 Jun;61(6):1034–8.

29. Al-Fozan H, Tulandi T. Left Lateral Predisposition of Endometriosis and Endometrioma. :3.

30. D'Alterio MN, D'Ancona G, Raslan M, Tinelli R, Daniilidis A, Angioni S. Management Challenges of Deep Infiltrating Endometriosis. Int J Fertil Steril [Internet]. 2021 Apr [cited 2021 Nov 9];15(2). Available from: https://doi.org/10.22074/ijfs.2020.134689

31. Vercellini P, Buggio L, Somigliana E. Role of medical therapy in the management of deep rectovaginal endometriosis. Fertil Steril. 2017 Dec;108(6):913–30.

32. Foti PV, Farina R, Palmucci S, Vizzini IAA, Libertini N, Coronella M, et al. Endometriosis: clinical features, MR imaging findings and pathologic correlation. Insights Imaging. 2018 Feb 15;9(2):149–72.

33. Nezhat C, Vang N, Tanaka PP, Nezhat C. Optimal Management of Endometriosis and Pain. Obstet Gynecol. 2019 Oct;134(4):834–9.

34. Mao AJ, Anastasi JK. Diagnosis and management of endometriosis: The role of the advanced practice nurse in primary care. J Am Acad Nurse Pract. 2010 Feb;22(2):109–16.

35. Parasar P, Ozcan P, Terry KL. Endometriosis: Epidemiology, Diagnosis and Clinical Management. Curr Obstet Gynecol Rep. 2017 Mar;6(1):34–41.

36. Lee S-Y, Koo Y-J, Lee D-H. Classification of endometriosis. Yeungnam Univ J Med. 2021 Jan 31;38(1):10–8.

37. Küpker W, Felberbaum R, Krapp M, Schill T, Malik E, Diedrich K. Use of GnRH antagonists in the treatment of endometriosis. Reprod Biomed Online. 2002 Jan;5(1):12–6.

38. Key T, Pike M. The dose-effect relationship between "unopposed" oestrogens and endometrial mitotic rate: its central role in explaining and predicting endometrial cancer risk. Br J Cancer. 1988 Feb;57(2):205–12.

39. Moghissi KS, Boyce CR. Management of endometriosis with oral medroxyprogesterone acetate. Obstet Gynecol. 1976 Mar 1;47(3):265–7.

40. Köhler G, Faustmann TA, Gerlinger C, Seitz C, Mueck AO. A dose-ranging study to determine the efficacy and safety of 1, 2, and 4mg of dienogest daily for endometriosis. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2010 Jan;108(1):21–5.

41. Abou-Setta AM, Houston B, Al-Inany HG, Farquhar C. Levonorgestrel-releasing intrauterine device (LNG-IUD) for symptomatic endometriosis following surgery. Cochrane Gynaecology and Fertility Group, editor. Cochrane Database Syst Rev [Internet]. 2013 Jan 31 [cited 2021 Feb 14]; Available from: http://doi.wiley.com/10.1002/14651858.CD005072.pub3

42. Vercellini P, Viganò P, Somigliana E. The role of the levonorgestrel-releasing intrauterine device in the management of symptomatic endometriosis. Curr Opin Obstet Gynecol. 2005 Aug 1;17(4):359–65.

43. Patwardhan S, Nawathe A, Yates D, Harrison G, Khan K. Systematic review of the effects of aromatase inhibitors on pain associated with endometriosis. BJOG Int J Obstet Gynaecol. 2008 Jun;115(7):818–22.

44. Farquhar C, Prentice A, Singla AA, Selak V. Danazol for pelvic pain associated with endometriosis. Cochrane Gynaecology and Fertility Group, editor. Cochrane Database Syst Rev [Internet]. 2007 Oct 17 [cited 2021 Feb 14]; Available from: http://doi.wiley.com/10.1002/14651858.CD000068.pub2

45. Donnez J, Nisolle M, Gillerot S, Anaf V, Clerckx-Braun F, Casanas-Roux F. Ovarian endometrial cysts: the role of gonadotropin-releasing hormone agonist and/or drainage **Partially supported by Fonds de la Recherche Scientifique grant 3.4587.90, Brussels, Belgium. Fertil Steril. 1994 Jul;62(1):63–6.

46. Davis L-J, Kennedy SS, Moore J, Prentice A. Oral contraceptives for pain associated with endometriosis. Cochrane Gynaecology and Fertility Group, editor. Cochrane Database Syst Rev [Internet]. 2007 Jul 18 [cited 2021 Feb 14]; Available from: http://doi.wiley.com/10.1002/14651858.CD001019.pub2

47. Rana N, Thomas S, Rotman C, Dmowski WP. Decrease in the size of ovarian endometriomas during ovarian suppression in stage IV endometriosis. Role of preoperative medical treatment. J Reprod Med. 1996 Jun;41(6):384–92.

48. Volpi E, De Grandis T, Zuccaro G, Vista AL, Sismondi P. Role of transvaginal sonography in the detection of endometriomata. J Clin Ultrasound. 1995 Mar;23(3):163–7.

49. Kives S, Brown J, Prentice A, Deary A, Bland ES. Progestagens and anti-progestagens for pain associated with endometriosis. In: The Cochrane Collaboration, editor. Cochrane Database of Systematic Reviews [Internet]. Chichester, UK: John Wiley & Sons, Ltd; 2000 [cited 2021 Feb 14]. p. CD002122. Available from: http://doi.wiley.com/10.1002/14651858.CD002122

50. Reis FM, Coutinho LM, Vannuccini S, Luisi S, Petraglia F. Is Stress a Cause or a Consequence of Endometriosis? Reprod Sci. 2020 Jan;27(1):39–45.

51. Sroyraya M, Songkoomkrong S, Changklungmoa N, Poljaroen J, Weerakiet S, Sophonsritsuk A, et al. Differential expressions of estrogen and

progesterone receptors in endometria and cyst walls of ovarian endometrioma from women with endometriosis and their responses to depo-medroxyprogesterone acetate treatment. Mol Cell Probes. 2018 Aug;40:27–36.

52. Vignali M, Belloni GM, Pietropaolo G, Barbasetti Di Prun A, Barbera V, Angioni S, et al. Effect of Dienogest therapy on the size of the endometrioma. Gynecol Endocrinol. 2020 Aug 2;36(8):723–7.

53. Kim M, Cho Y, Seong S. The use of levonorgestrel-releasing intrauterine system for long-term maintenance after conservative laparoscopic surgery for endometriosis: in the respect of recurrent ovarian endometrioma. Fertil Steril. 2015 Sep;104(3):e167.

54. Becker CM, Gattrell WT, Gude K, Singh SS. Reevaluating response and failure of medical treatment of endometriosis: a systematic review. Fertil Steril. 2017 Jul;108(1):125–36.

55. Woodward PJ, Sohaey R, Mezzetti TP. Endometriosis: Radiologic-Pathologic Correlation. RadioGraphics. 2001 Jan 1;21(1):193–216.

56. Ferrero S, Anserini P, Remorgida V, Ragni N. Body mass index in endometriosis. Eur J Obstet Gynecol Reprod Biol. 2005 Jul 1;121(1):94–8.

57. Nnoaham KE, Webster P, Kumbang J, Kennedy SH, Zondervan KT. Is early age at menarche a risk factor for endometriosis? A systematic review and meta-analysis of case-control studies. Fertil Steril. 2012 Sep;98(3):702-712.e6.

58. Wei M, Cheng Y, Bu H, Zhao Y, Zhao W. Length of Menstrual Cycle and Risk of Endometriosis. Medicine (Baltimore). 2016 Mar 7;95(9):e2922.

59. Wright KP, Johnson JV. Evaluation of extended and continuous use oral contraceptives. Ther Clin Risk Manag. 2008 Oct;4(5):905–11.