AOGD BULLETIN

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Dedicated Issue:

Benign Gynaecological Disorders

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Message from the President

Dear AOGD Friends,

The chill of the winter is around. It's a season of festivities and the jingle of Christmas carols ushers in the New Year. The present issue is the first issue of this year and it is about benign gynecological diseases. Some of these benign diseases can be very troublesome and are more chronic in nature- life changing if not life threatening. Dealing with conditions like endometriosis, PCOS can be very frustrating but challenging as well. Experience of a life time treating such diseases is enlightening for the future generations but advent of newer modalities can be very humbling and course changing in our profession. It's only when these modalities pass the stringiest test of time and safety do they become our protocols.

I also take this opportunity to invite you all to the International Conference on Critical Care in Obstetrics to be held on 12-14th February at Hotel Grand, Vasant Kunj. I promise you all a never before experienced scientific feast, served by the most eminent names in critical care- with hands on workshops, key note addresses, talks and lively panel discussions this conference will be of great benefit *for all practicing obstetricians*. I emphasize on "All" because it is a matter of minutes before a normally progressing pregnancy/labour can become a critical and disastrous event. Saving mothers lives is of utmost importance for us and I am sure you all agree with me.

While bringing you this warmth of knowledge, I wish you all a Very Happy New Year 2016.

"*If Winter Comes, Can Spring Be Far Behind*" --Percy Shelly

Dr Pratima Mittal
President, AOGD
drpratima@hotmail.com

Congratulations !

Dr Sharda Jain on being awarded the "Eminent Woman of The Year" award by International Human Rights

Dr Sonia Malik for being awarded the PNDT Plaque of Honour & IMA Lifetime Achievement Award

Message from the Secretary

I welcome you all to another issue of AOGD bulletin and into this beautiful New Year with new resolutions, hopes and promises. We are continuing our outreach activities and CME's in full force. Two programs for sensitisation of ASHA workers for screening for cancer cervix and breast cancer were carried out by Safdarjung hospital in last week of December under Aegis of AOGD and were attended by more than 300 workers.

It is a great honour for AOGD, that Dr Alka Kriplani is now holding the prestigious post of President of FOGSI. We are going to hold "International Conference in Critical Care in Obstetrics" in collobration with FOGSI from 12th to 14th Feburary. With the participation of you all we held the AOGD conference successfully and now for this conference also, the first one of this year by FOGSI, your active participation is needed.

Benign gynaecological disorders are the most commonly encountered problems seen by the gynaecologist. I am sure this issue about their management would be appreciated by you all.

Dr Achla Batra
Hon. Secretary, AOGD
achla_batra@yahoo.com

Important

Please note, the **Next Clinical Meeting** is on Saturday 30th January, 2016 at 3.00pm, 1st Floor LT, Kalawati Saran Hospital, Lady Hardinge Medical College & SSK Hospital, New Delhi. All are cordially invited. The change in date is due to Beating the Retreat on 29th January.

AOGD Sub Committee Nomination

Nominations are invited for the post of chairperson of the following sub-committees:

- Infertility
- Multidisciplinary patient management
- Rural Health
- Cervical Cancer Awareness & Prevention
- Breast Cancer Prevention

Eligibility criteria

1. Person should be member of AOGD and have at least 10 years standing in the profession with at least 5 years duration of holding senior position in the respective institutions.
2. Chairperson of a subcommittee has to be a member of any subcommittee earlier for at least 1 year.
3. No repeat nomination will be considered after one term of two years.
4. In case of two people applying for the same post, the decision of the executive will be final.
5. In case of any deviation, the decision would be taken by executive committee.
6. Two posts cannot be held by any member at one particular time.

The nominations should reach the AOGD Secretariat: Maulana Azad Medical College (MAMC), 2 Bahadur Shah Zafar Marg, New Delhi - 110002) by post by 30th May, 2016 along with the biodata.

From the Editor's Desk

Dear AOGD Friends,

Greetings for a very happy and prosperous 2016 to all of you! We wish you a year full of purposeful academic activities and will continue our endeavour to update you all with the latest scientific advancements in the coming months.

The focus this month is on benign gynaecological disorders. The medical and surgical management of endometriosis; non surgical options for fibroids; long term consequences of PCOS; the practical approach to AUB- using the new terminology; the problem of recurrent vaginal discharge; the enigma of premenstrual syndrome; the diagnostic dilemmas in managing a patient with adnexal mass and the latest in the management of PID have been craftily elucidated by experts in the field. The safe and rationale use of blood transfusion has also been included.

Dr P Chadha, who has taught many generations the nuances of obstetrics and gynaecology, kindly consented to share her lifetime experiences with the AOGD members. Her simplicity and sincerity is clearly visible through her beautiful thoughts which have been penned in the "Luminary" column.

Our next dedicated issue for the month of February will be on Critical Care Obstetrics to commemorate the 'International Conference on Critical Care Obstetrics'. We invite you all to this first of its kind conference in the city of Delhi. It is surely going to be a great scientific feast for all those who practice obstetrics and are faced with critical situations and challenges during its course.

"For last year's words belong to last year's language
And next year's words await another voice.
And to make an end is to make a beginning." - **T. S. Eliot**

Dr Jyotsna Suri
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RECENT ADVANCES

Adopting New FIGO Nomenclature for Optimal Diagnostic Evaluation of Abnormal Uterine Bleeding

Aruna Batra
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SGT Medical College and Hospital, Budhera, Gurgaon, Haryana

Abnormal uterine bleeding (AUB) is a very common condition seen amongst women attending gynecology OPD and has considerable negative impact on the health and quality of life of affected women. Until recently various traditional terms used in practice, for describing the symptoms as well as the potential underlying causes of abnormal uterine bleeding, were inconsistently defined and confusing, complicating the approach to this problem; that led to an exhaustive review process by a multinational group of clinicians and investigators, and introduction of standard nomenclature and classification of AUB by the International Federation of Gynecology & Obstetrics (FIGO).[1] The ultimate goal of simplifying and standardizing the terminology was to improve communication among health care providers and patients to help provide evidence-based care.

Nomenclature & Classification

FIGO advocates the use of simple self-explanatory terms such as heavy menstrual bleeding (HMB), heavy & prolonged menstrual bleeding (HPMB), intermenstrual (IMB), frequent/infrequent bleeding to record the patient's history; discarding terminologies such as menorrhagia, metrorrhagia, menometrorhhagia, polymenorrhea, epimenorrhea, hypermenorrhea etc.

Acute and chronic abnormal uterine bleeding is clearly defined, "Acute AUB" being an episode of bleeding of sufficient quantity to require immediate intervention to prevent further blood loss, as distinct from "chronic AUB" that is defined as bleeding from the uterine corpus that is abnormal in volume, regularity &/or timing, and has been present for the majority of past 6 months.

Assigning the cause/s of AUB in the non-gravid women of reproductive age, has also been made simpler by the formulation of **"PALM-COEIN classification system"**; abandoning the use of terms such as functional uterine bleeding, dysfunctional uterine bleeding (DUB), metropathia hemorrhagica. Nine main categories of FIGO classification (table 1) remembered by the acronym PALM-COEIN are divided into structural and non-structural entities, depending upon whether they can/cannot be evaluated and diagnosed by imaging and/or biopsy.

Table 1: AUB – FIGO Classification (PALM-COEIN)

Structural	Non-Structural
Polyp Adenomyosis Leiomyoma Malignancy	Coagulopathy Ovulatory dysfunction Endometrial Iatrogenic Not Yet Classified

The structural group-PALM

The 'Polyp' category (**AUB-P**) includes endometrial as well as cervical polyps.

The 'Adenomyosis' category (**AUB-A**) is now diagnosed easily by imaging technology.

The 'Leiomyoma' category (**AUB-L**) is further characterized depending upon the location and size of myomas. Secondary subdivision differentiates Submucosal myomas (L_{SM}) from Others (L_O) which do not impact the endometrial cavity, because submucosal lesions are considered most likely to contribute to the genesis of AUB (Figure 1). The tertiary classification further categorizes leiomyomas based on their relationship with endometrium &/or serosa. This is most useful for those who perform resectoscopic myomectomy.

Leiomyoma subclassification system

SM - Submucosal	0	Pedunculated intracavitary
	1	<50% intramural
	2	≥50% intramural
0 - Other	3	Contacts endometrium; 100% intramural
	4	Intramural
	5	Subserosal ≥50% intramural
	6	Subserosal <50% intramural
	7	Subserosal pedunculated
	8	Other (specify e.g. cervical, parasitic
Hybrid leiomyomas (impact both endometrium and serosa)	Two numbers are listed separated bya hyphen. By convention, the first refers to the relationship with the endometrium while the second refers to the relationship to the serosa. One example is below	
	2-5	Submucosal and subserosal, each with less than half the diameter in the endometrial and peritoneal cavities, respectively.

Fig 1: Leiomyoma Sub-classification, Submucosal (L_{SM}) Others (L_O), Adapted from M.G. Munro et al. / IJGO 113 (2011) 3–13

The 'Malignancy' category (**AUB-M**) includes malignant as well as premalignant lesions. Once a woman is diagnosed with malignancy, she is further classified according to FIGO classification.

Non-structural group-COEIN

The term "DUB," previously used when there was no systemic or locally definable structural cause is abandoned. Women classified earlier in this group may have one or a combination of coagulopathy, ovulatory dysfunction, and/or an endometrial disorder with a disturbance in local endometrial hemostasis, and should be distinguished accordingly.

The 'Coagulation' category **(AUB-C)** includes the spectrum of systemic disorders of haemostasis such as von Willebrand disease and other coagulopathies which may be associated with AUB and may be overlooked during evaluation. Coagulation could also be impaired because of systemic diseases as leukemia or liver failure and use of anticoagulants/ chemotherapeutic agents.

The 'Ovulatory dysfunction' **(AUB-O)** manifests as a combination of unpredictable timing of bleeding and abnormal flow; and can occur during transition phases of adolescence or menopause, or may be associated with endocrinopathies such as polycystic ovary syndrome, hypothyroidism, hyperprolactinemia, stress etc.

'Endometrial' Causes **(AUB-E)** include a primary disturbance in endometrial hemostasis that usually presents as heavy menstrual bleeding, may be associated with deficiency in local production of vasoconstrictors (prostaglandin F2α, endothelin-1), excessive production of plasminogen activators or prostacyclin; or a secondary disturbance following abnormal inflammatory responses or chronic endometritis which may present with intermenstrual bleeding.

'Iatrogenic' **(AUB-I)** include bleeding associated with the use of intrauterine devices, hormonal contraceptives, and other agents such as antidepressants (tricyclics, selective serotonin reuptake inhibitors), antipsychotics (1st generation, risperidone), tamoxifen, corticosteroids, herbs like ginseng, chastberry which may affect ovulation or bleeding as such.

The 'Not Yet Classified' category **(AUB-N)** is reserved for ill-defined or rarely encountered conditions such as myometrial hypertrophy or arteriovenous malformation.

The classification has been accepted worldwide. It may undergo review and modification in future depending upon advancements in knowledge/technology and would need to be adopted accordingly.

Evaluation

A structured history and systematic examination (Table 2) should include the nature of bleeding, its impact on the quality of life, related symptoms that might identify potential structural or histological abnormality, and look at other important issues such as co-morbidities, future fertility/contraception plans. Screening for coagulation disorders should be done especially in acute bleeding and is considered positive if any of the following history is present: HMB since menarche, bleeding related to PPH/ surgery/dental work, two or more of frequent episodes of bruising/epistaxis/gum bleeding (once or twice a month)/ family history of bleeding symptoms.[2] Physical examination to rule out systemic diseases that can cause abnormal bleeding should be followed by gynecological examination to evaluate the pelvis and lower genital tract for any structural cause of bleeding and obtain specimen for cervical cytology and cultures if required.

Table 2: Systematic Approach to Evaluation

Structured History taking
a. Define the Nature of Bleeding
- Volume, regularity, frequency, duration of menstrual episodes
b. Identify potential pathology
- Associated pelvic pain, pressure symptoms
- Coagulopathy screening
c. Other Important Issues
- Family history
- Fertility/contraception plans
- Previous investigations & treatment
General Examination
• Vital Signs, pallor
• Weight/BMI, Thyroid examination
• Skin (bruising, striae, petechiae, hirsutism)
• P/A for mass/ viscera
Gynaecological Examination
• P/S: vulva, vagina, Cervix, urethra, anus
• P/V: uterus & adnexal structures
• P/R: if bleeding from rectum or suspected concomitant pathology
• Pap smear, Cervical cultures (if risk for STI)

Following clinical evaluation, directed investigations (Figure 2) are performed for assigning a PALM-COEIN category for accurate diagnosis of the underlying cause.

General investigations include exclusion of pregnancy in sexually active women, complete blood counts and basic coagulation tests (PT, INR, APTT) in women with heavy bleeding, and thyroid function tests only if findings are suggestive of thyroid disease.[3]

For investigating the structural cause for AUB, Transvaginal ultrasound (TVS) should be the first line imaging modality.[4] It has improved the diagnosis of endometrial polyp, submucous myoma and cystic glandular hyperplasia especially with the advent of 3D/4D USG. Magnetic resonance imaging (MRI) and Saline infusion sonography (SIS) are the second line imaging techniques that may help further in diagnosing the cause of abnormal bleeding. MRI can distinguish between leiomyomas and adenomyomas, and clearly

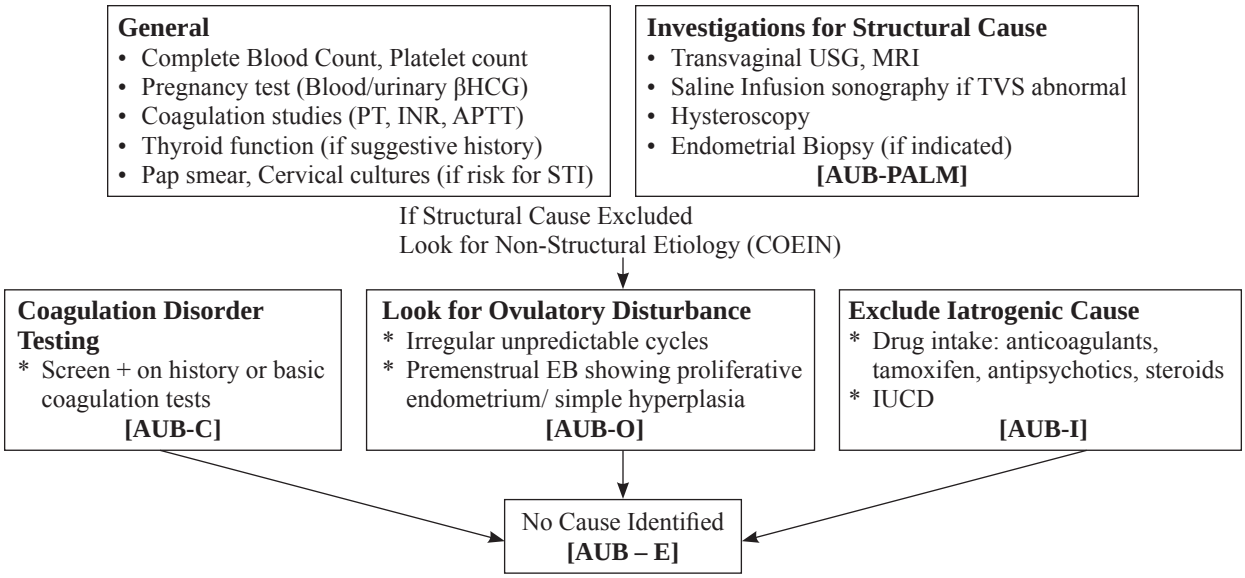

Fig 2: Investigative Work Up and Diagnosis for AUB

determine the relationship of the leiomyomas with the endometrium, myometrium, and serosa. It would have a beneficial role, if TVS findings are inconclusive or when vaginal access is difficult. It can depict the characteristics of individual fibroids and the response to treatment with non-surgical forms of therapy when uterine preservation is the goal of therapy. SIS helps in the diagnosis of intrauterine pathology by defining the location of lesion and its relationship to the uterine cavity. Addition of 3D to conventional sono-hysterography can achieve excellent results in the identification of polyps and submucous leiomyomas, and can obviate the need for MRI and hysteroscopy.

For the detection of histological abnormalities of endometrium, office endometrial biopsy is indicated in women aged 40 years and over; in younger women with factors at risk for endometrial cancer such as nulliparity, PCO, obesity, diabetes mellitus, HPNCC; or in case of failure of medical treatment. Office EB should replace D & C as the initial assessment of endometrium in AUB.[5] Hysteroscopy provides an option for direct visualization of uterine cavity and directed endometrial sampling. It provides advantage over blind D & C that may be associated with sampling errors and is essential for visualization of endometrial cavity in peri-menopausal women, when USG is inconclusive, or when focal lesion has been found on ultrasound.[6]

Having excluded the structural causes, one has to consider **Investigations for non-structural etiology (COEIN group)**. Coagulopathy, Ovulation disorder, and Idiopathic cause of abnormal uterine bleeding would have been suspected by patient's history and general investigations. Confirmation of AUB-C is made by special tests such as von Willebrand Factor, Ristocetin co-factor, and factor VIII in women who screened positive for coagulopathies on history or basic coagulation tests. Ovulation disorder as the underlying cause (AUB-O) is diagnosed in women with unpredictable episodes of bleeding with variable flow and proliferative endometrium/ simple hyperplasia on premenstrual endometrial biopsy. AUB related to endometrial dysfunction (AUB-E) is a diagnosis of exclusion when no other cause has been identified. Bleeding is generally cyclical but heavy or prolonged.

Notation for AUB

Thus, following appropriate investigations, an individual may be found to have one or more causes contributing to their symptoms. These are documented with a subscript against each category - "0" if absent, "1" if present, and "?" if not yet assessed. Since the full notation would be cumbersome in clinical practice; an abbreviation option has been developed (Figure 3).

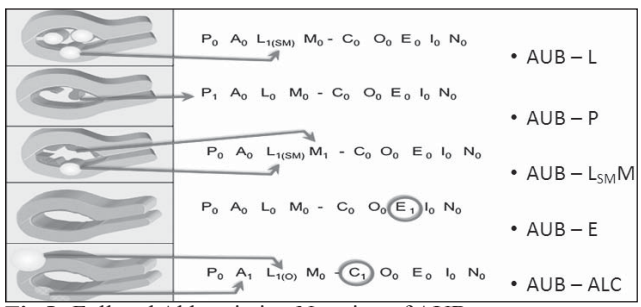

Fig 3: Full and Abbreviation Notation of AUB
Adapted from M.G. Munro et al. IJGO; 113 (2011) 3-13

Conclusion

New FIGO terminology and classification for assigning a

PALM-COEIN category to patients of abnormal uterine bleeding is a simple and practical approach that should be adopted in routine practice by all clinicians as it would help in systematic evaluation, judicious use of advanced diagnostic technology and optimized management of such patients.

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6. Van Dongen H, de Kroon CD, Jacobi CE. Trimbos JB, Jansen FW. Diagnostic hysteroscopy in abnormal uterine bleeding: a systematic review and meta analysis. BJOG 2007;114:664-75

FORTHCOMING EVENTS

1. **"Youth Mela" (**under aegis of Adolescent Health Subcommittee of AOGD) to be held on 18th January, 2016, timing: 12 to 2 pm Venue: Jagan Institute of Management Sciences.
2. "**Monthly CME of DGFS"** (under aegis of Safe Motherhood Committee, AOGD) to be held on 20th January, 2016 at Saket City Hospital. Panel Discussion on "Recurrent Pregnancy Loss", Contact: Dr Madhu Goel, M: 9810480920
3. **"Colposcopy Workshop"** at Action Balaji Hospital on 21st January, 2016, For details Contact: Dr S K Das, M: 9810059380
4. **"AOGD Monthly Clinical Meeting"** on 30th January, 2016, at 3pm at 1st Floor LT, Kalawati Saran Hospital, The meeting will be preceeded by talk on 'Art of Scientific Paper Writing' by Dr R M Pandey (2-3pm)
5. **"Annual International Conference RGCON" - 2016** to be held on 5th, 6th & 7th Feb 2016 at India Habitat Centre. ***Conference theme -"Gynae Oncology, Controversy to Consensus".*** The surgical workshop shall be held on 5th February 2016. Contact Dr. Rupinder Sekhon, rupysekhon@hotmail.com, M: 9810163076
6. **"International Conference on Critical Care in Obstetrics"** on 12th, 13th and 14th February, 2016 at Hotel Grand; www.ccob2016.com - for further information and online registration. Contact address: AOGD Office, Ward 8, Safdarjung Hospital, New Delhi, Tel.: 011-26714473; Contact person: Ms. Nisha Kashyap, M: 9654651720
7. **"Maternal Fetal Medicine Workshop"** on 5th and 6th March, 2016 at MAMC. Registration Fee is Rs 1000 for two days. For further details please contact: 9968604345, 9968604341.

CLINICAL UPDATE

Non Surgical Management of Fibroids

Anjila Aneja[1], Seema Bisht[2], Neena Bahl[3], Mamta Pattnayak[2]
[1]Director and Head, [2]Consultant, [3]Associate Director
Minimal Access Surgery Gynae Fortis Memorial Research Institute, Gurgaon & Fortis La Femme, New Delhi

Benign uterine leiomyomas (fibroids) are the most common pelvic tumor in women with an estimated lifetime risk of 70 percent in white women and 80 percent in black women. Uterine sarcoma is rare (3 to 7 per 100,000 in the United States population) with a poor prognosis.

Prophylactic therapy to avoid potential future complications from fibroids or their treatment is not recommended except for those women with significant submucosal leiomyomas who are contemplating pregnancy and women with ureteral compression

Medical management[1]

Until recently, medical management options for uterine leiomyomas have been of limited value because of their moderate efficacy and/or associated adverse effects. Novel therapies at the receptor and gene levels have emerged or are undergoing investigation and may eventually offer better long-term management options.

Hormonal treatment

***Combined oral contraceptive pills*:** Women with heavy menstrual bleeding associated with leiomyomas respond to OC therapy. It should be tried before invasive therapy. The mechanism of action is via endometrial atrophy.

Levonorgestrel releasing intrauterine system: Observational studies and systematic reviews have shown a reduction in uterine volume and bleeding, and an increase in hematocrit after placement. Also it provides contraception for women who do not desire pregnancy. One study concluded that the LNG-IUS significantly reduces menstrual blood loss and uterine volume in women with menorrhagia, with and without fibroids, while it does not significantly reduce fibroid volume. Another RCT found that although the rate of treatment failure was similar in both groups, the LNG-IUS was more effective in reducing menstrual blood loss than combined oral contraceptives in women with fibroid-related menorrhagia[2].

Progestational agents: As with combination OCs, there are no data to show the effectiveness of progestin-only contraceptive steroids specifically for treatment of leiomyomas. They can be considered for treatment of mild symptoms, especially for women who need contraception.

Gonadotropin releasing hormone agonists: Gonadotropin -releasing hormone (GnRH) agonists are effective medical therapy for uterine myomas. These drugs work by initially increasing the release of gonadotropins, followed by desensitization and downregulation to a hypogonadotropic, hypogonadal state that clinically resembles menopause. Most women will develop amenorrhea, improvement in anemia, and a significant reduction (35 to 60 percent) in uterine size within three months of initiating this therapy. However, there is rapid resumption of menses and pretreatment uterine volume after discontinuation of GnRH agonists. Severe hypoestrogenism side effects include hot flushes, sleep disturbance, vaginal dryness, myalgias and arthralgias, and possible impairment of mood and cognition. Bone loss leading to osteoporosis after long-term (12+ months) use is the most serious complication and most often limits therapy.

The side effects of long-term GnRH agonist administration can be minimized during therapy by giving add-back therapy with low dose estrogen-progestin after the initial phase of downregulation. Low dose estrogen-progestin therapy, such as used for menopausal replacement (equivalent to 0.625 mg of conjugated estrogen and 2.5 of medroxyprogesterone acetate/5mg norethindrone acetate) maintains amenorrhea and the reduction in uterine volume, while preventing significant hypoestrogenic side effects. Using OC add-back for leiomyomas is not indicated.

Gonadotropin-releasing hormone antagonists: The advantage of antagonists over agonists is the rapid onset of clinical effects without the characteristic initial flare-up observed with GnRH agonist treatment.

Progesterone receptor modulators: Drugs that modulate progesterone are increasingly used for medical treatment of fibroids. However, they not are approved by the US Food and Drug Administration (FDA). They have the advantage of oral administration and minimal symptomatic side effects. The major concern is whether there are endometrial effects with long-term use. **Ulipristal** acetate is a progesterone receptor

modulator (PRM) that inhibits ovulation, but has little impact on serum estradiol levels. The drug is approved for three months of preoperative therapy outside the US (European Conformity [CE mark]). Ulipristal acetate (oral, 5 mg or 10 mg once daily for 13 weeks) was compared with placebo in one randomized trial of 242 women with menorrhagia, fibroid-associated anemia, and a uterus that was ≤16 weeks of gestation size. Ulipristal acetate resulted in a significantly higher rate of resolution of menorrhagia (5 mg: 91 percent; 10 mg: 92 percent; placebo: 19 percent) and a significant, but only slightly higher increase in hemoglobin (5 mg: 4.3 g/dl; 10 mg: 4.2 g/dl; placebo: 3.1 g/dl). Significant reductions in fibroid volume occurred in women treated with ulipristal acetate (5 mg: -21 percent volume; 10 mg: -12 percent; placebo: +3 percent). There were no findings of endometrial hyperplasia or cancer. Approximately half of patients in each group chose to undergo surgical treatment of fibroids at the completion of the study[3].

The antiprogestin **Mifepristone** (RU-486) is the most widely studied PRM and reduces uterine volume by 26 to 74 percent in women with fibroids comparable to the reduction observed with GnRH agonists. Regrowth occurs slowly following cessation of the drug. Data from randomized trials and prospective studies have shown that high dose regimens (>10 to 50 mg/day) give comparable rates of amenorrhea to GnRH agonists, while lower doses (5 to 10 mg/day) achieve an amenorrhea rate of 40 to 70 percent and in other women, produce a reduction in menstrual flow.

Danazol and gestrinone: Androgenic steroids may be an effective treatment of leiomyoma symptoms in some women, but are associated with frequent unacceptable side effects.

Nonsteroidal anti-inflammatory drugs

NSAIDs have not been extensively studied in leiomyoma-related menorrhagia. NSAIDs do not appear to reduce blood loss in women with fibroids but provide relief in painful periods.

Antifibrinolytic agents

Antifibrinolytic agents, which are useful in the treatment of idiopathic menorrhagia, have not been well studied in leiomyoma related menorrhagia.

Other non surgical management of fibroids

Uterine Artery Embolization (UAE)

It should be offered as a mode of treatment alongside medical and surgical options for fibroids. Early and medium term (5 years) outcome are good. Symptoms control is as good as surgical treatment but the only caveat is one third patients need repeat procedure in five years. It is contraindicated in cases of recent or past pelvic infection, pregnancy, where a small risk of hysterectomy in case of complications is not acceptable and where there is significant doubt about the benign pathology[4].

Though successful pregnancies have been reported following UAE an RCT comparing UAE to myomectomy showed that pregnancy rates were lower and miscarriage rates higher following UAE. This option is still best reserved for women who do not desire future pregnancy[5].

Focused Energy Delivery Systems

A number of focused energy delivery systems have been tested in the past decade including those based upon radiofrequency electricity, supercooled cryoprobes and most recently, MRg-FUS or high frequency ultrasound guided transcutaneous ablation. A major disadvantage of all systems and techniques used to desiccate or ablate fibroids may be that they treat one fibroid at a time and they target the centre of fibroids, while fibroids have been shown to grow mostly from their periphery[6,7].

MR-guided focused ultrasound

The ExAblate 2000 (InSightec Inc., Haifa, Israel) was the first clinical MRg-FUS system approved by the FDA for treating uterine fibroids. Case series for MRg-FUS ranging from 51 to 359 patients have been published and short term efficacy is adequate, but complications such as skin burns have occurred in up to 7% of patients and at least one bowel perforation was reported. Disadvantages of the MRg-FUS system include high exclusion rate, requirement of an MR machine, prolonged time (minutes to several hours), treatment of one fibroid at a time, and ablation of fibroids centrally, while fibroids seem to grow peripherally[6].

Radiofrequency myolysis[8,9]

A new development in laparoscopic myolysis involves delivering of RF energy to myomas under ultrasonic guidance in an attempt to dessicate them directly. Mapping of myomas is performed by laparoscopic and ultrasound visualization. When a myoma is targeted for ablation, the RF probe is inserted percutaneously under laparoscopic guidance through a 2-mm skin incision. A recent multicentre trial concluded that RF myolysis of myomas is well tolerated and results in rapid recovery, high patient satisfaction, improved quality of life, and effective symptom relief. Total mean fibroid volume was reduced by 45.1% and mean blood loss by 38.3% at 12 months post-procedure. Disadvantages being the requirement of laparoscopy and concomitant use of ultrasound, additional percutaneous skin incision(s),

its treatment of one fibroid at a time (<8 cm diameter), and its ablation of fibroids centrally while fibroids grow peripherally.

Conclusion

Effective medical treatments for women with abnormal uterine bleeding associated with uterine fibroids include the levonorgestrel intrauterine system, gonadotropin-releasing hormone analogues, selective progesterone receptor modulators, oral contraceptives, progestins and danazol.

Effective medical treatments for women with bulk symptoms associated with fibroids include selective progesterone receptor modulators and gonadotropin-releasing hormone analogues. Ulipristal acetate is licensed for the preoperative use to decrease the size of fibroids and improve anaemia. Amongst the conservative interventional treatments currently available, uterine artery embolization has the longest track record and has been shown to be effective in properly selected patients. Newer focused energy delivery methods are promising but lack long-term data. Ongoing research and data collection are required to assess the relative merit of newer options as the technology continues to expand.

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The Red Alert

The hope against hope of being treated or mistreated
The uncountable pills and those hormonal surges
Just leaves bloated and helps me not
The pallor, edema flourishes like a fungus
Red tonics and capsules help me not
All this, as my cycles loose control
Unpredictable, erratic and voluminous
Injections only appear for rescue
Episodes just drains me financially emotionally physically
I heard when they talked about a womb device which
When Fitted relieves all the stress of compliance
Advancements for woman like me, the non responders
A step towards women empowerment.

-Dr Sarita Singh
Specialist, VMMC & Safdarjung Hospital

CLINICAL GUIDELINES

Medical Management of Endometriosis: Current Guidelines

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Endometriosis is a common gynecological condition presenting as chronic pelvic pain and infertility. The overall incidence is 6-10% in reproductive age group while 71-87% among women with chronic pelvic pain and 20-50% among infertile women However it has been seen as incidental finding in10% asymptomatic women undergoing laparoscopy or laparotomy for other reasons[1]. It has polygenic inheritance with strong association with monozygotic twins, positive family history (7 fold increased risk) and mullerian anomalies. Other risk factors are early menarche (<11 years), polymenorrhea (<27 days), low parity and menorrhagia.

Clinical evaluation

The classical features are chronic abdomino-pelvic pain, infertility and dysmenorrhea. The characterstic triple dysmenorrhea is found- premenstrual (due to congestion); intramenstrual (due to bleeding in closed space); and post menstrual (due to neural irritation by hemosiderin pigment absorption). Pelvic pain presents characteristically as secondary dysmenorrhea, deep dyspareunia or sacral pain during periods. Organ specific endometriosis give rise to site specific symptoms like non-gynaecological cyclical symptoms (dyschezia, dysuria, haematuria , cramps and hematuria).

In women with chronic pelvic pain (CPP), evaluation should include a thorough history, clinical examination to rule out other gynaecological causes of pain and non gynaecological causes like irritable bowel syndrome, interstistialcystitsis and urinary tract problems.

Pelvic examination can be helpful in diagnosis and localization of pelvic endometriosis. However as this is not feasible for adolescents and/or women without previous sexual intercourse, a rectal examination can be helpful. The diagnosis of deep endometriosis should be considered in women with tender induration and/ or nodules of the recto-vaginal wall or visible vaginal nodules in the posterior vaginal fornix .The ovarian endometrioma can be suspected by finding of tender adnexal masses during clinical examination.

Investigations

The diagnosis of endometriosis is suspected based on the history, the symptomatology, corroborated by physical examination and imaging techniques and is finally proven by histological examination of specimens collected during laparoscopy[1]. The combination of laparoscopy and the histological verification of endometrial glands and/or stroma is considered to be the gold standard for the diagnosis of the disease[1].

Radio-imaging modalities

In women with symptoms and signs of rectal endometriosis, transvaginal sonography (TVS) is useful for identification of ovarian endometrioma (ground glass echogenicity and no papillary structures with detectable blood flow) and rectal endometriosis, but needs expertise. Usefulness of 3D ultrasound to diagnose rectovaginal endometriosis and that of magnetic resonance imaging (MRI) to diagnose peritoneal endometriosis is not well established[2]. However MRI is often used for patients with equivocal ultrasound results and when rectovaginal/ bladder endometriosis is suspected. Specific medical technologies (Barium enema, transvaginal sonography (TVS), transrectal sonography and MRI) are needed to know the extent of deep endometriosis if there is a suspicion based of ureter, bladder and bowel involvement based on history or physical examination.

Biomarkers

Use of biomarkers in endometrial tissue, menstrual or uterine fluids and/or immunological biomarkers, including CA-125, in plasma, urine or serum is not recommended for screening or diagnosis of endometriosis. But Ca 125 can be useful to predict the disease recurrence. Levels of >100 IU/ML are suggestive of in extensive adhesions, obliteration of cul-de-sac and ruptured endometrioma.

Laparoscopy

It has been recommended that a positive laparoscopy should be confirmed by histology, since positive histology confirms the diagnosis of endometriosis even though negative histology does not exclude it. Obtaining tissue biopsies for histology in women undergoing surgery for ovarian endometrioma and/or deep infiltrating disease, is mandatory to exclude rare instances of malignancy(ESHRE Guidelines 2014) [2].

However a negative diagnostic laparoscopy seems to be highly accurate for excluding endometriosis.

Medical Management

Therapeutic options depend on the clinical presentation of the patient i.e. presence of pelvic pain, infertility or pelvic mass. The goals of the therapy must be recognized and properly addressed.Medical management is mainly indicated for alleviation of endometriosis related pain and as adjunct in endometriosis related infertility.

In patients diagnosed to have endometriosis with dysmenorrhea, NSAIDs and COCPS remain the first line of treatment. This is followed by GnRH agonist which has been recommended second line drug[1,2].

1) ***COCPs (Combined Oral Contraceptive Pills):*** can be given in cyclic or continuous manner as first line treatment (Table 1). Dysmenorrhoea- associated withdrawal bleeding can be avoided with use of extended cycle pills. Continuous COCPS can still be better as they create a state of psuedopregnancy leading to decidualisation and atrophy of endometriotic implants.

2) ***Progestins*****:** Oral or depot **medroxy progesterone acetate** is as effective as COC and also cost-effective. DMPA can be ideal for treatment of post hysterectomy residual disease. Some RCTs have shown subcutaneous DMPA equivalent and more cost-effective than GnRH agonist in decreasing pain without substantial bone loss. DMPA leads to bone loss which is completely reversible by 12 months. Oral **norethindrone acetate** and **subcutaneous DMPA** (**DMPA-SC, 104 mg**) are USFDA approved. **Norethindrone acetate** (5-20 mg /day) is effective in relieving dysmenorrhea and chronic pelvic pain. Although it has positive effect on calcium metabolism, negative effects are seen on serum levels of HDL cholesterol. **Dienogest**, a progestin with selective 19-nortestosterone and progesterone activity, 2 mg/day has been seen to give equivalent results to GnRH agonist (leuproloide acetate) in terms of effective long-term pain control with better side effect profile. **LNG-IUS** has been found equivalent to GnRH Agonist for pain control with comparative safety side effect profile. Advantage is continuous 5 year therapy without systemic side effects. It is particularly effective in recto vaginal endometriosis. However it is not yet Got FDA approval for pain relief. Once relieved by oral treatment patient can be switched to depot or intra-uterine preparation. **Danazol is** androgenic antiestrogen which although is very effective for endometriosis pain but not commonly used due to its side effect profile (Table1).

3) ***GnRH agonist*****:** Cochrane review have found no difference between GnRH agonist and other medical treatment options available hence it is not used as first line agent[3]. It has significant hypoestrogenic side effects which include hot flushes, dryness of vagina and osteopenia (reversible and short term use, but irreversible with long term use or multiple cycle use). Moreover recurrence of symptoms on discontinuation of drug has been found to be as high as 53% to 73% in mild and advanced cases respectively. Although recommended for 3-6 cycles, it can be used upto 1 year, if add-back therapy is added. In fact USFDA has only approved the use of 12 month course of GnRh agonist therapy (as second line).To avoid initial flare effect, GnRH agonist should be started in luteal phase of menstrual cycle.

Duration of therapy: It should be determined by choice of drug, response to treatment and adverse side effect profile. The COCPS and DMPA-SC can be used for long term but use of danazol and GnRH agonist is usually restricted to 6 months. With add back therapy, GnRHa can be given for long term (>6 months). COCPs owing to their comparatively better side effect profile can be used for prolonged treatment (i.e till menopause or till pregnancy is desired).

Recurrence rate at 5 year follow-up after discontinuing GnRHa treatment ranges from 54-73% in advanced endometriosis. The recurrence rate after 1 year of stopping danazol is 50%.

Efficacy of medical treatment can be evaluated by decreased pain scores (pre and post-treatment) and decrease in serum level of CA-125.The patients on GnRH agonist therapy should be monitored by serum estradiol level and bone mineral density.

'ADD-BACK' regimen

The idea behind use of add back therapy is to maintain a therepeutic window for serum estradiol level between 30-50 pg/ml so as to effectively treat the endometriotic implants by anti-estrogenic effect without causing menopausal symptoms (Fig 1).

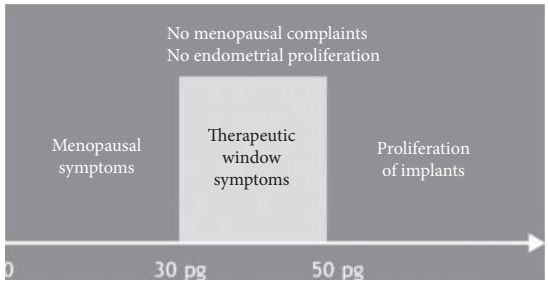

Fig 1: Therapeutic window phenomena of add back therapy

Table 1: Drug options available for medical management

S no.	Drug	Dose	Side effects
Drugs causing Psuedopregnancy effect			
1.	COCPs (low dose monophasic pills)	• Cyclic regime • Continuous regime-1 tab daily, increase by 1 to 2 tab biweekly till amenorrhoea.	Headache, nausea, breakthrough bleeding and other usual side effects of COCPs.
2.	Progestins	Medroxyprogesterone acetate (Deviry, Meprate) oral 30 mg/day	Weight gain, depression
		Dydrogesterone (Duphaston) 20-30mg/day	
		Norethindrone-acetate (Regesterone); Norethisterone –acetate (Primolut-N, Sysron-N), 5- 20 mg daily	Break through bleeding, Negative effects on serum levels of high-density lipoprotein cholesterol. Positive effect on calcium metabolism.
		Dienogest (Endoreg) 2 mg/day	
		Depot progestin therapy- DMPA(150 mg every 3 months) DMPA-SC(104 mg)	break through bleeding, detrimental to BMD.
		LNG-IUS (Levonorgesterol Intra Uterine System) 20 mg/d of levonorgestrel	Expulsion rate (5%) Risk of pelvic infection (1.5%). No systemic side effects.
Drugs causing pseudo menopause			
3.	Danazol (antiprogesterone)	200-800mg/day depending on Disease severity, dose titrated to achieve amenorrhea.	weight gain, acne, hirsuitism, breast atrophy, and, rarely, virilization
4.	Gestrinone (antiprogesterone)	2.5mg/day	weight gain, acne, hirsuitism, positive effect on cholesterol
5.	GnRH agonist treat ment with HT add back	**Intramuscular injection** Leuprolide acetate (Lupron Depot),(3.75 mg per month) Goserelin (Zoladex),(3.6 mg/month) Triptorelin (Decapeptyl, Gonapeptyl) Buserelin **Nasal Spray** Naferelin (Synarel) (200microgm/day) Buserelin (Suprecur)	Hypoestrogenic side effects especially bone mineral density changes (6% decrease in trabecular bone density after 6 months)

Add-back regimen addition eliminates GnRH agonist induced bone-mineral density loss thereby providing symptomatic relief without reducing its efficacy for pain relief. Add back regimen are thus advocated for women especially undergoing long term therapy (>6 months); however as it doesn't decrease the pain relief efficacy seen during 3 months or 6 months of GnRH agonist therapy, it can be started immediately with GnRH agonist even with short term therapy (<6 months).(Evidence Level A, ESHRE Guidelines)[1,2].

Strategies for add back therapy are progestins alone; progestins with bisphosphonates and low dose progestins with estrogens[1]. In addition use of low dose estrogen alone has also been described[4]. However due to concerns of estradiol level, estrogen alone is not preferred as add back. Moreover low dose estrogen (0.625 mg conjugated equine estrogen or estradiol valerate 1 mg/day) can be used only for short term in comparison to progestins alone or in combination with low dose estrogen.

Out of these, Norethindrone acetate (NETA) 5 mg/day has been FDA approved as add back therapy[1]. NETA is a unique progestin with both estrogenic and androgenic properties and is effective as add-back therapy without estrogen supplementation. Through its estrogenic action it has beneficial effect on BMD and vasomotor symptoms in women treated with GnRHa. In addition, it has strong endometrial antiproliferative effects. Thus GnRH agonist and norethindrone with or without low dose estrogen (conjugated estrogen 0.625 mg/day) has been seen to decrease the side effects while preserving therapeutic efficacy. Other options for norethindrone are transdermal estradiol with medroxy progesterone acetate (transdermal estradiol 25 mg/day with MPA 2.5 mg orally daily), but this regime is not FDA approved. Daily calcium supplementation (1000 mg) must be given to the patients using GnRH agonist with add back therapy.

Role of analgesics in early part of medical therapy:

Targeted medical therapies may require at least 1 cycle

to initiate pain relief. Especially with GnRH agonist therapy, started in the luteal phase or during periods won't prevent dysmenorrhea and may even accentuate pain because of the initial flare effect in the first cycle. In this situation, analgesia in the form of NSAIDs or even opioids must be started to make the patient more comfortable until the primary medical management becomes effective.

Role of pre-operative and post-operative medical suppressive treatment: There is no data to support the use of pre-operative medical suppressive therapy[1,2]. Post-operative medical treatment is indicated in presence of residual disease and to extend pain free interval after surgery[1,2].

Medical treatment options in post-operative case: COCPs have been very effective in reducing endometrioma recurrence, the frequency and severity of dysmenorrhoea. There is no difference between continuous or cyclic combined COCPs. A recent Cochrane review has suggested post-operative use of LNG-IUS for treatment of dysmenorrhoea after endometriosis surgery.[5]

Treatment of incidental finding in asymptomatic women: There is no current evidence to support the use of medical treatment to prevent disease progression[1].

Medical treatment in adolescent endometriosis

Endometriosis in adolescents is often early stage and atypical. Endometriosis is most common cause of secondary dysmenorrhoea in adolescents and physical examination rarely reveal abnormalities as most have early stage disease. Empiric treatment with NSAIDS and COCPs is appropriate for most adolescents with dysmenorrhoea. Those who do not respond require early referral for further investigations which involves laparoscopy for diagnosis and treatment[2,6].

Laparoscopist should look intra abdominally for clear vesicles (can be clearly delineated and visualised after putting 200ml saline over the lesions) and red lesions in adolescents (Evidence, Level II, ACOG). All available therapies for endometriosis may be used in adolescents but age of patient and side effects profile of medication should be considered. Step wise approach is appropriate starting with NSAIDS, COCPs in extended or continuous regime. Empiric GnRH agonist therapy with hormone therapy, add back is reserved for adolescents more than 18 years, with chronic or persistent pain, due to detrimental effects on BMD. Once diagnosis is confirmed, COCPs (continuous) can be given[2,6].

Medical treatment of infertility related to endometriosis

Hormonal suppression is contra-indicated before or after surgical treatment of endometriosis in view of no available evidence of increased effectiveness over that of surgery alone and moreover it prolongs or delays the opportunity for conception to occur (Evidence level A).[1,2]

Women with endometriosis related infertility over 35 years age should be referred for IVF. For those with chronic and advanced endometriosis, studies have shown positive results with long term treatment with (3-6 months) with GnRH agonist with HT add-back before an IVF cycle. Cochrane review has shown that down-regulation for 3-6 months with GnRH agonist in women with endometriosis increase the odds of clinical pregnancy rates by more than four times. In females with minimal to mild endometriosis (as per AFS/ASRM stage I and II), controlled ovarian hyperstimulation with gonadotropins with IUI have shown higher pregnancy rates in comparison to expectant management.[7]

Management of menopause in women with endometriosis

In women with surgical menopause due to endometriosis, estrogen/progesterone therapy or tibolone can be effective for treatment of menopausal symptoms. It is recommended that clinicians should continue treatment in women with history of endometriosis after surgical menopause with combined estrogen and progesterone or tibolone at least upto age of natural menopause[1,2]. In postmenopausal women after hysterectomy and with a history of endometriosis, unopposed estrogen should be avoided.

Medical treatment in extra-pelvic endometriosis

Symptoms of extra-pelvic endometriosis depend on location and depth of infiltration. Ovarian suppression with GnRH agonist has been well supported by evidence in literature as first line of management except in cases of obstruction of ureter/bowel which is best treated surgically. There have been observational studies of successful use of suppressive medical treatment even in case of recto vaginal endometriosis,resulting in Level D recommendation[2].

Newer drugs under research

The basis of the use of aromatose inhibitors is the over-expression of aromatase in endometriosis implants.

Anastarazole and letarozole are although promising in observational trials, unavailability of RCTS does not permit these drugs to be recommended for routine use. Drugs acting through estrogen receptor (ER-alpha and ER-beta) over endometriotic implants, SERMs and progesterone antagonists are still under trial.Selective progesterone receptor modulators and GnRH antagonist have been seen to have potential use but it is still under phase III TRIAL. Atorvastatin [high dose (2.5mg/kg)] has been seen to cause significant reduction in VEGF level in animal models. Human studies are awaited. TNF-α Inhibitors and immunomodulators like pentoxiphylline are still under trial.

Conclusion

Endometriosis is a chronic disease with debilitating symptom of pain along with infertility. Medical treatment is mainly indicated for management of endometriosis related pain. Combined hormonal contraceptives should be first line agent. In addition, administration of progesterone alone (orally, intramuscular or subcutaneous) may also be considered as first line therapy. GnRH agonist with addback or LNG-IUS should be considered as second line therapeutic option. All options should be administered for a minimum of 3 months with evaluation of efficacy at end of trial. As such effective medical treatment can be continued till age of menopause or until pregnancy is desired. While awaiting symptom resolution by medical treatment, NSAIDS can be used as effective analgesics.

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Congratulations !

Dr Monika Gupta, Assistant Professor, VMMC & Safdarjung Hospital & Joint Sect AOGD has been awarded the prestigious **"Kamini A Rao Yuva FOGSI Orator Award 2016"** from North Zone. This includes an oration at North Zone YUVA FOGSI conference at Ghaziabad, April 2016 and also at AICOG 2017 at Ahmedabad.

Metabolic Consequences of Polycystic Ovary Syndrome and Interventions to Reduce Cardiovascular Disease

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Polycystic ovarian syndrome (PCOS) is the commonest endocrinopathy among women of reproductive age with an estimated prevalence of about 5-10%. It usually manifests as menstrual disturbances, hirsuitism, obesity and infertility in the adolescent and reproductive period. However what is particularly of concern are the long term metabolic consequences. These women are more prone to develop impaired glucose tolerance, diabetes mellitus, hypertension, hyperlipidemia, sleep disordered breathing and coronary artery disease. Insulin resistance has been incriminated as a key factor in the pathogenesis of PCOS and the same may be responsible for many of the metabolic effects seen in PCOS.

Which phenotype of PCOS is most susceptible for metabolic dysfunctions?

Women with classic PCOS have greater menstrual irregularity, hyperandrogenism, total and abdominal obesity, and insulin resistance (IR) and have more severe risk factors for T2 Diabetes Mellitus and Cardio Vascular Disease than PCOS patients' diagnosed using non-NIH criteria. Ovulatory PCOS patients have lower body mass index (BMI) and abdominal obesity, lesser degrees of hyperandrogenism and hyperinsulinemia, reduced metabolic syndrome (MBS) prevalence, and milder forms of dyslipidemia, whereas non hyperandrogenic PCOS patients have the most metabolically favourable profile, often indistinguishable from normal women[1].

Diabetes mellitus

Type 2 Diabetes- IR occurs in approximately 60–80% of women with PCOS and in 95% of obese women with PCOS[2]. In most classic cases of PCOS, insulin-mediated glucose uptake is impaired, but the precise mechanism remains elusive. These women with PCOS have IR independent and additive with that of obesity, with PCOS and obesity acting synergistically to impair insulin sensitivity. Approximately, 25% to 30% of women with PCOS will show impaired glucose tolerance by the age of 30 and 8% of affected women will develop type 2 diabetes annually. **The prevalence of type 2 diabetes in women diagnosed with PCOS is 7 times higher than normal population**. Insulin resistance combined with abdominal obesity is thought to account for the higher prevalence of type 2 diabetes in PCOS. However, the risk of developing type 2 diabetes is also increased in non-obese women with PCOS.

Gestational Diabetes- Gestational diabetes mellitus (GDM), defined as carbohydrate intolerance at onset of pregnancy (or first recognition), affects~10% of pregnancies overall. Common risk factors include nonwhite race/ethnicity, older age, obesity, and prior GDM. Some studies suggest the risk of GDM is higher among PCOS versus non-PCOS women, and several studies note an increased prevalence of polycystic ovarian morphology and symptoms in women with prior GDM[3]. Other conditions predisposing to glucose intolerance might also increase the risk of GDM. It was seen in one **study that women diagnosed with PCOS had a 2.4-fold increased odds of GDM, independent of age, race/ethnicity, and multiple gestation**[4]. Thus the prevalence of gestational diabetes mellitus is twice as high among women with PCOS. 2-hour post 75 gm oral glucose tolerance test is recommended for all pregnant women with PCOS.

Dyslipidemia

Dyslipidemia is very common in PCOS patients (up to 70%) and may present with different patterns, including low levels of high-density lipoprotein (HDL)-cholesterol (HDL-C), increased values of triglycerides and total and low-density lipoprotein (LDL)-cholesterol (LDL-C), as well as altered LDL quality[5]. These different patterns may be related to the associated effects of IR and hyperandrogenism that combine with environmental (diet, physical exercise) and genetic factors

Metabolic syndrome

Metabolic syndrome has been differently defined by various bodies. However, all of the definitions incorporate measures of central obesity, glucose intolerance, dyslipidemia, and high blood pressure. The prevalence of the metabolic syndrome in PCOS has been reported to be 43-47%, which is twice as high as the prevalence in the

general population of comparable age, even after adjusting for BMI.[6] The components of the metabolic syndrome most commonly present in PCOS are central obesity and low serum high-density lipoprotein cholesterol (HDL); however, elevated blood pressure, impaired fasting glucose, and glucose intolerance are commonly present. There is also an increased prevalence of metabolic syndrome among the sisters of women with PCOS.

Hypertension

Several mechanisms potentially responsible for the development of hypertension in PCOS are hyperandrogenemia, insulin resistance, obesity, and increased sympathetic nervous system activity. There is direct relationship between insulin plasma levels and blood pressure. The prevalence of treated hypertension is three times higher in women with PCOS between the ages of 40-59 years. The incidence of preeclampsia in obese pregnant women with PCOS compared to the general pregnant population is 4 times higher. Whether hypertension is associated with PCOS independent of obesity remains controversial. Nevertheless, detection and subsequent treatment of hypertension in this population should decrease the adverse sequelae from hypertensive cardiovascular disease. Moreover, treatment of the risk factors inherent to PCOS, such as hyperandrogenism, insulin resistance, and obesity, may minimize the risk not only for the development of hypertension but also for incident cardiovascular disease independent of hypertension.[7] Treatment of hypertension in the PCOS population may take the form of lifestyle modification or pharmacotherapy.

Cardiovascular disease (CVD)

Hyperinsulinemia appears to be the main reason for the increased cardiovascular risk of women with PCOS. There are two mechanisms by which insulin resistance in PCOS contributes significantly to higher incidence of cardiovascular disease in these women. One mechanism is the direct atherogenic action and the other mechanism is the adverse affect of the lipoprotein profile. Women with PCOS are seen to have more extensive coronary artery disease by angiography. Impaired glucose tolerance and diabetes caused by PCOS are known risk factors for cardiovascular disease. Women with PCOS should be screened for cardiovascular risk by determination of BMI, fasting lipid and lipoprotein levels and metabolic syndrome risk factors.

Sleep disordered breathing

Sleep Disordered Breathing(SDB) is a spectrum of disorders in which there is fragmentation of sleep due to repeated episodes of partial or complete cessation of breathing for 10 s or more during sleep. It includes, snoring, upper airway resistance syndrome, obstructive sleep apnea and obesity hypoventilation syndrome. The risk factors are male gender, age and obesity. The most common presentation of SDB is excessive daytime sleepiness (EDS). This can lead to lack of concentration, poor work output, and the serious risk of road traffic accident. The metabolic consequences include increased risk of glucose intolerance, type II diabetes mellitus, hypertension, stroke, and coronary artery disease. As is seen for PCOS, insulin resistance has been associated with the pathogenesis of SDB. The prevalence of SDB has been reported as high as 44.4% among obese PCOS women when compared with 5.5% in the control group[8]. Obesity and high testosterone levels in PCOS seem to play a role in the development of sleep apnea.

It has been seen that the metabolic disorders in PCOS are increased in severity in the women who are suffering from SDB.[9] **Hence it is imperative to screen all women with PCOS for SDB. All women who give history of snoring and those who are obese should undergo an overnight polysomnography which is the gold standard for the diagnosis of SDB**.[9]

How can we reduce the risk of CVD in PCOS?

Screening for CVD risk factors

- Hypertension is a significant contributor to the risk for cardiovascular disease. The increased prevalence of hypertension in women with PCOS may contribute to the increased risk of cardiovascular disease in women with PCOS. **The Androgen Excess and Polycystic Ovarian Societies recommend that blood pressure be obtained in women with PCOS at every visit and that pre hypertension be detected and treated given the potential benefit of lowering blood pressure for the prevention of CVD.[7]**
- Women with PCOS should be screened for cardiovascular risk by determination of waist circumference at every visit. Waist circumference is measured at the top of the iliac crest with the patient standing, using a nonfolded tape held parallel to the ground at the end of expiration. A waist circumference of at least 88 cm (35 inches) in Caucasian/African-American women or at least 80 cm (31.5 inches) in Hispanic, Native American, Asian (East and South), and European women is the easiest way to establish the presence of abdominal obesity.[10]
- A complete lipid profile (total cholesterol, LDL-C,

non-HDL-C, HDL-C, and triglycerides) should be determined. Based upon AHA guidelines[11], if the fasting serum lipid profile is normal, it should be reassessed every 2 yr or sooner if weight gain occurs. It is recommended that in women with PCOS without additional CVD risk factors, LDL-C levels should be less than 130 mg/dl. Those with MBS or T2DM/ overt vascular/renal disease should have serum LDL-C levels less than 70–100 mg/dl; serum triglyceride levels, as an independent CVD risk factor in women, should be less than 150 mg/dl .[11]

- A 75 gm OGTT as baseline and if normal to be repeated every 2 years. However in the presence of impaired glucose tolerance it should be repeated annually.
- Screening of women for sleep disordered breathing, by taking history of snoring from self or partner is very important. All snorers and obese women with PCOS should be referred to sleep specialist for overnight polysomnography. Treatment of SDB goes a long way in ameliorating or reducing the metabolic dysfunctions in women with PCOS.

Primary prevention of CVD: interventions recommended

Life style modifications which include diet, exercise, smoking cessation and behavioural modifications is the first line management to prevent CVD in PCOS.

- Hypocaloric, low saturated fat, increased mono- and polyunsaturated fat diet (500–1000 kcal/d reduction; <30% calories from fat, <10% calories from saturated fat; increased consumption of fiber, whole-grain breads, cereals, fruits, and vegetables) is recommended, along with at least 30 min of moderate-intensity physical activity daily to maintain weight. Together, both reduce BMI and improve IR and cardiopulmonary function in overweight PCOS women and provide greater reductions in fat mass in PCOS women.
- It is suggested that overweight/obese PCOS women should initially attempt 5–10% weight loss to reduce obesity-related CVD risk factors, with long-term goals of achieving and maintaining reduced weight of 10 to 20% and a waist circumference of less than 88–80 cm, depending upon ethnicity[10].
- An individualized exercise program assures optimal compliance and includes group or home exercise and walking (10,000 steps = 30 min daily exercise; 15,000 steps are usually needed for weight loss). Even without weight loss, sedentary women with PCOS undergoing moderate-intensity exercise experience improved insulin resistance and dyslipidemia. Screening for premature CVD is important before initiating exercise[12].

Pharmacotherapy for risk factors for CVD

- Insulin-sensitising agents such as metformin and the thiazolidinediones to reduce insulin resistance and thereby reduce the risk of developing diabetes and other metabolic sequelae. However, there is no strong evidence regarding the long-term benefits for the use of these agents in women with PCOS.
- Use of weight reduction drugs (Orlistat) may be helpful in reducing hyperandrogenaemia.
- Cholesterol lowering drugs should be reserved for women with PCOS proven to have dyslipidaemia
- Antihypertensives are indicated if blood pressure is >140/90 mm Hg.

Role of bariatric surgery

Bariatric surgery has been shown to be effective in women with PCOS and may be an option for severely obese women with PCOS in whom long-term diet-based strategies are seldom successful. Bariatric surgery should be performed only when standard weight loss regimes have failed in PCOS women with a BMI greater than 40kg/m^2 or greater then 35kg/m^2 with a high-risk obesity related condition.

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CLINICAL UPDATE

Premenstrual Syndrome - When and How to Treat

Sanjivni Khanna[1], Nivedita Kaul[2], Priyanka Bhardwaj[3]
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Background

Symptoms associated with the premenstrual phase of the menstrual cycle have been acknowledged by physicians and in the general culture for a little more than 80 years, while the notion that they define a clinical syndrome is about 60 years old. In 1931, Robert T. Frank presented a paper to the New York Academy of Medicine in which he called attention to a "large group of women" who were subject to "premenstrual tension". Their "intense personal feeling" he claimed, far surpassed the "varying degrees of discomfort", which preceded the onset of menstruation in normal women.

In 1953, Katharina Dalton, a British doctor, co-authored an article in the British Medical Journal, introducing the term "premenstrual syndrome". In 1981, Dalton served as the chief defense medical expert in a murder trial in London, wherein she successfully argued that the defendant was not responsible for murdering her lover as she suffered from severe PMS. "PMS" or the disease of 1980s became a media event. More importantly, PMS acquired medical legitimacy, after years of telling women that the problems were "all in the head".

Definition

There is no single precise definition of PMS. A working definition of PMS is 'a condition which manifests with distressing physical, behavioral and psychological symptoms, in the absence of organic or underlying psychiatric disease, which recurs regularly during the luteal phase of each menstrual cycle and disappears or significantly regresses by the end of each menstruation'[1]. Symptoms of PMS are distinguished from normal physiological premenstrual symptoms because they cause significant impairment to daily activity.

The term PMDD (Premenstrual Dysphoric Disorder) has been adopted by the American Psychiatric Association and denotes a severe subtype of PMS. Research criteria for the diagnosis of PMDD have been laid down in the Diagnostic and Statistical Manual of Mental disorders, 4th edition[2].

Classification

The International Society for Premenstrual Disorders (ISPMD)[3,4] defined precise criteria for diagnosing premenstrual disorders and divided PMS into '**core**' and '**variant**'.

Core (or typical) PMD is associated with spontaneous ovulatory menstrual cycles and may be sub-divided into *predominantly physical symptoms, predominantly psychological or mixed.*

Criteria for diagnosing core premenstrual disorder[3]
- It is precipitated by ovulation
- Symptoms are not defined, although typical symptoms exist
- Any number of symptoms may be present
- Physical and psychological symptoms are important
- Symptoms recur in luteal phase
- Symptoms disappear by end of menstruation
- A symptom-free week between menstruation and ovulation
- Symptoms must be prospectively rated
- Symptoms are not an exacerbation of underlying psychological or physical disorder
- Symptoms cause significant distress and disruption of daily activities

Variants of PMD encompass more complex features, divided into the four following categories: *premenstrual exacerbation of an underlying condition, PMS in the absence of menstruation, progestogen-induced PMS and PMS with anovulatory ovarian activity.*

Symptoms of PMS and PMDD- These are summarized in the box below.

Symptoms associated with PMS and PMDD[4]	
Physical	**Psychological and Behavioral**
Abdominal bloating, weight gain	Anger, irritability
Breast tenderness or fullness	Anxiety
Cramps, abdominal pain (overeating/food craving)	Changes in appetite
Fatigue	Decrease in concentration
Headache	Depressed mood, mood swings
Nausea	Changes in libido
Swelling of extremities	Increased or decreased sleep

Grading of severity

PMS can be graded as mild, moderate and severe[1]

Mild- symptoms do not interfere with personal/social and professional life

Moderate- symptoms interfere with personal/social and professional life, however the individual is still able to function and interact, although may be sub-optimally

Severe- Individual is unable to interact personally/socially/professionally—withdraws from social and professional activities (treatment resistant)

Diagnosis

Clinical history is the key to the diagnosis of PMS. The ISPMD highlighted the importance of an accurate history of symptoms, which should be recorded prospectively for a minimum of 2 months. The Daily Record of Severity of Problems (DRSP)[5] is a well-validated tool used in diagnosis of PMS. A new App, PreMentric S is now available (currently for iphones only) which graphically documents symptoms, provides a diagnosis and monitors therapy, however it has yet to be scientifically validated.

Some medical or gynaecological conditions such as anemia, hypothyroidism, endometriosis or physiological ovarian cysts may replicate physical symptoms of PMS. It is important to check thyroid function and hemoglobin levels to rule out hypothyroidism and anemia. Women with premenstrual symptoms may have concurrent psychiatric illnesses that may warrant referral to a specialist before initiating treatment.

Treatment

Management of PMS is usually performed in a stepwise manner from non-pharmacological strategies, antidepressant medications, hormonal strategies, with surgical options being a last resort. It is important to individualize treatment based on severity and timing of symptoms. Lifestyle adjustments such as improved self care, low glycemic-index diet and stress reduction should be considered before starting treatment. There is evidence from non-randomized trials that exercise improves PMS symptoms.

Non-pharmacological treatment

Dietary calcium and vitamin D may play a role in PMS symptoms. Women with a high dietary calcium and vitamin D[6] intake were less likely to have PMS symptoms as compared to women with a low intake, but whether supplementation decreases PMS symptoms is not known. Supplementation or high dietary intake of calcium and vitamin D may be considered for symptom relief in women with PMS.

A course of cognitive behavioral therapy (CBT) to address relaxation, stress management and assertiveness training has been shown to be effective in mild PMS, with success being comparable to treatment with fluoxetine. Successful CBT could obviate the need for pharmacotherapy. In a randomized study[7] comparing the treatment effects of fluoxetine and CBT and combined therapy (fluoxetine + CBT), significant improvement occurred in all three groups after 6 months of treatment. Fluoxetine provided more rapid improvement but CBT was associated with better maintenance of treatment effect compared with fluoxetine.

Complementary therapies

Various complementary therapies like, Vitamin B6, magnesium, Agnuscastus, St John's wort, Ginkgo biloba, evening primrose oil, light therapy and isoflavones, have been tried but data from clinical studies is limited and underpowered. Rigorous research on complementary therapies[8] is limited, with only a few well designed randomized trials. One study on extract of the Agnuscastus fruit[9] showed improvement in PMS symptoms in upto 52% patients. Complementary therapies may have drug interactions with other medications and this should always be considered before prescribing them.

Antidepressants

The use of selective serotonin reuptake inhibitors (SSRIs), particularly fluoxetine 20-60mg, paroxetine 20-30mg, citalopram 20-40mg or sertraline 50-150 mg on a daily basis, have shown a substantial reduction in the physical and psychological symptoms of PMS compared to placebo[10]. In view of their proven safety and efficacy, SSRIs should be considered one of the first line pharmaceutical management options in severe PMS.

SSRIs may be prescribed in the luteal phase or continuously. Some studies suggest that intermittent SSRI use is as effective as continuous use in reducing mood symptoms but less effective in relieving somatic symptoms. Women with PMS being treated with SSRIs must be informed about possible **side-effects** such as insomnia, nausea and reduction in libido, and, that the side-effects are lesser with intermittent use. If SSRIs are being prescribed on a continuous basis, abrupt withdrawal should be avoided; dose should be tapered over a few weeks to avoid **withdrawal symptoms** (gastrointestinal disturbances, headache, anxiety, sleep disturbances, sweating).

Paroxetine should be avoided for PMS treatment in women of child-bearing age without adequate contraception because of its increased risk for congenital abnormalities when taken in the first trimester of pregnancy.

Hormonal agents

Oral contraceptives

Newer OCPs containing drospirenone, 3 mg and ethinyl

estradiol 20microgm are effecting in relieving symptoms of PMS when administered for 24 days followed by 4 days of inactive pills compared to placebo[11]. Continuous therapy with the pill (such as an extended 42 to 168 day regimen) may be more effective in treating PMS than cyclical therapy[12]. Drospirenone is a newer progestogen with anti-androgenic and anti-mineralocorticoid activity. Although this pill is licensed in the USA for PMS, it is not in the UK.

Second generation OCPs containing levonorgestrel or norethisterone, regenerate PMS like symptoms and thus have no role in its treatment.

Estrogen

Percutaneous estradiol, either as an implant (100 µg) or patch (100 µg twice a week) has been shown to be effective in the management of physical and psychological symptoms of severe PMS.

Add-on progestogen with estrogen

Use of continuous estradiol normally necessitates the addition of cyclical progestogen (10–12 days) to avoid endometrial hyperplasia in women who have a uterus. As progesterone itself can cause PMS like symptoms, when treating women with PMS, lowest possible dose of progestogen is recommended. *Choice of progestogen is important because norethisterone and levonorgestrel can produce PMS like effects.* Natural progesterone such as micronized progesterone, however, is a diuretic and a CNS anxiolytic and thus a better choice. Alternatively the LNG-IUS may be used as it provides excellent endometrial protection and a few months after insertion there are minimal systemic levels of progesterone. However, patients should be advised that low systemic levels of LNG can initially produce PMS like adverse effects.

Gonadotropin-releasing hormone (GnRH) analogues

Long acting GnRH analogues suppress ovarian function and inhibit the menstrual cycle. They provide significant symptom relief in physical and behavioral symptoms of PMS[13] so much so that lack of efficacy suggests a questionable diagnosis rather than a limitation of treatment.

GnRH analogues induce a hypoestrogenic state – in the short term this causes hot flashes, night sweats, low mood and insomnia and in the long term, vaginal atrophy, osteoporosis and increased cardiovascular risk. Thus therapy should be recommended only as second or third line treatment and reserved for women with the most severe symptoms where other treatments have failed. Treatment without add-back therapy should not be continued for more than 6 months.

When treating women with GnRH analogues where add-back therapy is required, continuous combined HRT or tibolone should be used as these reduce the estrogen deficiency symptoms and trabecular bone loss without causing PMS like progestogenic side effects. Besides, there is no reversal of the beneficial effect of GnRH when using add-back.

Women on long-term treatment with GnRH analogues must receive advice on exercise, diet and avoidance of smoking. They must have yearly monitoring of their bone mineral density ideally by dual energy x-ray absorptiometry. Treatment should be stopped if bone density falls significantly on scans done one year apart.

Surgical approach (hysterectomy and bilateral salpingo-oophorectomy)

TAH and BSO is a permanent form of ovarian suppression and has a beneficial effect on PMS. Understandably surgical options should be considered only as a last resort in severely affected patients who have failed to respond to other therapies, and, also have significant gynecologic problems for which surgery may be appropriate. A trial of GnRH with positive effect is advisable before deciding to perform BSO & hysterectomy. Oophorectomy alone can relieve PMS symptoms but hysterectomy is required to allow unopposed estrogen therapy post surgery as use of progestogens can cause PMS like effects.

Surgery will render the woman infertile and induce menopause. HRT should be considered in women undergoing surgical oophorectomy before the age of 50 years.

Conclusion

PMS occurs in about 30 – 40% of reproductive age women causing recurrent physical, affective and behavioral symptoms. These can significantly impair the daily lives of women including their work and personal activities. An individualized step-wise therapeutic approach based on type and severity of symptoms is recommended.

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Rajiv Gandhi Cancer Institute and Research Centre

Sector-5, Rohini, Delhi-110085, India, Web : www.rgcon2016.com • www.rgcirc .org

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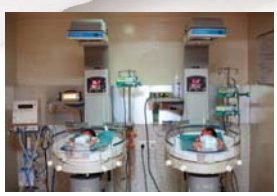


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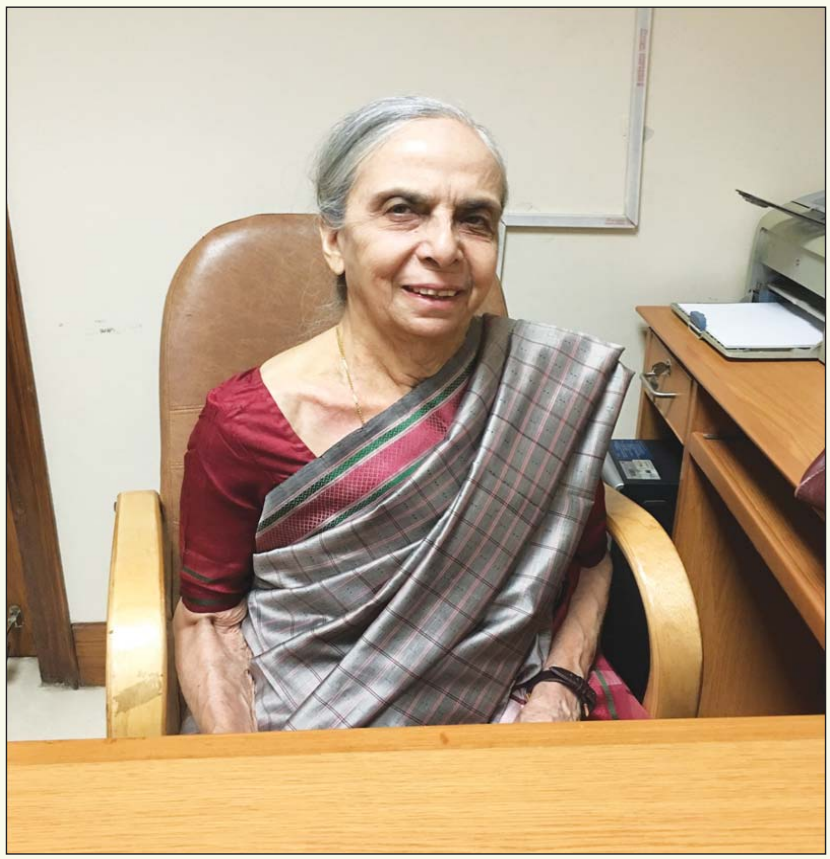
Meet the Luminary

Dr P Chadha

Dr P Chadha, the dedicated clinician and devoted teacher, who has shaped the life of many generations of gynaecologists, was sitting in front of us..... we were simply awed by her honesty, fortitude and selflessness. The inner strength and character of her persona is reflected in her beautiful thoughts, which we penned down for all of you.

Dr Jyotsna Suri, Dr Rekha Bharti

Birthday 16th June	**Place of birth** Jammu & Kashmir	**Graduation** Lady Hardinge Medical College, 1959	**Post-graduation** Lady Hardinge Medical College
If not a gynaecologist, what would you have been? I would have been a physician		**What makes your day?** Teaching postgraduate students- I feel happy and satisfied the day I teach my students.	
High point of your life When I delivered my granddaughter, I was blessed by God.		**One habit that you are proud of** Reading light literature.	**How do you de-stress?** I don't get stressed easily.
Something you are proud of Whatever I learnt from seniors, I taught that to my students, I have never harmed any student.		**What ruins your day?** I don't get rattled at small things	
Favourite food I used to be a non-vegetarian till long time back as nobody in my family used to eat meal without non-veg. Now I am a vegetarian but I take eggs. I like black tea and coffee.			
Your favourite pastime I have 2 grand children 17 and 13 years old, I love to talk to them.	**Favourite Singer** No particular singer, I listen to aartis, ghazals and any type of old music.		**Any regrets?** Nil



What disappoints you? Students not studying, I want the students to pass in the first attempt, their performance goes down when they don't clear in their first attempt. Another thing which disappoints me is when staff doesn't pay attention to their duties and when people criticize one another and their colleagues.

Your favourite books Any good book that is light reading. I purchased "The World's Best Fairy Tales" in 1968, I still have these books with me and enjoy reading them. I also like all books authored by Charles Dickens. My all time favourite book is "Gone with the wind".

Your role model Dr Miss L R Phatak- she was FRCS of 1930's, was in Safdarjung Hospital and then went to Lady Harding Medical College & Maulana Azad Medical College. She was later appointed as Commissioner, Family Planning. She was a very rough and tough lady but a very nice person who would help you in crisis. She instilled so many things about the art of good administration into me. I learnt a lot from her.

Do you meditate Once I was in Pondicherry as an examiner, there I went to Aruvilla Ashram. It was a round hall with roof in the shape of a tomb, the only source of light in that hall was a prism in the centre of the roof. Everyone had to wear white socks and the sheets were also white in colour, the place was so calm and peaceful for meditation. That's the only place where I enjoyed meditating.

Your strategy in a crisis I am quiet and calm; when I am operating and there is bleeding, I have learnt over years of experience to pack the area of bleeding and wait for 2-5 minutes. In the meantime I formulate what I want to do.

Your professional journey After MBBS from Lady Hardinge Medical College I did 3 house jobs in Surgery, Medicine and Gynaecology. Dr Usha Sabharwal, Dr Sheila Mehra and Dr Chandrama Anand were my MBBS batch mates. I got MD in Preventive and Social Medicine at All India Institute of Medical Sciences and MD Obstetrics and Gynaecology at Lady Hardinge Medical College. Joined Hardinge as postgraduate, got married during my MD and completed registrarship from Lady Hardinge Medical College. Then, I worked at Hindu Rao hospital as senior Gynaecologist & Obstetrician for 5 years, joined Maulana Azad Medical College as Lecturer in 1971 and superannuated from MAMC in 1995. I was Head of the Obstetrics & Gynaecology department from 1982 to 1995. After superannuating from government job, I joined Sir Ganga Ram hospital and am presently working there as Consultant.

Memories of Safdarjung Hospital I did 6 months house job in medicine. That time Dr. Col. R. D. Ayyar was the Medical Superintendent and Dr Col. Rao was the Head of Medicine Department. I remember sharing the dining hall with the nurses at nurses' hostel. I also remember having a very cordial relationship with my other co-residents.

Experience at Sir Ganga Ram Hospital It has lot of advantages of being a multispeciality hospital. Staff here is very helpful, it is like a family. Nobody is cutting each other and everybody is willing to help. If you have a problem, professional or personal, you can sit and discuss with colleagues and they will give you an honest opinion.

Awards and Achievements Best student Prize in Obstetrics & Gynaecology; Stood first in MD examination; awarded a prize from Delhi Medical Association.

I have more than 60 publications to my credit. I was the President AOGD from 1989-1993 and Organizing Chairperson of AICOG in 1992. It was the first gynae conference which was organized in such a large scale.

A piece of advice you want to give to budding gynaecologist They should be honest to their profession and be ethical. Money is not everything, don't kill your conscious.

What does AOGD mean to you I have seen AOGD grow from a scratch, I was a member of this Association as house surgeon. AOGD was established during that time. I was President AOGD for 4 years.

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Milestones

1991	• First IVF baby (Garv) of North India born
1993-1994	• First successful pregnancy by ovum donation
1997-1998	• First pregnancy from cryopreserved embryos
1999-2000	• First pregnancy by intra cytoplasmic sperm injection (ICSI)
2001	• First pregnancy using testicular sperm
	• Surrogacy started
2004	• Freezing of testicular tissue introduced
2006	• Laser assisted ICSI & Assisted Hatching started.
	• Centre recognized by NBE for fellowship in Reproductive Medicine
2007	• Centre recognized by FOGSI for advanced infertility training
2008	• Vitrification of embryos & oocytes started
2009	• First pregnancy of northern India using vitrified oocytes
	• PICSI introduced and first pregnancy reported
2010	• First pregnancy after prenatal genetic screening (using FISH) day 3 Embryo Biopsy
2012 onwards	• Centre has been conducting FNB exit exams annually
2014	• Centre recognized by Indian Fertility society for fellowship in clinical ART.
	• Introduction of 24 chromosome screening of embryos/blastocysts (by a-CGH technique) and reporting of first pregnancy
	• Introduction of PGD (for detection of single gene mutation and translocation errors)
2015	• Centre recognized by ISAR for embryology training.

Consultants

Dr Abha Majumdar
Dr M Kochhar
Dr Shweta Mittal Gupta
Dr Neeti Tiwari
Dr Ruma Satwik
Dr Veena Acharya

Embryology Team

Mr Gaurav Majumdar
Mr Kapil P Kulluwar
Mr Puneet Singh
Mr Arun Kumar
Mr Naveen Sharma

3 Fellows
17 supportive staff

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Events Held

Events held under the aegis of AOGD in December 2015

- **FERTIVISION 2015- 11th Annual National Conference** of IFS on 4th-6th December, 2015 at Hotel Ashok, New Delhi.
- **CME on Immunizations during Pregnancy to** at Fortis Hospital Shalimar Bagh under aegis of Reproductive Endocrinology subcommittee AOGD and DGF North on 8/12/2015, Auditorium Fortis Hospital, Shalimar Bagh Delhi.
- **Health Camp** organised by Dr Rupali Dewan & Dr Nalini Tolia (Safdarjung Hospital) at Aliganj on 9th December, 2015 under **AOGD outreach activities.**
- **Annual Conference and Public Awareness programe of RCOG Northern Zone (UK)** on "Multi Discriplinary Approach to Domestic Violence Against Women" On Sunday, 13th **December** at **auditorium, Sant Parmanand Hospital** to enhance awareness in our society regarding issues related to domestic violence against women.
- **Urogynaeclogy CME** at Hindu Rao Hospital under aegis of Urogynaecology subcommittee of AOGD on on 18th December 2015 from 2-4pm
- **AOGD monthly clinical meeting** on 18th December in Hindu Rao Hospital
- **Cancer Awareness Programme for ASHA** (south Delhi) was held at Safdarjung Hospital on 16th and 23rd December.
- **CME held on "New Aspects of Male Infertility** " & "Role of DHEAS, Melatonin, and Co Q 10 at Fortis Hospital Shalimar Bagh, 24th December under aegis of Reproductive Endocrinology subcommittee AOGD and DGF North at Fortis Hospital, Shalimar Bagh.
- **'Gynae Oncology Update: For Practising Gynecologists'** by FOGSI Oncology committee and AOGD Oncology Subcommittee at GTB on 3rd January, 2016



Cancer awareness programme for ASHA at Safdarjung Hospital

CME on male infertility at Fortis, Shalimar Bagh

Monthly meeting at Hindu Rao Hospital

FERTIVISION 2015 at Ashoka Hotel

Gynae Oncology Update at IHC

AOGD representation at ICOG World Congress

CME on Immunization in pregnancy at Fortis, Shalimar Bagh

International Conference on Critical Care in Obstetrics

Venue: Hotel Grand, Vasant Kunj, New Delhi **Date:** 13th - 14th February, 2016

Advisor
Dr Alka Kriplani
FOGSI President 2016

Organizing Co-Chairperson
Dr Alpesh Gandhi
Dr J C Suri

Organizing Chairperson
Dr Pratima Mittal

Organizing Secretary
Dr Sunita Malik

Scientific Programme

Day 1 : Saturday, 13th February, 2016

08.00-09.00am	**Registration**		
	Dr. N.A. Purandare Hall	**Dr. C.S. Dawn Hall**	**Brig. S.D. Khanna Hall**
Session 1			Free Papers + round table discussions
09.00-10.30am	**Major killer….Sepsis**	**Major responsible…..Severe anaemia**	
	Chairpersons: Dr Ragini Aggarwal, Dr Mitra Saxena, Dr Kiran Aggarwal, Dr Anita Kumar	Chairpersons: Dr Hemant Bhatt, Dr Sujata Sharma, Dr Geeta Radhakrishnan, Dr Harsha S. Gaikwad	
09.00am	How to confirm septicaemia ? Fungemia? Role of Biomarkers?..... Dr Vivek Nangia	Severe anaemia in India…… Pregnancy adds fuel to fire!.....Dr Sadhana Gupta	
09.20am	Evidence based / Guidelines for management of sepsis……. Dr Dhruv Chaudhary	Severely anaemic mother….. Antenatal management options……..Dr Lila Vyas	
09.40-10.30am	Panel discussion….. Sepsis in different conditions (like incomplete abortion, perforation, in drug resistant, chorioamnionitis, in immunocompromised host, it's complications etc.)	Panel discussion on managing mother with preexisting severe anaemia in critical conditions….. in haemorrage, sepsis, during Labour, immediate postpartum period and surgery, Sickle cell anaemia	
	Moderators: Dr Chandravati and Dr Ritu Joshi	**Moderators:** Jaydeep Tank and Dr Madhuri Patel	
	Dr Alok Sharma, Dr Suvarna Khadilkar, Dr Reeti Dutta, Dr Tarini Taneja, Dr Madhu Nagpal, Dr Manju Puri, Dr Sohani Verma	**Panelists:** Dr Anuradha Khanna, Dr Anupam Gupta, Dr Archana Verma, Dr Rajat Mohanty, Dr Sarita Agarwal, Dr Deepti Goswami, Dr Varsha Lahade	
10.30-11.30am	**Inaugration & Tea**		
Session 2			
11.30-12.30pm	**Challenges in obstetric practice**	**Situations worsen during pregnancy…… Thrombophilias**	
	Chairpersons: Dr Jyoti Bindal, Dr M.C.Patel, Dr J.C. Suri, Dr Sikdar	**Chairpersons:** Dr Dilip Walke, Dr Bipin Pandit, Dr Neerja Malik, Dr Neeta Dabai	
11.30am	HDU…….. Need of an hour and it's set up……Dr Pratima Mittal	APLA…..Investigation protocol , monitoring and making journey safe…… Dr Indrani Ganguli	
11.50am	Transfering a critically ill mother…..A challenge…….Dr Shobha Gudi	SLE…….Pregnancy and labour monitoring?.... Ensuring safe delivery……..Dr. Bhaskar Pal	
12.10-12.30pm	When to deliver a foetus in critically ill mother……..Dr Kanwal Gujral	DVT……..How to prevent and manage?..... Dr Achla Batra	
	Chairpersons: Dr Sonawala, Dr Lubna Hassan, Dr Sunita Malik, Dr Amita Suneja	**Chairpersons:** Dr.C.N.Purandare, Dr Sharada Jain, Dr Harsha Khullar, Dr Sangeeta Gupta	
12.30 -01.00pm	Key note address… Educational modules for future. By Dr Alka Kriplani	Key note address…Blood transfusion practices in India: My Experience…..By Dr Alpesh Gandhi	
	Chairpersons: Dr Alka Kriplani, Dr Kamal Buckshee, Dr Alpesh Gandhi, Dr Aruna Batra	**Chairpersons:** Dr Shirish Sheth, Dr Vilasben Mehta, Dr Pratima Mittal, Dr Shalini Rajaram	
01.00-01.30pm	Key note address..Pt's Satety….A main concern in critical care in obstetrics BY Dr S. Arulkumaran	Key note address……. Near Miss Review: A way forward by Dr C.N.Purandare	
01.30-02.00pm	**Lunch**		
Session 3			
02.00-05.00pm	**Life threatening complication….PPH (Video+ Lectures+Panel discussion)**	**HDP and life threatening complications-severe preeclampsia, eclampsia, APH, HELLP**	
	Chairpersons: Dr Sadhana Gupta, Dr A.K.Debdas, Dr Banashree Das, Dr Chitra Rahunandan	**Chairpersons:** Dr Smruti Malvi, Dr Anjali Dabral, Dr Manju Khemani Dr Usha Rani	

02.00pm	Newer modalities(including transfer)in the management of PPH……. Dr Sheela Mane	Early onset hypertension… What to do? How to manage?..... Dr Saswati Chaudhari	
02.20pm	Management of Lower segment bleeding……..Dr Ajesh Desai	New insights into the pathogenesis of pre-eclampsia /eclampsia and its implication on treatment - Dr Jyotsna Suri	
02.40pm	Compression sutures…. Simple way to tackle PPH…….Dr Ajit Rawal	APH……How to save mother and baby?..... Dr Atul Munshi	
03.00pm	Stepwise devascularization and How to avoid it's complications…….. Dr Lakhbir Dhaliwal	HELLP…….. Obstetrician's nightmare…… Dr Alokendu Chatterjee	
03.20pm	Obstetric Hysterectomy……How to do? When to do?..... Dr Asis Mukhopadhyay	Hypertensive crisis….. How to prevent and control?.....Dr Suyajna Joshi	
03.40pm	UAE…..When? Where? How?..... Dr S Tyagi	Algorithm and managing eclampsia with recurrent convulsions…….. Dr Sanjay Gupte	
04.00-05.00pm	Panel discussion on cases of PPH in different critical conditions……(placenta previa with accreta Percreta, Broad ligament hematoma, rupture uterus, bleeding disorders etc)	Panel discussion on how to monitor and manage the patient of severe preeclamsia and eclampsia, best options in planning delivery, precautions at emergency section, it's complications, fluids etc.	
	Moderators: Forgien Faculty	**Moderators:** Dr Girija Wagh and Dr Vivekanand Achanta	
	Panelists: Dr Dipesh Dholakia, Dr Dilip Dutta, Dr Arun Baruah, Dr Tamkin Rabbani, Dr Veena Agrawal, Dr Vanie Thappar, Dr Saritha Shamsunder	**Panelists:** Dr Veerendra kumar, Dr Gorakh Mandrupkar, Dr Sudhir Shah, Dr Sangeeta Kamra, Dr Pragnya Mishra, Dr Kiran Guleria, Dr Maninder Ahuja	

Day 2 : Sunday, 14th February, 2016

08.00-09.00am	**Registration**		
	Dr. N.A. Purandare Hall	**Dr. C.S. Dawn Hall**	**Brig. S.D. Khanna Hall**
Session: 4			
09.00-11.10am	**Critical Situations in L.R./ O.T.....Swift thinking prompt actions**	**Pregnancy with medical disorders - need critical care**	**Operative obstetrics and critical conditions**
	Chairpersons: Dr A.S. Saini, Dr Arti Luthra, Dr Rupali Dewan, Dr Archana Sharma	**Chairpersons:** Dr Manjeet Kaur, Dr Nivedita Sarda, Dr K.K. Roy, Sunita Yadav	**Chairpersons:** Dr Dipesh Dholakia, Dr Sudhir Shah, Dr Abha Singh, Dr M.D. Goswami
09.00am	Cord problems..... How to predict? How to manage?..... Dr Vijay Zutshi	Managing Malaria and it's Critical complications during pregnancy……. Dr Sunita Malik	LSCS in choripamnionitis ……. Dr J. B. Sharma
09.20am	AFE/PE.....Genesis.....When to be alert? What more we can do?.....Dr Hiralal Konnar	Managing Dengue and Critical complications during pregnancy…….. Dr Shibdas Chakravarty	Life threatening anaesthesia complications in O.T……. Dr Rajeev Chawla
09.40am	Acute inversion of uterus....What not to do? What to do?..... Dr Revathy Janakiram	Acute psychiatric crisis in Obstetrics……. Dr Niranjan Chavan	Post operative paralytic ilius…….. Dr H.P Pattnaik
10.00am	Rupture of the uterus.....Changing trends….How to manage?..... Dr Gokuldas	Fulminating Jaundice at Term……. Dr Suchitra Pandit	Unexpected severe bleeding during D & E……. Dr Nozer Sheriar
10.20am	Difficulty in delivery of a baby in LSCS……...Dr Parul Kotadawala	Pregnancy with H1N1 infection…….. Dr Jayam Kannan	Panel discussion….Rare serious & critical complications during obstetrics surgeries and It's solutions
10.40am	Sudden obstetrical collapse..... Immediate resuscitation, spot D/D of causes……..Dr Seema Singhal	Post delivery ARF……..prevention, early measures and management……. Dr Gita Arjun	**Moderators:** Dr.Sunita Ghike & Dr Ragini Verma
11.00am	open house discussion with audience participation	open house discussion with audience participation	**Panelist:** Dr Shyamal Sett, Dr Palaniappan, Dr Nalini Anand, Dr M.G. Hiremath, Dr Smiti Nanda, Brig. S Mohan, Dr Ramaraju H.E.
Session : 5			
11.10am-1.00 pm	**Critically ill foetus**	**Critical endocrinal crisis/ Miscelleneous**	**Rational use of blood / Miscelleneaous.**
	Chairpersons: Dr Archana Baser, Dr Mira Agnihotri, Dr Sudha Prasad, Dr Renu Arora	**Chairpersons:** Dr Dilip Dutta, Dr Rekha Kurian, Dr Suresh Kumar, Dr Raksha Arora	**Chairpersons:** Dr. Sushma Baxi, Dr Bebu Seems Pandey, Dr Anil Jain, Dr P. Singh

11.10am	Foetal monitoring and it's interpretations in high risk mothers……Dr P.K.Shah	Pregnancy and Diabetes Ketoacidosis…….. How to prevent and it's management…….. Dr Vinita Das	Rational use of blood and its components…….. Dr Navneet Magon
11.30am	Severe IUGR/FGR……..further management……..Dr Narendra Malhotra	Pregnancy and Thyrotoxicosis…… Dr Garima Kachhawa	Blood transfussion reactions….. when? why? what to do?..... Dr Shusheela Rani
11.50pm	PPROM…….Dr Anita Singh	Managing Pregnancy with severe Respiratory Problems (Bronchial Asthma/ ARDS)….Dr M.K.Sen	Pregnancy in Transplanted Patient……..Dr Vinnet Mishra
12.10pm	Anaemic foetus……How to monitor? What to do?..... Dr S.Suresh	Managing Recurrent Vasicular Mole and Choriocarcinoma ……. Dr P.K.Sekharan	How to deal critical complications of drugs used in obstetric ……. Dr Durgasankar Das
12.30pm	Critical/emergent condition in the management of twin pregnancy ……. Dr Aparna Sharma	Managing Ectopic Pregnancy with shock……..Dr Prakash Trivedi	Referring a Critically ill obstetric pt…….When to? Why to? Whom to? ..... Dr P.C.Mahapatra
12.50pm	open house discussion with audience participation	open house discussion with audience participation	open house discussion with audience participation
01.00-01.30pm	Key note address…Effects of critical care and MBRACE's on the latest maternal mortality result…... by Dr Paul Fogarty	Key note address ……. Physiological basis of oxytocic response …...by Dr Hemnatha Parera	Key note address… Preparing a critically ill obstetric patient for an operation….. by Dr Lubna Hassan
	Chairpersons: Dr Alka Kriplani, Dr Alpesh Gandhi, Dr Dinesh Baswal, Dr S.B. Khanna	**Chairpersons:** Dr.S. Arulkumaran, Dr Prakash Trivedi, Dr Pratima Mittal, Dr Suneeta Mittal	CHAIRPERSONS: Dr P.C. Mahapatra, Dr Prakash Mehta, Dr Roza, Dr Shashi Prateek
01.30-02.00pm	**Lunch**		
Session 6			
02.00-03.20pm	**Obst. HDU/Obst. ICU……. About concept, set up and procedures**	**Panel discussion on managing rare & interesting critical clinical cases**	**ART and critical obstetric issues**
	Chairpersons: Dr Paul Fogarty, Dr Amita Sharma, Dr Neeraj Gupta, Dr. Mridula Pawar	Pregnancy with Burns, Anaphylatic reactions, Acute abdomen, Acute Pancreatitis, Post CS ARF, Chicken pox at term, Ca ovary etc……	**Chairpersons:** Dr Sonia Malik, Dr G.K.Tripathi, Dr Nalini Mahajan, Dr Shivani Gaur
02.00pm	Identifying and Assessing Critically ill Parturient……..Dr G.C. Khilnani	**Moderator:** Prakash Mehta	ART and critical issues (OHSS and others)…….Dr Rishma Pai
02.20pm	Teamwork and Ethics at HDU and Obst. ICU……..Dr R.K.Mani	**Panelists:** Dr B.S.Jodha, Dr Tushar N. Shah, Dr Rekha Kurian, Dr Vidya Thobi, Dr Deepak Govil, Dr Shyjus Nair, Dr Pushpa Sethi	Obstetric risk assessment in IVF pregnancy……..Dr Jaideep Malhotra
02.40pm	Role of bed side ultrasound and ECHO in the management of critically ill obstetric patient ……. Dr J.C. Suri		Panel discussion on…….. How to prevent and deal pregnancy complications after ART.
03.00pm	Importance and interpretation of Laboratory investigations in critically ill obst pt……Dr Nuzat		**Moderators:** Dr Nandita Palsetkar, Dr Sunita Tendulwadkar
			Panelists: Dr. Bharati Dhorepatil, Dr. Kanthi Bansal, Dr. Kamini Patel, Dr Asha Baxi, Dr Poonam Loomba, Dr Vidya Bhat, Dr Malvika Mishra
Session 7	**Obst. HDU/Obst. ICU……. About Concept, Set up and procedures**	**Cardiac disease in pregnancy**	**Pregnancy complication need urgent care...DIC**
03.20-04.20pm	Chairpersons: Dr Anup Raj Gogia, Dr Reena Yadav, Dr Reva Tripathi, Dr Pawan Gurha	Chairpersons: Dr Uma pandey, Dr Anjali Tampe, Dr Sujata Sharma, Dr Pushpa Singh	**Chairpersons:** Dr Kawita Bapat, Dr Jaishree Sunder, Dr Sushma Rani, Dr Ila Gupta
03.20pm	Fluid, Electrolytes & Nutrition management in critical obst. Care…… Dr Sumit Ray	Pregnancy after cardiac surgery……..... Dr Hemant Despande	DIC…. How to make early diagnosis? How to monitor the pt?.....Dr Krishnendu Gupta
03.40pm	Pain Management in obstetric HDU and ICU…… Dr Saveena Raheja	Post partum Cardiomyopaphy…… Dr Sujata Mishra	Panel discussion on Managing critical conditions in DIC (in IUFD, haemorrage, Sepsis, bleeding disorders, Preparing for surgery etc.)
04.00pm	Infection prevention and control in ICU in context of Obst. practice……Dr Dipak Bhattacharya	CVT…….How to manage?..... Dr Suman Mittal	**Moderators:** Milind Shah and Dr Alka Pandey
	(Central Line Associated Blood Stream infections, Catheter Related, Ventilator associated Infections)	Pulmonary artery hypertension…….... Dr Sandeep Bansal	**Panelists:** Dr Ambigaimeena, Dr Kiran Pandey, Dr.Archana Baser, Dr Shahnaj Taing, Dr Yashodhara Pradeep, Dr Manjula Sharma, Dr Ashok Kumar,

04.20-04.40pm	open house discussion with audience participation	open house discussion with audience participation	
04.40pm	Joint consensus statements by Faculty and Organizers and Valedictory in Hall A.		

Programme for Pre-congress Workshops

ABC of Critical Care Obstetrics

Date: **12th February, 2016** • *Venue:* **Old Lecture Theatre, Safdarjung Hospital, New Delhi**

Course Directors: Dr Pratima Mittal, Dr J. C. Suri **Co-ordinators:** Dr Sunita Malik, Dr Jyotsna Suri

Time	Topic	Speaker
08.15-08.45am	Registration	
08.45-09.00am	Introduction & welcome	Dr Pratima Mittal
09.00-09.30am	Haemodynamic & physiological changes in pregnancy	Dr M. K.Sen
09.30-10.00am	Assessment of the critically ill patients & haemodynamic monitoring	Dr. Jyotsna Suri
10.00-10.30am	Pathophysiology & management of shock in obstetrics	Dr Dhruv Chaudhary
10.30-11.00am	ABG – Systematic approach to interpretation	Dr J. C. Suri
11.00-11.30am	Non invasive ventilation	Dr M. K. Sen
	Mechanical Ventilation	
11.30-12.00noon	- Principles & modes	Dr J. C. Suri
12.00-12.30pm	- Initiation & Monitoring	Dr P. K. Verma
12.30-01.00pm	- Weaning	Dr Shibdas Chakarvarty
01.00-02.00pm	LUNCH	
02.00-05.00pm	**Hands on (six stations of 30 min each)**	
	Airway management, resuscitation of pregnant women	Dr Nikki Sabharwal
	ABG exercises	Dr J. C. Suri
	Ventilatory settings	Dr P. K. Verma
	Oxygen therapy	Dr Shibdas Chakarvarty
	NIV	Dr M. K. Sen
	Neonatal resuscitation	Dr Chellani

Medico-Legal Issues in Critical Care in Obstetric

Date: **12th February, 2016** • *Venue:* **Old Lecture Theatre, Safdarjung Hospital, New Delhi**

Course Directors: Dr Alpesh Gandhi, Dr Pratima Mittal **Co-ordinators:** Dr Achla Batra, Dr Manjula Sharma

08.15-09.00am	**Registration**	
09.30-11.00am	**Session 1 EXPOSED TO**	
	1. When notice served……step wise approach……. 2. Criminal Liabilities in obstetric practice…… 3. How to counsel in critical obstetric situations?..... 4. New Acts/Laws and amendments affecting obstetric practice…….. Audience participation	Dr Jyoti Bindal Dr Charu Mittal Dr Hemant Bhatt
11.30- 01.15pm	**Session 2 STITCH IN TIME…..ACTION**	
	5. Death on Table…….. 6. Sudden IUFD in LR…… 7. Obstetric Hysterectomy in Primigravida……. 8. MTP perforation…… 9. Critical ART complications in surrogate mother……. Audience participation	Dr Dilip Walke Dr Hitesh Bhatt Dr Manish Machave Dr M.C. Patel Dr Geetendra Sharma
01.15- 01.50pm	**Lunch**	
01.50-03.00pm	**Session 3**	
	10. Post mortem examination……when and why?..... 11. Court compensations….. Is there any limit?..... 12.Landmark cases and court's judgements……. Audience participation	Dr Sanjay Gupte Dr Bipin Pandit Dr Hitesh Bhatt
03.00- 03.50pm	**Session 4 COURTROOM CULTURE (Dr M. C. Patel)**	
	Moot Court (3 HIGH RISK OBST OR ICU CASES)	
03.50-04.50pm	**Session 5 DISCUSSING THE DEADLY……Panel Discussion**	
	Moderator: Dr. Geetendra Sharma and Dr Bipin Pandit Medico-Legal issues/cases in Critically ill Obstetrics Patients *Panellists:* **Dr Mahesh Jariwala, Dr Indu Chawla & Dr Archana Misra** CDHO/Collector, Medicolegal counsellor, Advocate, Police officer, Magistrate or Judge	

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Blood Transfusion and Blood Components in Obstetrics

Tania G. Singh
Director, Bodyline Trauma and Maternity Center, New Delhi

Introduction

Blood transfusion is a life saving intervention and is an essential part of modern health care. However, as with any therapeutic intervention, it may result in acute or delayed complications and carries the risk of transmission of infectious agents, though rare nowadays.

Total blood volume in an adult is approximately 7% of body weight or 70 mL/kg. The separation of whole blood into it's constituent components – red cells, platelets and plasma is widely practised for use when these specific components only are required.

Preliminaries before requesting for blood

- It's clinical need; indication
- When it is required (date /time); it's urgency
- Inform the patient's relatives and document the same (i.e. informed consent explaining the risks, benefits and alternatives to transfusion)
- Enquire about any transfusion and/or transfusion reaction history
- Select the blood product and the quantity required
- Complete the blood requisition form accurately and legibly
- If the blood is needed urgently, contact the bloodbank by telephone

If the particular blood group or blood product is available

The patient should be identified and blood sample should be obtained for compatibility testing in a tube with no anticoagulant. The tube should be labelled correctly with patient's complete name, hospital registration number, ward and date, at the patient's bedside**.** *Do not* pre label the tube before sample is obtained.The tube should not be taken to the nursing station before labelling or given to second staff for labelling.The blood sample along with the blood request form should be sent to the blood bank.

If required blood group is not available, give alternative blood as packed cells

Patient Blood group	Alternative Blood group
O	None
A	O
B	O
AB	A; B; O

Administration of blood products

Only 0.9% sodium chloride can be infused in the same line as the blood. A break in the integrity of the infusion line may increase the risk of bacterial contamination of the component. If using a pre-existing IV site, assess the quality of the line by checking for swelling, redness or pain at the infusion site. The size of peripheral I/V access should be 18-20 Gauge preferably.

Compatible solutions with blood products

The practice of priming or flushing administration sets used for the transfusion of blood components with isotonic (0.9%) saline is not evidence based. 5% Dextrose in water will cause clumping of red cells and haemolysis and should be used only when administering I/V immunoglobulin. Lactated Ringer's may cause clotting due to calcium content.

Filter

Use a new sterile blood administration set containing an integral 170-200 micron filter. The set should be changed at least 8 hourly during blood component infusion. In a very warm climate, change the set more frequently and usually after every four units of blood, if given within a 12 hour period. Use a fresh blood giving set for platelets as platelets must NOT be infused through a set that has already been used for red cells as this may result in a loss of platelet yield in the filter

Blood warming

There is NO evidence that warming blood is beneficial to the patient when infusion is slow. Keeping the patient warm is more important than warming the infused blood. Warm the blood in blood warmer only. Blood should never be warmed in a bowl/oven, or inserting the pack in warm water as this can result in hemolysis of red cells

Warmed blood is commonly required in 'Large volume rapid transfusion' as this can lead to hypothermia and arrhythmias or cardiac arrest, or impaired coagulation in surgical or trauma patients; in cases of exchange transfusion in infants and in patients with clinically significant cold agglutinins.

Receiving the blood bag from blood bank

- Check the compatibility label on the blood bag; blood pack number
- Patient's name, hospital reference number, ward number
- Patient's ABO and Rh D group
- Date of compatibility test, it's expiry date
- Blood group of blood pack
- Return blood promptly to blood bank if not used

Checking the blood pack

Check for:

- Any sign of hemolysis in the plasma indicating that the blood has been contaminated, allowed to freeze or had become too warm
- Any sign of contamination, such as a change of colour in the red cells, which often looks darker or purple/ black when contaminated
- Any clots, which may mean that: the blood was not mixed properly with the anticoagulant when it was collected or there may be bacterial contamination due to the utilization of citrate by proliferating bacteria
- Any signs that there is a leak in the pack or that it has already been opened
- Do not administer the transfusion if the blood pack appears abnormal or damaged and inform the blood bank immediately

Blood transfusion notes

General Information

- Patient's name, hospital reference number, patient's ward and her ABO Rh D group
- Date of compatibility test and it's expiry date
- Blood group of blood pack and blood bag number

Monitoring

When?

- Before starting the transfusion
- As soon as the transfusion is started
- 15 minutes after starting the transfusion
- Afterwards hourly monitoring
- On completion of the transfusion
- 4 hours after completing the transfusion

Document

- Time of starting and completion of transfusion
- Volume and type of all products transfused
- PR, BP, RR, temperature, fluid intake
- Any adverse effects and measures taken

Blood transfusion reactions

A hypersensitivity reaction is suspected if ≥ 1 clinical signs appear during or up to 4 hours after the transfusion

Mild Reactions

Mucocutaneous signs: Urticaria (localized or generalized); generalized edema; pruritis; angioedema of the face or mucous membranes *(Quinckeedema)*

Management

Slow down the transfusion and monitor vital signs every 15 minutes until stable. Administer paracetamol ± antihistamine I/M. If symptoms worsen, manage as moderate/severe reaction.

Moderate reactions

Involvement of atleast 2 organ systems:

Mucocutaneous signs (pruritis, urticaria); CVS symptoms (tachycardia, palpitations, mild hypotension); Respiratory symptoms: cough, mild dyspnoea, bronchospasm, fever, rigors, headache; Gastrointestinal involvement

Management

Stop the transfusion but do not discard the blood unit. Replace the infusion set and keep I/V line open with normal saline. Administer antipyretic and antihistamine I/M. Give I/V glucocorticoids and bronchodilators, if there are anaphylactic reactions like bronchospasm, stridor. When there is clinical improvement, restart infusion with a new blood bag and monitor closely

Severe reactions

Hypotension (fall in systolic BP by > 20%); tachycardia (rise in heart rate by >20%) or severe bradycardia or arrhythmia. Chest pain, fever, rigors, headache; severe dyspnoea; loin/back pain; haemoglobinuria; unexplained bleeding (DIC)

Management

Stop the transfusion IMMEDIATELY and start normal saline. Elevate the patient's legs and provide high flow oxygen by mask. Attach the pulse oximeter and administer:

- Inj. Adrenaline 0.01mg/kg body weight slowly I/M
- I/V corticosteroids and bronchodilators
- Diuretic (Frusemide 1mg/kg body weight)
- Antipyretic (paracetamol)
- Send blood samples and 1st urine sample post reaction for analysis
- *Blood Samples:* Group and antibody screen, crossmatch, IgA Levels, Mast Cell Tryptase, blood cultures, plasma haemoglobin, haptoglobin, coagulation profile, FDPs

and bilirubin
- Assess for bleeding from puncture sites till investigation reports come. If there is clinical or laboratory evidence of DIC, manage with platelets, FFPs, cryoprecipitate
- Inotropes can be started, if hypotension continues even with fluids

Delayed transfusion reactions

Delayed haemolytic Transfusion Reactions may occur 2-14 days after transfusion. Most are unrecognised or clinically benign.

Blood Components

Whole blood
- Volume-: 400-500mL
- Anticoagulant-preservative sol: 63mL
- Hemoglobin: min. 45gm
- Hematocrit: 45-55%
- Packed red cells: 120-250mL
- Plasma: 200-300mL
- Maximum storage time at +2°-6°C is 21-35 days
- Must be ABO and Rh compatible with the recipient
- Start the transfusion within 30 minutes of removing from refrigerator and must be completed within 4 hours

PackedRBCs
- Volume: 300 (220-340) mL
- Contain mostly red cells mixed with preservatives and anticoagulants
- Hematocrit: 55-75% (very viscous)
- For rapid transfusion, can be diluted with 100 mL normal saline
- RL should not be mixed with RBCs because it's calcium content may precipitate when it interacts with citrate preservatives
- Packed RBCs have NO platelets and only ≈ 10-15 mL of residual plasma
- 1 unit of RBCs increases hemoglobin by 1 gm/dL and PCV by 3% points
- For 4 units of Packed RBCs, give 1 unit FFP
- Maximum storage time at +2° to +6°C is 21-35 days
- Must be ABO and Rh compatible with the recipient
- Complete transfusion within 4 hours of commencement

Fresh frozen plasma (FFP)
- Is a component of whole blood that remain once platelets and cellular elements are removed
- Prepared either from single unit of whole blood (Random donor FFP) which has a volume of ≈ 250 mL or from plasma collected by apheresis technique (Jumbo FFP) with a volume of ≈ 800 mL
- Frozen at -18 to -30°C within 6 hours of collection and can be preserved for 1 year
- Contents: All coagulation factors including Factor VIIIc>0.7 IU/mL and other proteins present in original blood
- It should be ABO compatible, <u>not</u> necessary Rh specific
- Plasma volume in adults is ≈ 40 mL/kg, this requires a dose of FFP of ≈ 15 mL/kg, therefore 2 units should be given at a time
- It should be transfused when PT and/or aPTT is >1.5 times normal
- 1 unit FFP increases fibrinogen by 10 mg/dL
- Use within 6 hours of thawing and infused over 30-60 minutes

Cryoprecipitate
- Prepared by thawing 1 unit of FFP at 1-6°C and collecting the formed precipitate in a concentrated volume of 10-40 mL/bag
- Each bag of cryoprecipitate contains:
  - 200-300 mg/dL fibrinogen
  - 100 units of factor VIII
  - vWF, factor XIII and
  - 55 mg fibronectin in equal volume of 15-20mL/bag
- Usually supplied as a single donor pack or a pack of 6 or more single donor units that have been pooled
- Usually infused over 30 minutes
- Indication for transfusion:
  - Fibrinogen < 75-100 mg/dL
  - 1U/10 kg of body weight with fibrinogen < 75mg/dL
- Each unit increases fibrinogen by 10 mg/dL
- Can be stored for 1 year at below –25oC
- If possible, use ABO-compatible product
- No compatibility testing is needed

Platelet concentrates
- Indication: < 50000/mm^3 plateletin presence of diffuse microvascular bleedings
- Goal: minimum 1 lakh/mm^3
- In non-bleeding patients, a goal of 20,000/mm^3 is usually taken but can be taken as 10,000/mm^3 as the difference between the two is clinically not significant
- Commence infusion within 30 minutes of collection and finished over a period of 30-60 minutes

Platelets collected from Whole blood donations

- ABO typing is NOT essential but Rh typing is necessary
- In case Rh negative platelets is required and is not available, transfuse Rh positive platelets with a cover of Anti D immunoglobulin
- Dosage: 1 unit of platelet concentrate/10kg body weight
- Shelf life is 5 days at 20-24^{o}C when stored under continual gentle agitation on a platelet mixer in blood bank
- Platelets must not be refrigerated

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CLINICAL GUIDELINES

Adnexal Masses in Pre-Menopausal Women: A Diagnostic Dilemma

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Any mass arising from the ovary, fallopian tubes, or surrounding connective tissues, is defined as an adnexal mass but other pelvic masses may also clinically present as an adnexal mass. They represent a diagnostic dilemma to the clinician because of wide variety of eiologies. The differential diagnosis of adnexal mass includes benign and malignant gynaecologic and non gynaecological etiologies (Table 1).

Table 1: Differential diagnosis of adnexal masses

TYPE	Benign	Malignant
Gynaecological	Tuboovarian abscess	Epithelial cancer
	Ectopic pregnancy	Germ cell tumour
	Endometrioma	Stromal tumour
	Functional cyst	
	Hydrosalpinx	
	Leiomyomata	
	Mature teratoma	
	Mucinous cystadenoma	
	Serous cystadenoma	
Non-gynaecological	Appendiceal abscess or mucocele	Gastrointestinal cancer
	Bladder diverticulum	Metastasis
	Diverticular abscess	Retroperitoneal sarcoma
	Nerve sheath tumour	
	Pelvic kidney	
	Ureteral diverticulum	

Clinical evaluation

When an adnexal mass is found, the initial work up must focus on identifying acute pathology, which might require immediate surgical intervention[1]. The majority of adnexal masses in premenopausal women will present in a less acute manner, warranting a thorough evaluation.

In premenopausal women functional cysts, leiomyomata, and ectopic pregnancy are more commonly seen and ovarian cancers are rare. *However the most critical and crucial step in evaluation is to differentiate between benign and malignant conditions*.

History and examination

A thorough medical history should be taken from the woman with specific attention to risk factors or protective factors for ovarian malignancy and a family history of ovarian or breast cancer[2]. In the acute presentation with pain the diagnosis of ectopic or accident to the ovarian cyst should be considered (torsion, rupture, haemorrhage). Tuboovarian abscesses tend to be found in the patient with purulent vaginal discharge and fever. Infrequently, premenopausal patients with acute symptomatology can have a malignancy that presents with rapid growth or acute rupture[1]. Some nonacute gynecologic adnexal masses also have specific symptoms. Women with an endometrioma often report cyclical pain that worsens with menses or give a history of infertility. Similarly women with leiomyomas often report pelvic pain or pressure with abnormal uterine bleeding.

Women with adnexal malignancy tend to have less specific symptoms. Often, women with benign adnexal masses are asymptomatic or experience many of the same vague symptoms seen in women with malignancy. Physical examination should include a thorough abdominal and pelvic examination to define the size, consistency, and mobility of the adnexal mass. Ascites, lymphadenopathy, pleural effusion, nodularity along the uterosacral ligaments, solid consistency, and fixation within the pelvis are all features that may be present in women with adnexal malignancy[1].

Ultrasonography

Pelvic ultrasound remains the mainstay of evaluation with up to 90% of adnexal masses being adequately characterized by ultrasound alone[3,4]. Ultrasonography should assess the size, mass characteristics (cystic, solid, or both), complexity (internal septae, excrescences and papillae), and the presence or absence of abdominal or pelvic fluid (ascites or blood). The combination of ultrasonography and Doppler flow studies is superior to either alone[5].

According to an American College of Radiology guidelines, simple cysts in premenopausal women are considered benign[5]. Complex masses may rarely be malignant in premenopausal women[1]. These masses are most likely to be hemorrhagic cysts or endometriomas; however, tubo-ovarian abscess, ectopic pregnancy, and

ovarian torsion can also present as a complex mass. Solid masses are most commonly pedunculated fibroids, but can be benign ovarian tumors, fibromas, thecomas ovarian tumors, or an ovarian torsion.

Other imaging modalities

Computed tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography (PET) are not recommended for initial evaluation of adnexal masses, and their use after transvaginal ultrasound is of limited utility (Table 2).

Table 2: Imaging modalities for the evaluation of adnexal masses[1].

Modality	Sensitivity	Specificity	Positive Likelihood Ratio	Negative Likelihood Ratio
Doppler ultra-sonography	86%	91%	9.6	0.16
MRI	91%	88%	7.6	0.10
CT	90%	75%	3.6	0.13
Gray-scale Transvaginal Ultra-sonography	91%	81%	3.3	0.19
PET	67%	79%	3.2	0.42

If the diagnosis of an adnexal mass remains uncertain after ultrasound, a second imaging modality can be used to aid in determining the risk of malignancy. Magnetic resonance imaging is generally the next imaging modality of choice[6]. Gadolinium contrast MRI is helpful in differentiating and characterizing the origin of pelvic masses[7]. Computerized tomography is better utilized to evaluate for abdominal metastasis including involvement of the omentum, peritoneum, liver, and lymph nodes[1]. Positron emission tomography and computed tomography can further increase the detection of nodal metastasis, but is not routinely used in the preoperative evaluation of adnexal masses[8].

Lab evaluation

A urine pregnancy test should be performed in any woman of reproductive age who presents with an adnexal mass. If the pregnancy test is positive, then a transvaginal pelvic ultrasound should be performed to evaluate for an intrauterine pregnancy or ectopic.

A complete blood count with differential is useful if PID or tubo-ovarian abscess is suspected. Patients with these conditions usually have an elevated white blood cell count with a predominance of neutrophils.

Serum marker screening

Once emergency has been ruled out, the next step is evaluation for malignancy and should include serum marker screening. Currently there is no screening test or serum marker to reliably detect ovarian cancer at an early stage[1]. CA 125 is the most studied serum marker in adnexal masses and is widely accepted as an adjunctive test to determine the risk of malignancy[1]. In premenopausal women CA-125 is unreliable in differentiating benign from malignant ovarian masses because of the increased rate of false positives and reduced specificity. This is as a result of CA-125 being raised in numerous conditions including fibroids, endometriosis, adenomyosis and pelvic infection. Consequently a raised serum CA-125 should be interpreted cautiously.

HE4 (Human epididymis protein 4) is a specific and sensitive ovarian cancer marker and is elevated in serum from women with ovarian cancer and its expression in normal tissues, including ovary, is low. It has been observed that using HE4 alone or in combination with CA125 may improve the accuracy for detection of ovarian cancer at an earlier stage[9].

ACOG[1] and RCOG[2] guidelines recommend:

- A serum CA-125 assay is not necessary when a clear ultrasonographic diagnosis of a simple ovarian cyst has been made.
- α-FP and hCG should be measured in all women under age 40 with a complex ovarian mass because of the possibility of germ cell tumours. ACOG also recommends measuring LDH in these women.

Estimation of risk of malignancy

Several recent studies have demonstrated that ovarian cancer patients managed by gynecologic oncologists and at high volume institutions are more likely to undergo complete surgical staging, and optimal cytoreductive surgery with fewer complications and better survival rates than patients treated by surgeons less familiar with the management of ovarian cancer. At present the **Risk of Malignancy Index (RMI)**[10] is the most widely used model to refer a patient to a gynaecological oncologist. It is simple to use and reproducible but its utility is negatively affected in the premenopausal woman primarily because of increased level of CA-125 in benign conditions also.

It is a product of three presurgical features: the ultrasound scan score, the menopausal status and the serum CA-125 level (IU/ml) and is calculated as follows:

RMI = U x M x CA-125.

RMI has a sensitivity of 78% (95% CI 71-85%), specificity of 87% (95% CI 83-91%)[11,12].

ROMA is another qualitative serum test that combines the results of 2 biomarkers - HE4, CA125 and menopausal

status into a single score. ROMA has 100% sensitivity at 74.5% specificity, a positive predictive value (PPV) of 13.8% and a negative predictive value (NPV) of 100% for stratification of premenopausal women with epithelial ovarian cancer into low likelihood and high likelihood groups of having malignancy.

Recent studies have shown a specific model of ultrasound parameters, the ultrasound 'rules' derived from the International Ovarian Tumor Analysis (IOTA) Group, to have increased sensitivity and specificity[13,14] (Table 3). Using these rules the reported sensitivity was 95%, specificity 91%, positive likelihood ratio of 10.37 and negative likelihood ratio of 0.06.

Table 3: IOTA Group ultrasound 'rules' to classify masses as benign (B-rules) or malignant (M-rules)

B-rules	M-rules
Unilocular cyst	Irregular solid tumour
Presence of solid components where the largest solid component <7mm.	Ascites
Presence of acoustic shadowing	At least four papillary structures
Smooth multilocular tumour with a largest diameter <100mm.	Irregular multilocular solid tumour with largest diameter ≥100mm.
No blood flow	Very strong blood flow

Women with an ovarian mass with any of the M-rules ultrasound findings should be referred to a gynaecological oncological service. Women who do not fit in either group should be further evaluated by another method.

The American College of Obstetricians and Gynecologists and the Society of Obstetricians and Gynaecologists of Canada consider the following features suspicious for ovarian malignancy and their presence would warrant referral to a gynaecological oncologist in premenopausal women- serum CA-125 of more than 200 units/ml; ascites; evidence of abdominal or distant metastasis; a first-degree relative with breast or ovarian cancer.

Management of adnexal masses

The management of an adnexal mass depends upon the location and etiology of the mass and the characteristics of the patient. In general, there are three options for managing an ovarian mass- expectant management, surveillance and surgery.

- **Expectant management**- According to RCOG guidelines 2011, younger, pre-menopausal women with asymptomatic simple ovarian cysts < 50 mm diameter, in non-emergent situations like pain, do not require follow up since these cysts are most likely physiological and resolve within 3 menstrual cycles. There is no evidence that oral contraceptive use can promote the resolution of functional ovarian cysts[2].
- **Surveillance**- As per RCOG, simple cysts between 50-70 mm in diameter should have yearly ultrasound follow up, and those larger than 70 mm should be considered either for further imaging (MRI) or surgical intervention due to difficulties in examining. Whereas, ACOG recommends no intervention in simple cysts upto 10cm. Continued surveillance is indicated if the suspicion of malignancy is low, but it has not been completely excluded. Surveillance usually includes serial pelvic ultrasounds and/or measurement of serum tumor markers. During surveillance, if the mass, increases in size to ≥ 10 cm, or the CA 125 increases or it develops features of malignancy further evaluation/ surgery is done. The optimal interval for repeat USG is controversial and varies from 4 to 12 weeks.
- **Surgery** can be performed by laparotomy, traditional laparoscopy, laparoendoscopic single-site, or robotic approach based on the mass characteristics, and patient or provider preference. Regardless of the surgical approach attention should be paid to limit the potential for cyst or tumor rupture or dissemination. With benign conditions such as teratomas and early-stage cancers any spillage of contents can increase morbidity and mortality.

Surgery is performed for the following indications- if malignancy is suspected; there are other risks associated with the mass (eg, torsion, infection); or the mass is symptomatic. Many ovarian masses will neither be obviously malignant nor clearly benign. In this case surgical excision is needed for a pathologic diagnosis. If clinical suspicion favors benign pathology, consideration can be given to ovarian cystectomy rather than oophorectomy. Unilateral oophorectomy or salpingo-oophorectomy is indicated in patients in whom ovarian tissue cannot be preserved, torsion with necrosis, abscess unresponsive to antibiotics or in definitive treatment for endometriosis. In perimenopausal women and women over 35 years who have completed their family and have high risk factors for development of malignancy should have bilateral salpingoopherectomy.

Aspiration of cysts

Aspiration of ovarian cysts, either vaginally or laparoscopically, is associated with a high rate of recurrence[2]. ACOG does not recommend aspiration of cysts whereas, according to RCOG it can be done in selective cases. In premenopausal women, ultrasound guided needle aspiration of cysts had similar resolution rates as expectant management (44.6% versus 46% respectively[15] Transvaginal or laparoscopic aspiration may be an appropriate management in selected cases only among premenopausal women, after discussion with patient.

Conclusion

Evaluation of adnexal masses is a challenge for the gynaecologists. It is important not miss a malignant mass and at the same time not to over treat an entirely benign pathology. Following the established guidelines helps the clinician to take the right decision. In case a clear cut diagnosis cannot be made inspite of using all diagnostic modalities, it is advisable to use a surgical approach so as to get the tissue for histopathological evaluation.

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Recent Advances in the Management of Acute Pelvic Inflammatory Disease

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Pelvic inflammatory disease (PID) comprises a spectrum of inflammatory disorders of the upper female genital tract, including any combination of endometritis, salpingitis, tubo-ovarian abscess, and pelvic peritonitis[1]. Although sexually transmitted organisms, especially N. gonorrhoea and C. trachomatis, are implicated in many cases, recent studies suggest that the proportion of PID cases attributable to N. gonorrhoeae or C. trachomatis is declining; only <50% cases of acute PID test positive for either of these organisms[2,3]. Newer data suggest that M.genitalium may play a role in the pathogenesis of PID and might be associated with milder symptoms[2,4]. Other organisms which have been implicated are G.vaginalis, Haemophilus influenzae, Streptococcus agalactiae, Cytomegalovirus, M. hominis and U. urealyticum.

Diagnostic considerations

PID is difficult to diagnose because of the wide variation in symptoms and signs associated with this condition. Delay in diagnosis and treatment contributes to inflammatory sequelae in the upper reproductive tract. Laparoscopy provides more accurate diagnosis and a more complete bacteriologic diagnosis. However, its use is not easily justifiable when symptoms are mild or vague. Moreover, laparoscopy will not detect endometritis and might not detect subtle inflammation of the fallopian tubes. Consequently, a diagnosis of PID usually is based on clinical acumen and imprecise clinical findings[5].

Data indicate that a clinical diagnosis of symptomatic PID has positive predictive value (PPV) for salpingitis of 65%–90% compared with laparoscopy[6]. The PPV of a clinical diagnosis is higher among sexually active young women (particularly adolescents), women attending STD clinics, and those who live in communities with high rates of gonorrhea or chlamydia. Regardless of PPV, no single historical, physical, or laboratory finding is both sensitive and specific for the diagnosis of acute PID.

Although some cases are asymptomatic, others are not diagnosed because the patient or the health-care provider fails to recognize the implications of mild or nonspecific symptoms or signs (e.g., abnormal bleeding, dyspareunia, and vaginal discharge).

Presumptive treatment for PID should be initiated in sexually active young women if they are experiencing **pelvic or lower abdominal pain (**if no cause for the illness other than PID can be identified) and if **one or more** of the following minimum clinical criteria are present on pelvic examination.

Minimal clinical criteria

Cervical motion tenderness or *Uterine tenderness* or *Adnexal tenderness*

Additional criteria

One or more of the following additional criteria can be used to support the diagnosis of PID:

- oral temperature >101˚F (>38.3˚C)
- abnormal cervical mucopurulent discharge or cervical friability
- presence of abundant numbers of WBC on saline microscopy of vaginal fluid
- elevated erythrocyte sedimentation rate
- elevated C-reactive protein
- laboratory documentation of cervical infection with N. gonorrhoeae or C. trachomatis.

Most women with PID have either mucopurulent cervical discharge or evidence of WBCs on wet prep. In the absence of these, diagnosis of PID is unlikely and alternative causes of pain should be considered.

Specific criteria

The most specific criteria for diagnosing PID include:

- endometrial biopsy with histopathologic evidence of endometritis;
- transvaginal sonography or magnetic resonance imaging techniques showing thickened, fluid-filled tubes with or without free pelvic fluid or tubo-ovarian complex, or Doppler studies suggesting pelvic infection (e.g., tubal hyperemia); or
- laparoscopic findings consistent with PID.

Treatment[7]

Treatment should be initiated as soon as the presumptive diagnosis has been made because prevention of long-

term sequelae is dependent on early administration of appropriate antibiotics. Although the optimal treatment regimen and long-term outcome of early treatment of women with subclinical PID are unknown, it must provide empiric, broad spectrum coverage of likely pathogens especially N. gonorrhoeae and C. trachomatis because negative endocervical screening for these organisms does not rule out upper-reproductive–tract infection. Treatment regimen should be selected on the basis of availability, cost, and patient acceptance[8]. In women with PID of mild or moderate clinical severity, parenteral and oral regimens both appear to have similar efficacy.

Management of PID in outpatient setting

Information on current and recent medication as well as hormonal contraception should be obtained and their interaction with antibiotic therapy should be assessed. The recommended and alternative regimen is shown in the boxes below.

Recommended intramuscular/oral regimens

Ceftriaxone 250 mg IM in a single dose/ **Cefoxitin*** 2 g IM in a single dose and **Probenecid**, 1 g orally administered concurrently in a single dose/ **ceftizoxime or cefotaxime**)

PLUS
Doxycycline 100 mg orally twice a day for 14 days
WITH** or WITHOUT

Metronidazole 500 mg orally twice a day for 14 days

**Cefoxitin has a better evidence base for the PID treatment than ceftriaxone but is not easily available*

***The recommended third-generation cephalsporins are limited in the coverage of anaerobes. Thus, addition of metronidazole to treatment regimens with third-generation cephalosporins should be considered.*

Note- No clinical trials are available that have assessed combination of only doxycycline and metronidazole (without ceftriaxone). Its use in isolation is not recommended.

Alternative regimen

Intramuscular ceftriaxone 250 mg single dose followed by azithromycin 1gm/week for 2 weeks

Data supporting azithromycin monotherapy for PID is limited and its use without ceftriaxone is not recommended

Hospitalization

Hospitalization is indicated in patients who present with any of the following conditions

- surgical emergencies (e.g., appendicitis) cannot be excluded
- Tubo-ovarian abscess
- PID in pregnancy
- severe illness, nausea and vomiting, or high fever
- unable to follow or tolerate an outpatient oral regimen
- no clinical response to oral antimicrobial therapy.

Parenteral treatment

Inpatient antibiotic treatment should be based on intravenous therapy. Clinical experience should guide decisions regarding transition to oral therapy, which usually can be initiated within 24–48 hours of clinical improvement. In women with tubo-ovarian abscesses, at least 24 hours of inpatient observation is recommended.

Recommended parentral regimens

Cefotetan 2 g IV every 12 hours/ Cefoxitin 2 g IV every 6 hours/ Ceftriaxone 1g IV every 12 hours
PLUS

*Doxycycline 100 mg orally or IV every 12 hours

PLUS

**Tab Metronidazole 400 mg twice daily for 14 days

OR

Clindamycin 900 mg IV every 8 hours

PLUS
Gentamicin loading dose IV or IM (2 mg/kg), followed by a maintenance dose (1.5 mg/kg) every 8 hours. Single daily dosing (3–5 mg/kg) can be substituted.

*Doxycycline should be administered orally because of similar bioavailability with oral and IV doxycycline and severe pain associated with intravenous infusion.

**However, when tubo-ovarian abscess is present, clindamycin (450 mg orally four times daily) or metronidazole (500 mg twice daily) should be used to complete at least 14 days of therapy with doxycycline to provide more effective anaerobic coverage than doxycycline alone.

Alternative parenteral regimens

Ampicillin/Sulbactam 3 g IV every 6 hours

PLUS
Doxycycline 100 mg orally or IV every 12 hours

OR

Azithromycin (500 mg IV daily for 1–2 doses, followed by 250 mg orally daily for 12–14 days)

With or Without Metronidazole

OR

Azithromycin (1 g orally once a week for 2 weeks in combination with ceftriaxone 250 mg IM single dose)

With or Without Metronidazole

Limited data are available to support the use of other parenteral regimen.

As a result of the emergence of quinolone-resistant N. gonorrhoeae, regimens that include a quinolone agent are no longer routinely recommended for the treatment of PID. If allergy precludes the use of cephalosporin therapy, if the community prevalence and individual risk for gonorrhea are low, and if follow-up is likely, use of fluoroquinolones for 14 days (levofloxacin 500 mg orally once daily, ofloxacin 400 mg twice daily, or moxifloxacin 400 mg orally once daily) with metronidazole for 14 days (500 mg orally twice daily) can be considered[9].

Other management considerations

To minimize disease transmission, women should be instructed to abstain from sexual intercourse until therapy is completed, symptoms have resolved, and sex partners have been adequately treated. All women who have been diagnosed as acute PID should be tested for HIV, as well as gonorrhoea and chlamydia, using nucleic acid amplification test (NAAT). Counselling of the patient regarding the following issues is important:

- Following treatment, fertility is usually maintained but there remains a risk of future infertility, chronic pelvic pain or ectopic pregnancy
- Repeat episodes of PID are associated with an exponential increase in the risk of infertility
- Future use of barrier contraception will significantly reduce the risk of PID
- The need to screen her sexual partners for infection to prevent her from becoming reinfected
- Clinically more severe disease is associated with a greater risk of sequeale
- The earlier treatment is given the lower the risk of future fertility problems

Follow-up

- Women should demonstrate clinical improvement within 3 days after initiation of therapy.
- If no clinical improvement has occurred within 72 hours after outpatient therapy, hospitalization, assessment of the antimicrobial regimen, and exclusion of competing diagnoses are recommended.
- Women who have received a diagnosis of chlamydial or gonococcal PID should be retested 3 months after treatment, regardless of whether their sex partners were treated[10].
- If retesting at 3 months is not possible, these women should be retested whenever they next present for medical care in the 12 months following treatment.

Management of sex partners

- Men who have had sexual contact with a woman with PID during the 60 days preceding her onset of symptoms should be evaluated, tested, and presumptively treated for chlamydia and gonorrhea, regardless of the etiology of PID or pathogens isolated from the woman.
- If a woman's last sexual intercourse was >60 days before onset of symptoms or diagnosis, the most recent sex partner should be treated.
- Male partners of women with PID frequently are asymptomatic. Arrangements should be made to link male partners to care.
- Partners should be instructed to abstain from sexual intercourse until they and their sex partners have been adequately treated

Special considerations

HIV infection

Women with HIV infection have clinically more severe PID but respond equally well to the same antibiotic regimens as women without HIV.

Intrauterine contraceptive devices

The risk for PID associated with IUD use is primarily confined to the first 3 weeks after insertion. If an IUD user receives a diagnosis of PID, the IUD does not need to be removed.[11] However, the woman should receive treatment according to these recommendations and should have close clinical follow-up. If no clinical improvement occurs within 48–72 hours of initiating treatment, providers should consider removing the IUD.

Conclusion

The diagnosis of PID is mainly based on clinical criteria. Aggressive and timely treatment of acute PID goes a long way in preventing its long term sequel of infertility, ectopic pregnancy and chronic pelvic pain. Counselling and partner treatment are also an integral part of the management of acute PID.

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Recurrent Vaginal Discharge - A Challenge for the Gynaecologist

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Most women experience one or more episodes of abnormal vaginal discharge during their lifetime, many of these will respond to the over-the-counter medications or treatment provided by the health care providers. However a significant number of the women experience recurrent episodes of abnormal and/or excessive vaginal discharge, a condition that is distressing for both women and their health care providers.

History alone may not provide accurate diagnosis, therefore to determine the aetiology of vaginal discharge, detailed history should be combined with examination and in clinic testing of the discharge (pH, KOH test and microscopic examination). In settings where pH paper, KOH, and microscopy are not available, laboratory testing for gram staining of air dried vaginal discharge can be used to diagnose vaginitis.

Although three conditions most frequently associated with vaginal discharge are Bacterial vaginosis (BV), Candidiasis and Trichomonas vaginalis, cervicitis can also cause abnormal discharge. Women with vulval inflammation where no pathogen can be identified should be evaluated for mechanical or chemical irritation and allergic conditions. If there is recurrence of symptoms or failure of initial treatment which can be syndromic management, women should be evaluated for the aetiology. (Refer to the first issue of AOGD Bulletin for syndromic management)

Bacterial Vaginosis- Women diagnosed with *Bacterial Vaginosis can be treated with Metronidazole 400 mg twice a day for 7 days* or any of other alternative regimens: Tinidazole2 g orally once daily for 2 days, **or** Tinidazole1 g orally once daily for 5 days, **or** Clindamycin 300 mg orally twice daily for 7 days, **or** Clindamycin ovules 100 mg intravaginally once at bedtime for 3 days. Regimens with Metronidazole gel 0.75% and Clindamycin cream are not mentioned as they are not available in India. With BV, persistence and recurrence are common and women should be counselled regarding the same and asked to return if symptoms persist or recur. Retreatment with the same recommended regimen is found to be an acceptable approach for treating recurrence or persistence of BV after first occurrence. For women with **multiple recurrences** after completion of a recommended and alternative regimen, various suppressive therapies have been tried; *0.75% metronidazole gel twice a week for 4–6 months* (reduces recurrences but the benefit might not persist when therapy is discontinued), **or** an oral nitroimidazole (metronidazole 400 mg or tinidazole 500 mg twice daily for 7 days) followed by intravaginal boric acid 600 mg daily for 21 days and then suppressive 0.75% metronidazole gel twice weekly for 4–6 months **or** monthly oral metronidazole 2 g administered with fluconazole 150 mg (reduces the incidence of BV and promotes colonization with normal vaginal flora). Intravaginal clindamycin can be used in women with allergy or intolerance to metronidazole or tinidazole.

A Cochrane review has suggested that there is currently insufficient evidence to recommend the use of probiotics either before, during or after antibiotic treatment as a means of reducing recurrence.[2]

Data suggest that douching changes vaginal flora and may predispose women to BV, although not all studies have reported this finding. Overall, the evidence suggests that douching should be discouraged as there is no proven health benefits.[3]

Trichomonas Vaginalis- In case of persistent infection which is not due to reinfection, women can be treated with *metronidazole 400 mg orally twice daily for 7 days*. If this regimen fails, oral metronidazole or tinidazole 2 g daily for 7 days can be prescribed. If several 1-week regimens have failed in a person who is unlikely to have nonadherence or reinfection, testing of the organism for metronidazole and tinidazole susceptibility is recommended. Higher dose of tinidazole at 2–3 g daily for 14 days, often in combination with intravaginal tinidazole, can be considered in cases of nitroimidazole-resistant infections; however, such cases should be managed under supervision of experts.

Candidiasis- Short-course topical formulations (i.e., single dose and regimens of 1–3 days) effectively treat uncomplicated VVC. The topically applied azole drugs are more effective than nystatin. During pregnancy, Single-dose treatment is less effective than longer regimens of up to 7 days.[3]

Recurrent Vulvovaginal Candidiasis (RVVC), usually

defined as four or more episodes of symptomatic VVC within 1 year, affects a small percentage of women (<5%). The pathogenesis of RVVC is poorly understood, and most women with RVVC have no apparent predisposing or underlying conditions. C. Glabrata and other nonalbicans Candida species are observed in 10%–20% of women with RVVC. Conventional antimycotic therapies are not as effective against these nonalbicans species as against C. albicans. Each individual episode of RVVC caused by C. Albicans responds well to short duration oral or topical azole therapy. However, to maintain clinical and mycologic control, a longer duration of initial therapy (7–14 days of topical therapy or oral fluconazole every third day for a total of 3 doses [day 1, 4, and 7]) is recommended for remission before initiating a maintenance regimen of weekly oral fluconazole for 6 months. If this regimen is not feasible, topical treatments used intermittently can also be considered. Suppressive maintenance therapies are effective in reducing RVVC. However 30%–50% of women will have recurrent disease after maintenance therapy is discontinued.

Severe vulvovaginitis (i.e., extensive vulvar erythema, edema, excoriation, and fissure formation) is associated with lower clinical response rates in patients treated with short courses of topical or oral therapy. Either 7–14 days of topical azole or 150 mg of fluconazole in two sequential oral doses (second dose 72 hours after initial dose) is recommended.

Non-albicans VVC- Because at least 50% of women with positive cultures for non-albicans Candida might be minimally symptomatic or have no symptoms and because successful treatment is often difficult, other causes of vaginal symptoms should be excluded in women with non-albicans yeast. The optimal treatment of non-albicans VVC remains unknown. Options include longer duration of therapy (7–14 days) with a non-fluconazole azole regimen (oral or topical) as first-line therapy. If recurrence occurs, 600 mg of boric acid in a gelatin capsule is recommended, administered vaginally once daily for 2 weeks. This regimen has clinical and mycologic eradication rates of approximately 70%, but is not yet available in India. For Candida Glabrata, Nystatin pessary 200 000 units for 14 days is also effective.[3]

Cervicitis- Women with persistent or recurrent cervicitis despite having been treated should be re-evaluated for possible re-exposure or treatment failure to gonorrhea or chlamydia. If relapse and/or reinfection with a specific STD have been excluded, BV is not present, and partner has been evaluated and treated, management options for persistent cervicitis are undefined; in addition, the utility of repeated or prolonged administration of antibiotic therapy for persistent symptomatic cervicitis remains unknown. The etiology of persistent cervicitis including the potential role of M. Genitalium is unclear. M. Genitalium might be considered for cases of clinically significant cervicitis that persist after azithromycin or doxycycline therapy in which re-exposure to an infected partner or medical nonadherence is unlikely. M. Genitalium should be suspected in cases of persistent or recurrent cervicitis and PID. Moxifloxacin (400 mg daily x 7, 10 or 14 days) has been successfully used to treat M. Genitalium in men and women with previous treatment failures.

Because cervicitis might be a sign of upper-genital–tract infection, women with a new episode of cervicitis should be assessed for signs of PID. Women with cervicitis also should be evaluated for the presence of BV and trichomoniasis, and if these are detected, they should be treated. ***A finding of >10 WBC per high power field in vaginal fluid, in the absence of trichomoniasis, indicates endocervical inflammation caused specifically by C. trachomatis or N. gonorrhoeae.*** Although HSV-2 infection has been associated with cervicitis, the utility of specific testing (i.e., PCR, culture or serologic testing) for HSV-2 is unknown. FDA-cleared diagnostic tests for M. Genitalium are not available.

To minimize transmission and reinfection, women treated for cervicitis should be instructed to abstain from sexual intercourse until they and their partner have been adequately treated (i.e., for 7 days after single-dose therapy or until completion of a 7-day regimen) and symptoms have resolved. Women diagnosed with cervicitis and Trichomoniasis should also be tested for HIV and syphilis.

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DRUG REVIEW

Dienogest in Endometriosis

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Introduction

Endometriosis is a chronic condition affecting women of child bearing age in which endometriotic lesions form outside the uterus leading to painful pelvic symptoms. Nearly half of those who are affected have chronic pelvic pain, dyspareunia and dysmenorrhoea. Endometriosis is estimated to occur in roughly 5 – 10 % of patients.

Role of Dienogest

This is a non-ethinylated progestin which is structurally related testosterone. Dienogest combines the advantages of 19-nor progestin and progesterone derivatives. This molecule is orally active, semi-synthetic, steroidal progestin. It has anti-androgenic property and hence can improve androgenic symptoms in endometriosis

Mechanism of action and its biological effects

Dieongest is completely absorbed and has high bio-availability after oral administration. This molecule binds with the progesterone receptors with high specificity and produces a potent progestogenic effect. Its binding affinity for estrogen and glucocorticoid receptor is very less. It has a half life of 10 hours and majority of this drug is excreted in urine in 24 hours. Its biological action includes inhibition of gonadotropin secretion leading to the reduction in the production of estradiol. This produces a hypo-estrogenic state with continuous use which leads to the decidualization of endometriotic tissue. This molecule also demonstrates anti-proliferative and anti-inflammatory action. Also it demonstrates anti-angiogenic effect.

Dose

This is given in a dose of 2 mg daily for 3-6 months. Ovarian activity is suppressed by this daily dose leading to a hypoestrogenic state. There is a rapid return of ovulation upon stopping the drug.This drug has predictable side effects as with other progestins like irregular menstruation, headache and bloating sensation.

Conclusion

The impact of endometriosis on woman's life is substantial. Use of this drug shows that improvement in both physical and mental symptoms occur in 12 to 24 weeks. Effective symptom control and predictable adverse effects significantly contributes to its high compliance. Pain relief in endometriosis is significant which may persist for 1-2 years upon stopping the drug.

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CLINICAL UPDATE

Fertility Preserving Surgery in Endometriosis

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Endometriosis affects young women, many of whom are yet to begin their obstetric career. It typically presents with symptoms of pain, infertility, or both. Surgery for endometriosis in these women should therefore be targeted not just to relieve the symptoms of pain, but also to preserve or enhance fertility. Laparoscopic approach should be preferred to laparotomy if appropriate equipment and expertise are available. Appropriate training for laparoscopic surgery is therefore vital to optimize surgical outcomes. The most commonly performed **conservative surgical procedures** include:

- Cystectomy or drainage and ablation for endometriotic cysts
- Fulguration of endometrial implants
- Adhesiolysis

Surgery for ovarian endometriomas

Ovary is the most commonly involved pelvic organ in endometriosis. Medical treatment is usually inadequate to treat endometriotic cysts. These are thick walled cysts which contain old blood. Surgical treatment includes either excision of the cyst wall (cystectomy) or drainage and ablation of the implants on the cyst wall. There is a continuing debate whether cystectomy or ablation should be performed in women desiring fertility. According to Cochrane review, cystectomy results in higher pregnancy rates, better pain relief, and lower chance of recurrence.[1] European Society of human reproduction and embryology guidelines (2014) recommend that excision should be performed for cysts more than 3 cm in women with infertility.[2] There are however concerns regarding reduction of ovarian reserve after cyst excision as some primordial follicles may be inadvertently stripped off the ovarian cortex during cystectomy. In a study evaluating the impact of cystectomy for endometriomas, serum AMH showed a significant decline one month after surgery. Additionally, the rate of decline in AMH after surgery was significantly greater in the subgroup with cysts more than 7 cm in size.[3]

Endometriosis is known to be a recurrent disease. Temporary suppression may be achieved with medical management although medication alone usually gives suboptimal control. Surgery is considered as the gold standard for the treatment for endometriosis, but at the cost of risk of operative morbidity and complications. Besides these risks, there is a high chance of recurrence. The estimated recurrence rate after primary surgery is reported as 21.5% at 2 years and 40–50% at 5 years.[4]

The therapeutic advantage from the first surgery is significantly greater than that from the subsequent ones. Surgery for recurrent endometriotic cysts should be therefore considered very carefully because the deleterious effect of repeat surgery on the ovarian reserve can be very pronounced. The cyst wall specimens after repeat cystectomies was found to be significantly thicker, and the operated ovary had a significantly lower AFC and volume.[5] Surgery should only be undertaken if pain is a significant component and not relieved by medical treatment. In case of infertility with recurrent endometriomas, in-vitro fertilization should be advised without prior surgery, if cysts are small and will not interfere with ovum pickup.

Every effort should be made to minimize ovarian damage due to surgery. Identify clear cleavage when performing cystectomy. It can also be aided by hydro-dissection. The cyst wall can usually be differentiated clearly from the ovarian cortex in large cysts. However, it may be difficult to do so in smaller cysts; drainage and ablation may be performed in such cases. The use of diathermy should be restricted to the minimum essential to achieve hemostasis and fulgurate any obvious implants. Generalized fulguration of the cyst wall should not be undertaken.

Surgery for endometrial implants

Peritoneal implants can be treated surgically by fulguration, laser vaporization, or excision. Laparoscopic ablation of endometriotic implants in stage I/II disease was found to have a small but significant increase in pregnancy rates.[6] Conservative surgery in moderate to severe endometriosis has also shown to increase pregnancy rates compared to expectant management, although there are no randomized studies.[7]

There is no clear recommendation based on available data, whether there is any difference in fertility outcomes if the implants are ablated or excised. The advantage of excision is both therapeutic and diagnostic as it provides tissue for histopathology. Ablation is quicker, easier, and technically less demanding, but may not be necessarily

safer due to the potential of injury from the thermal spread.

Though the best surgical approach for deep seated implants is not clear, general consensus seems to be in favour of excision.[7] This needs to be balanced with the increased risk of operative complications like injury to rectum and ureter.

Adhesiolysis

Adhesions and scarring are integral to the physical characteristics of endometriosis. Once the endometrial glands which are shed into the pelvis via the tubes implant on the pelvic peritoneum, they behave like the ectopic endometrium and bleed when the normal endometrium bleeds. This results in inflammation, scarring and adhesions with the adjacent organs, leading to distortion of pelvic anatomy and infertility.

Adhesiolysis is necessary to release the pelvic organs, such as the ovaries adherent to the uterus and/or pouch of Douglas, and the tubes which may be buried in adhesions. Bowel loops and omentum are often adherent to the uterus and adnexa, and need to be released with great caution to restore pelvic anatomy. At times, it may be wise not to be too aggressive in doing adhesiolysis if the risk of complications is deemed to be greater than the benefit achieved by the procedure.

In conclusion, surgery for the treatment of endometriosis in young women must be performed by an experienced operator with intent for maximal fertility preservation. Adequately performed primary surgery not only gives the best results in terms of pregnancy rates but also reduces the chances of recurrence.

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A Heartfelt Wish

A woman's life is inextricably linked
to her menstrual cycle, the days are inked.
Fever, headache, a fall she will tolerate
but hypo, oligo, menometrorrhagia she will hate.

The ovaries are her lifeline,
Their estrogen makes her skin shine.
But hark when they fail!
PCOS, Infertility, menopause will trail.

If only there was a way
to predict the disorder before it made hay.
If only the ovaries would function on demand
How much better if only the menstruation was under her command!

-Dr Sumitra Bachani,
Specialist, VMMC & Safdarjung Hospital

Journal scan

Sunita Malik[1], Sheeba Marwah[2]
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Treatment of Provoked Vulvodynia in a Swedish Cohort using Desensitization Exercises and Cognitive Behavioral Therapy.

Lindström S, Kvist LJ

BMC Womens Health. 2015 Nov 25;15:108.

Background: Problems related to pain during vaginal penetration are complex and the etiology is multi-factorial. It was the aim of the present study to measure whether treatment using desensitization exercises and cognitive behavioral therapy (CBT) for women with provoked vulvodynia (PVD) could increase sexual interest, sexual satisfaction and response whilst decreasing experiences of sexual pain.

Methods and Outcome Measures: Sixty women suffering from PVD were treated during a 10-week period with a combination of mucosal desensitization and pelvic floor exercises and CBT. The McCoy Female Sexuality Questionnaire (MFSQ) was used to measure efficacy of the treatment. The Hospital Anxiety and Depression Scale (HADS) was used to measure psychological distress. The primary outcome measurements were changes in scores for the MFSQ and changes in individual items on the MFSQ directly after treatment completion. Secondary outcome measurements were changes in the MFSQ items 6 months after treatment and changes in HADS sub-scales 6 months after treatment. Statistical comparisons of answers to the MFSQ were carried out using the Wilcoxon signed rank test (paired). Validity of the MFSQ in this study was measured by testing one global question about sexuality and total scores on MFSQ using Spearman's correlation test.

Results: Study participants reported a statistically significant increase in sexual fantasies, increased sexual pleasure, excitement and vaginal lubrication after treatment was completed. PVD occurred less often which resulted in significantly less avoidance of sexual intercourse, increased frequency of masturbation and intercourse. All improvements were sustained at 6 months after treatment ended. Two questions showed no significant changes, these pertained to the individual's contentment with her partner as a lover and a friend. The anxiety sub-scale of the HADS showed a significantly decreased level of anxiety at 6 months follow-up but no change in the scores on the depression sub-scale.

Conclusion: Treatment for PVD using desensitization exercises and cognitive behavioral therapy significantly improved sexual interest, response and activity and decreased the experience of pain. Larger studies and RCTs are required in order to draw conclusions about treatment and long term effects should be studied. Partners should be encouraged to participate in treatment regimes.

Factors Associated with Bacterial Vaginosis among Women Who Have Sex with Women: A Systematic Review

Forcey DS, Vodstrcil LA, Hocking JS, Fairley CK, Law M, McNair RP, Bradshaw CS

PLoS One. 2015 Dec 16; 10(12):e0141905.

Background: Women who have sex with women (WSW) have a higher burden of bacterial vaginosis (BV) than heterosexual women; studies of risk factors specific to this population are limited. We summarised current knowledge regarding risk factors for BV among WSW by systematic review.

Methods: This systematic review was conducted according to the PRISMA statement. PUBMED, EMBASE, Web of Science and The Cochrane Library were searched to 31st December, 2014.Inclusion Criteria:1) WSW included in the study population; 2) accepted BV diagnostic method; 3) investigated or could extrapolate factors(s) associated with BV acquisition, persistence or transmission in WSW specifically by comparing BV positive to BV negative women. Search was limited to English-language publications.

Results: A limited number of studies have investigated BV in WSW. Of 71 unique references, 18 full-text articles were assessed and 14 studies fulfilled inclusion criteria. BV was positively associated with higher numbers of female partners, both lifetime and in the three months prior to diagnosis, and confirmed BV in a female partner, but inconsistently associated with partners' BV history or symptoms. BV was not associated with ethnicity, vaginal douching or hormonal contraception. The impact of specific sexual activities, male sexual contact, smoking and the menstrual cycle varied considerably between study populations.

Conclusion: BV in WSW is associated with increased

numbers of recent and past female partners and confirmed BV in a female partner. There are limited studies of BV in WSW populations, and research is needed to further elucidate risk factors for BV among WSW. However these data provide epidemiological evidence that BV risk in women is directly related to exposure to other female partners and a partner with BV, providing support for the concept that BV is likely to be transmitted between women.

Norethindrone Acetate or Dienogest for the Treatment of Symptomatic Endometriosis: A before and after Study

Vercellini P, Bracco B, Mosconi P, Roberto A, Alberico D, Dhouha D, Somigliana E.

Fertil Steril. 2015 Dec 8.pii: S0015-0282(15)02092-0. doi: 10.1016/j.fertnstert.2015.11.016. [Epub ahead of print]

Objective: To assess the proportion of patients satisfied with their treatment before and after a systematic change from norethindrone acetate to dienogest as the first-line progestin for symptomatic endometriosis.

Methods: Design: Before and after study Setting: Academic department. Patient(s): The last 90 new consecutive endometriosis patients in whom norethindrone acetate was used, and the first 90 new consecutiveendometriosis patients in whom dienogest was used. Intervention(s): Norethindrone acetate at the oral dose of 2.5 mg once a day until June 6, 2013, then dienogest at the oral dose of 2 mg once a day thereafter. Main Outcome Measure(s): Degree of satisfaction with treatment after 6 months of progestin therapy and assessment of any variations in pain symptoms, psychological status, sexual function, or health-related quality of life associated with the introduction of dienogest.

Results: The proportion of satisfied plus very satisfied women after 6 months of treatment was 71% in the "before" period (norethindrone acetate) and 72% in the "after" period (dienogest). The implementation of dienogest was not associated with statistically significant ameliorations in overall pain relief, psychological status, sexual functioning, or health-related quality of life. Treatment was well tolerated by 58% of norethindrone acetate users compared with 80% of dienogest users. After dienogest implementation, the absolute risk reduction in the occurrence of any side effect was 13.9% (95% confidence interval, 0.8%-28.6%).

Conclusions: Considering the large difference in the cost of the two drugs, dienogest should be suggested selectively in women who do not tolerate norethindrone acetate

Proceedings of AOGD Monthly Meeting held on 18th December, 2015 at Hindu Rao Hospital

Compiled by Dr Archana Mishra
Assistant Professor, Obstetrics & Gynaecology, VMMC & Safdarjung Hospital, New Delhi

Case 1

Bilateral Dysgerminoma with Term Live Pregnancy with Pre-Eclampsia

Supriya Kumari, Vandana Saini, Mamta Gupta, Rita Jindal

A 28 year old woman presented to us at near term pregnancy, complicated with preeclampsia (BP 160/104, urine albumin-1+). A firm lump of ~15x8cm was felt in continuity to the uterus in left hypochondrium which was diagnosed as subserous fibroid on ultrasound. All her ANC investigations were normal. As suspicion was of subserosal fibroid, workup was not of the same order as would be ordered if the suspicion were of a malignant ovarian tumor, such as testing for tumor markers, pelvic magnetic resonance imaging. Ultrasound done pre-pregnancy was normal. During cesarean, omentum appeared normal, bilateral tubo-ovarian masses were found and removed which later on, turned out to be dysgerminoma on histopathological examination. Patient was called back for further workup by PET scan and tumor markers. As she was unstaged, she was sent for chemotherapy (BEP).

Spontaneous conception in patients with dysgerminoma is quite rare and having a live birth is even rarer. Most dysgerminomas with pregnancy are asymptomatic and diagnosed on clinical examination or picked up on sonography. Frequency of ovarian tumor associated with pregnancy ranges from 1:80 to 1:2200 deliveries. Dysgerminomas account for 1-5% of all ovarian malignancies mostly occurring in reproductive age group, causing problem in conception, if pregnancy occurs, leads to feto-maternal compromise and preservation of fertility is also a challenge.

Case 2
Autoimmune Hepatitis in Pregnancy- A dilemma

Arvind, Mala Shukla

A 28 yr old $G_3P_2L_1A_0$ with 32 week High-Risk Pregnancy (previous 2 LSCS with one perinatal death) presented with sudden onset projectile vomiting with H/o similar episodes of sudden vomiting & epigastric pain in her previous 2 pregnancies in the 3rd trimester. Her clinical evaluation revealed a massive hepato-splenomegaly. Her Ultra-Sound review showed gross dilatation of portal, intra-hepatic, splenic & mesentric veins, hepatomegaly of size 22cm and splenomegaly. Fetal parameters and fetal doppler flow studies were normal. Patient was +ve for ANA in a titre of 1:80 but tests for SLE B-2 glycoprotein /LA/ & ACA were negative. Mild elevation of ALT (165 IU/L) & AST (116 IU/L) were seen. Patients vomiting subsided on conservative supportive treatment, & she continued till 36^{+5} weeks, when she went into spontaneous labour, underwent LSCS on 2/11/15 & delivered a healthy male child of 2.5 kg, intraoperatively abdomen was plastered with dense adhesions. In family history patient's younger sister also had moderate hepatomegaly on ultrasound but she refused further testing.

On multidisciplinary review this was a case of Auto Immune Hepatitis (ANA +ve in 1:80 titre) with negative SLE parameters which has a genetic, metabolic component. It is thought to be part of PE spectrum of disorders with predilection for AFL of pregnancy & HELLP syndrome. It is a possible marker of long chain fatty acid disorder, with AR inheritance, if mother is heterozygous & baby homozygous the incidence of AFLP is 31% -70% .

Case 3
OHVIRA Syndrome with Rare Presentation- A Case Report

Ritu Sharma, Ojasvi Shanker, Sudha Salhan

26 year old female, married for 1 year presented with continuous foul smelling vaginal discharge. She had menarche at 14 years and was having normal menstrual function with normal sexual activity. On per speculum examination a bulge was noted anterolaterally on right side of vagina and copious foul smelling discharge was seen coming out from cervix. On per vaginal examination, vagina was found to be roomy, the same bulge as mentioned above could be palpated as soft, fluctuant and non tender longitudinal swelling extending up to the fornix. Uterus was anteverted, bulky - broad at fundus with depression at the middle, firm, non tender and mobile. Bilateral fornices were non tender. Ultrasound examination showed bicornuate uterus. Keeping Mullerian abnormality in mind, MRI was done which revealed two well defined uterine cavities and two hemivagina with distal obstruction in right hemivagina along with fluid collection. Also right sided kidney was absent with compensatory hypertrophy on left side. So the diagnosis of Obstructed Hemivagina with Ipsilateral Renal Anomaly (OHVIRA) was made and the patient was planned for vaginal septal resection. We presented this case in view of its rare presentation- late onset chronic unresponsive vaginal discharge.

Brain Teasers

Dr Monika Gupta
Assistant Professor
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We have a **lucky dip** for all the right answers received and winner's name will be announced in the next monthly AOGD clinical meeting. So, mail your answers to **aogdsjh2015@gmail.com within 7 days of receipt of the bulletin**.

1. All of the following about Dienogest are true except:
   a. Potent progestogenic effect
   b. Improves androgenic symptoms in endometriosis
   c. High affinity for glucocorticoid receptors
   d. Anti-angiogenic and anti-inflammatory effect

2. As per new FIGO Nomenclature for Optimal Diagnostic Evaluation of Abnormal Uterine Bleeding, first line investigation for AUB- PALM should be
   a. Pelvic ultrasound
   b. MRI
   c. Saline infusion sonography
   d. TVS

3. Which of the following strength of Depot Medroxy-progesterone acetate is USFDA approved for management of endometriosis
   a. 100 mg
   b. 104 mg
   c. 154 mg
   d. 184 mg

4. Which of the following is not a drug used in medical management of uterine leiomyoma
   a. Mifepristone
   b. Dienogest
   c. Ulipristal Acetate
   d. Gestrinone

5. Which of the following is not a component for screening of Cardiovascular disease risk factors in PCOS patients.
   a. Waist circumference
   b. Polysomnography
   c. Blood pressure
   d. Thyroid function testing

6. CA-125 levels which are suggestive of rupture of endometrioma are
   a. >35 IU/ML
   b. >55 IU/ML
   c. >75 IU/ML
   d. >100 IU/ML

7. According to the International Ovarian Tumor Analysis (IOTA) Groupultrasound rules, which of the following is categorised under 'M' rules
   a. Presence of acoustic shadowing
   b. Ascites
   c. Unilocular cyst
   d. Absent blood flow

8. In theVariants of Premenstrual Disorders which have complex features, all of the following categories are included except
   a. Estrogen-induced Premenstrual syndrome
   b. PMS with anovulatory ovarian activity
   c. Premenstrual exacerbation of an underlying condition
   d. PMS in the absence of menstruation

9. All of the following are the specific criteria for diagnosing pelvic inflammatory disease except
   a. Histopathological evidence of endometritis
   b. Laboratory documentation of cervical infection
   c. Tuboovarian complex on TVS
   d. Laparoscopic findings consistent with PID

10. Which of the following is true regrarding platelets collected from whole blood donations
    a. 1 unit of platelet concentrate/10kg body weight
    b. Platelets must be frozen
    c. ABO typing is essential
    d. Shelf life is 15 days at 20-24^{o}C

Answers to Quiz 7: 1. c; 2. b; 3. c; 4. c; 5. b; 6. a; 7. b; 8. a; 9. c; 10. b

The Winner of Quiz 7: Dr Aastha Aggarwal, Dr BSA Hospital, Rohini. Congratulation!

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  Contact: Dr. Prathima Radhakrishnan, Phone: +91 9035173865/ 9742875424 email: prathimabfmc@hotmail.com
- **27th September, 2015**: Inaugural CME of the Patiala SFM Chapter, Patiala, Punjab.
  Contact: Dr. Chander Mohini. Phone: +91 9814087891, email: chandermohini15@gmail.com
- **25th October, 2015:** Fetal Day CME, Jabalpur, Madhya Pradesh.
  Contact: Dr. D'Pankar Banerji. Phone: +91 9826166952, email: dpankar@idealfertility.com
- **31st October- 1st November, 2015:** Rainbow Fetus Day and Society of Fetal Medicine Mid-term CME, Hyderabad, Andhra Pradesh.
  Contact: Dr. Chinmayee Ratha. Phone: +91 9885348600, email: chinmayee3@gmail.com
- **12-13th December, 2015:** Fetal Heart 2, Amrita Institute of Medical Sciences, Kochi. Kerala covering Fetal Heart Imaging from 2D to 4D STIC with a STIC workshop and Therapeutics Focussing on Comprehensive Care of a Fetus with Heart Disease
  Contact: Dr Balu Vaidyanathan. Phone: +91 9495820684, email: baluvaidyanathan@gmail.com

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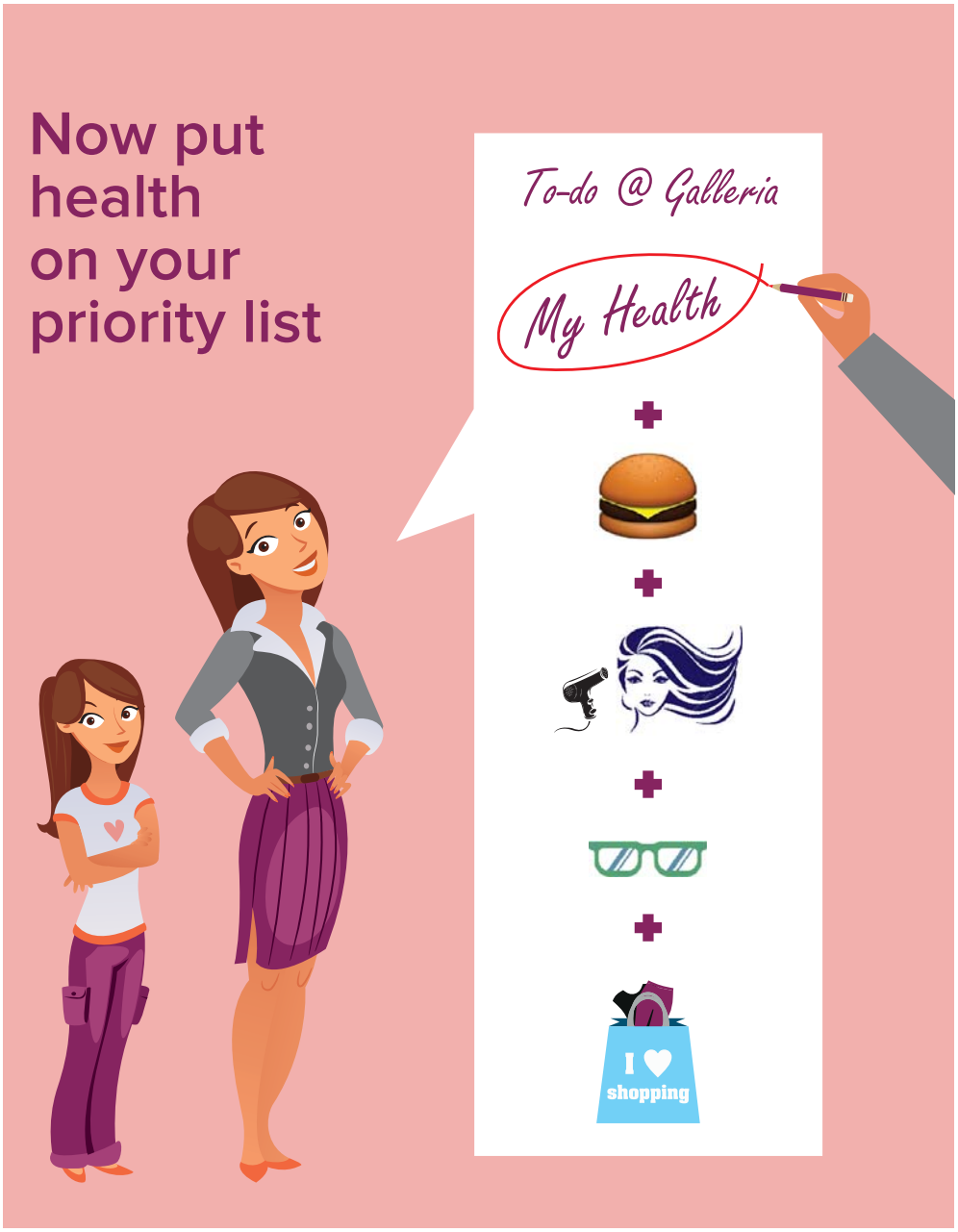

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