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Enlightening the Path for Next Generation of Gynaecologists

Dedicated Issue:
Benign Gynaecology

AOGD SECRETARIAT

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From the President's Pen

It gives me immense pleasure to write foreword for September Issue of AOGD Bulletin dedicated to day to day conditions seen in clinical practice. Although all of us manage these conditions in best possible manner yet time to time review helps us keeping updated. Towards end of month we shall assemble for. Annual Conference of. AOGD . I hope programme will be liked by all

Dr Sunesh Kumar
President, AOGD

From the Secretary's Desk

Dear friends

The AOGD Secretariat at AIIMS has been working hard to publish informative bulletins which have been highly appreciated by one and all. Thank you all for the support. The current issue is important because it covers the conditions we deal with in day to day practice.

The month of August was busy with many academic activities organized under the aegis of AOGD. A National conference, Breaking Silos Across: Adolescent to Menopause" was organized by Dr Sudha Prasad. Masterclass in Gynecologic Oncology was organized by UCMS & GTB Hospital under aegis of AGOI, FOGSI & AOGD Oncology committee and AOGIN. QI Workshop in LHMC under Quality Improvement Sub Committee of AOGD, CME on Infertility by RML Hospital under aegis of Infertility and Reproductive Endocrinology Committee and Hyteroscopy live workshop under Endoscopy Sub Committee of AOGD were other academic highlights of the month. The monthly meeting was held at Army Hospital R&R.

The academic bonanza, 41st Annual Conference, is around the corner. We have worked hard to make an exciting and useful programme, which will definitely enlighten the path of young gynecologists. Besides the main conference, there are nine workshops. We have received around 150 abstracts. My humble request to join in large numbers to make the conference a grand success. A Safe Abortion Walk is organized in the morning of 29th Sept.

Looking forward to welcome one and all at the 41st Annual Conference of AOGD on 28-29th Sept, 2019 at Hotel Eros, Nehru Place.

Dr Vatsla Dadhwal
Hon. Secretary

Monthly Clinical Meeting

Monthly Clinical Meet will be held at ESI Hospital, New Delhi
on **Friday, 25th October, 2019 from 04:00pm to 05:00pm**.

From the Editor's Desk

Dr J B Sharma
Editor

Dr Reeta Mahey

Dr P Vanamail

Dr Vidushi Kulshreshtha

Co-Editors

We are pleased to write the Editor's desk for this issue of AOGD Bulletin on Benign Gynaecology with the satisfaction that previous issues have been liked by the esteemed Colleagues especially the last Bulletin on Fetal Medicine by Dr Vatsla Dadhwal and Dr Aparna Sharma has been really appreciated. I congratulate and thank them and the authors for working hard.

In this issue we have an interesting and clinically useful article on day to day problem of Vaginal Discharge by Dr Nilofar and Dr Rajesh Kumari which highlights various types of vaginitis and its syndromic treatment.

We have two articles on very common clinical gynae problem of Abnormal Uterine Bleeding (AUB); Updated PALM-COEIN classification of FIGO by Dr Vidushi and Dr Monica from AIIMS and another article on Evaluation and Management of AUB by Dr Kanika and Dr Swati from LHMC for the benefit of our esteemed readers. Dr Taruna and Dr Bindiya enlighten us about another difficult gynae problem of adenomyosis including its management. Fibroid Uterus is a common problem and important indication of hysterectomy though it can easily be managed medically in most cases as highlighted by Dr Aditi and Dr Anupama from AIIMS Rishikesh.

Endometriosis remains an enigmatic disease causing lot of misery to the affected person but can be medically treated in most cases necessitating surgery (laparoscopy or laparotomy) in selected cases as highlighted by Dr Manu and Dr Pratibha from AIIMS Jodhpur.

Dr Radhika from UCMS enlightens us on Evidence Based Practice in Obstetrics and Gynecology which is the need of the hour. We have an interesting flowchart on management of Pelvic Inflammatory Disease (PID) by Dr Anukriti and Dr. Jyoti.

Besides, we have Journal scan, Clinical Proceedings of last meeting and Maze of knowledge for the benefit of our esteemed readers.

We wish our esteemed readers a happy reading and solicit their suggestions to further improve the Bulletin.

Editor
Dr J B Sharma

Vaginal Discharge

Nilofar Noor[1], Rajesh Kumari[2]
[1]Senior Resident, [2]Assistant Professor, Department of Obstetrics and Gynaecology, All India Institute of medical Sciences, New Delhi

Overview

The most common cause of vaginal discharge in women of reproductive age is normal physiological discharge. Normal physiological discharge changes with the menstrual cycle. It is thick and sticky for most of the cycle, but becomes clearer and stretchy around the time of ovulation.

Abnormal vaginal discharge is characterized by a change of colour, consistency, volume, or odour, and may be associated with symptoms such as itch, soreness, dysuria, pelvic pain, or intermenstrual or post-coital bleeding.

Abnormal vaginal discharge is most commonly caused by vaginal and cervical infections. Other etiologies include STIs, PID and occasionally non-infective causes.

Infective causes	Non- infective causes
Vulvo-vaginal candidiasis	Retained foreign body- tampoms/ condoms/ forgotten pessary
Bacterial vaginosis	Tumours of the vulva, vagina, cervix, and endometrium
Trichomonas vaginalis	Atrophic vaginitis/ cervicitis in post-menopausal women
Chlamydia trachomatis	Cervical ectopy or polyps
Neisseria gonorrhoea	
Other STIs	
PID	

A careful history, examination (including inspection of external genitalia, speculum examination of cervix and vagina, and bimanual palpation), and laboratory testing should be done to determine the etiology of vaginal symptoms. High risk behaviours such as sexual practices, gender of sex partners, menses, vaginal hygiene practices (e.g., douching), and self-treatment with medications should be elicited.

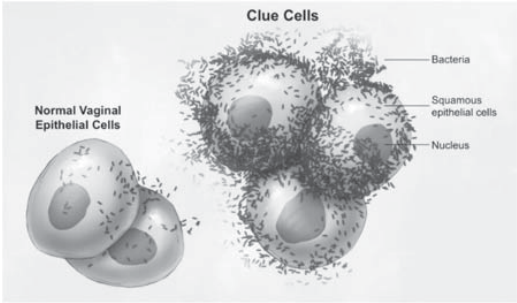
The three diseases most frequently associated with vaginal discharge are Bacterial vaginosis (BV), Vulvovaginal candidiasis (VVC) *and Trichomonas vaginalis (TV)*.

Bacterial vaginosis (BV)

BV is a polymicrobial clinical syndrome resulting from replacement of the normal vaginal flora comprising of Lactobacillus, with high concentrations of anaerobic bacteria (e.g., Prevotella sp. and Mobiluncus sp.), G. vaginalis, Ureaplasma, Mycoplasma, and numerous fastidious or uncultivated anaerobes.

Diagnosis

1. Amsel's Diagnostic Criteria- This criterion requires three of the following symptoms or signs:
   - homogeneous, thin, white discharge that smoothly coats the vaginal walls
   - clue cells (e.g., vaginal epithelial cells studded with adherent coccobacilli) on microscopic examination



   - pH of vaginal fluid >4.5
   - fishy odour of vaginal discharge after addition of 10% KOH (i.e., the whiff test)
2. Gram stain- gold standard laboratory method for diagnosing BV

Vulvo-vaginal candidiasis (VVC)

VVC can be classified into uncomplicated or complicated on the basis of clinical presentation, microbiology, host factors, and response to therapy.

Classification of vulvovaginal candidiasis	
Uncomplicated VVC	• Sporadic or infrequent • Mild-to-moderate disease • Likely to be Candida albicans • Non-immunocompromised women
Complicated VVC	1. **Recurrent VVC**- 4 episodes of symptomatic VVC in 1 year 2. **Severe VVC**- extensive vulvar erythema, edema, excoriation and fissure formation • Nonalbicans candidiasis • Women with diabetes, immunocompromising conditions (e.g., HIV infection), debilitation, or immunosuppressive therapy (e.g., corticosteroids)

Trichomonas vaginalis (TV)

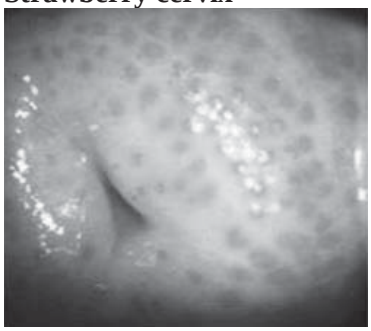
TV is a sexually transmitted disease and these women should be screened for other STIs as well as their partners should be treated.

Clinical features

Clinical features of the three commonest infections are summarized below.

Feature	Bacterial vaginosis	Vulvovaginal candidiasis	Trichomoniasis
Symptoms	Thin discharge	Thick white, curdy discharge	Scanty to profuse or frothy yellow discharge
	Offensive or fishy odour	Non-offensive odour	Offensive odour
	No discomfort or itch	Vulval itch Superficial dyspareunia Dysuria	Vulval itch or soreness Dysuria (external) Low abdominal pain Dyspareunia
Signs	Discharge coating vagina and vestibule	Vulval erythema, oedema, fissuring	Vulvitis and vaginitis
	No inflammation of vulva	Satellite lesions	"Strawberry" cervix
pH of vaginal fluid	Vaginal pH > 4.5	Vaginal pH < 4.5	Vaginal pH > 4.5
Microscopy	"Clue" cells	Yeasts and pseudohyphae	

Strawberry cervix



Complications

- Bacterial vaginosis is associated with increased risk of spontaneous miscarriage and preterm birth, post-abortion endometritis, post-hysterectomy vaginal cuff infection, increased risk of acquiring STI, especially genital herpes and HIV. Symptomatic pregnant women should therefore be treated.
- Symptomatic VVC is more frequent in women with HIV infection and correlates with severity of immunodeficiency and may require more prolonged treatment.
- Vaginal trichomoniasis has been associated with adverse pregnancy outcomes, particularly premature rupture of membranes, preterm delivery, and low birth weight. It is also a risk factor for vertical transmission of HIV. Hence treatment is recommended in pregnant women. There is also evidence that trichomoniasis may enhance HIV transmission in the population. *T. vaginalis* co-infection with HIV is significantly associated with PID, and treatment of trichomoniasis is associated with significant decreases in genital-tract HIV viral load and viral shedding. Hence, routine screening and prompt treatment are recommended for all women with HIV infection.

Investigations

Vaginal swab can be sent for testing. In patients with cervical discharge or evidence of cervicitis, endocervical swab can be sent to test for chlamydia and gonorrhoea.

Clinical indicators for testing vaginal discharge:

- High risk for STI
- High risk behaviour
- Failed previous treatment
- Post abortion, post-partum, and pregnant women
- Recent intrauterine device (IUD) insertion
- Signs or symptoms suggestive of pelvic inflammatory disease (PID)
- Diagnosis uncertain
- Woman's request

Perform cervical screening if overdue, abnormal bleeding, or suspicious findings on examination.

Diagnostic tests for vaginal and cervical infections

Infection	Site/Specimen	Test
Bacterial vaginosis	High vaginal swab	Microscopy and gram stain Whiff test (release of fishy odour on adding alkali (10%KOH) pH test (pH> 4.5 indicative of bacterial vaginosis)
Trichomoniasis	High vaginal swab	NAAT
Candidiasis	High vaginal swab	Microscopy, gram stain and culture
Chlamydia	Endocervical swab	NAAT
Gonorrhoea	Endocervical swab	NAAT

NAAT – Nucleic Acid Amplification Test

Treatment

Drug treatment for each type of infection is summarized in the box below.

- Syndromic approach can be applied and treatment offered based on symptoms and signs
- Where vaginal/ endocervical swabs have been sent, treatment can later be modified based on the results
- Women should be counselled to avoid alcohol if taking metronidazole as it can result in disulfiram-like reaction.

- Intravaginal azoles and clindamycin can damage latex condoms and hence additional contraception should be used.
- Contact tracing and treatment for trichomoniasis, chlamydia and gonorrhoea should be performed in all patients with confirmed infection. Education regarding their transmission and use of condoms encouraged.

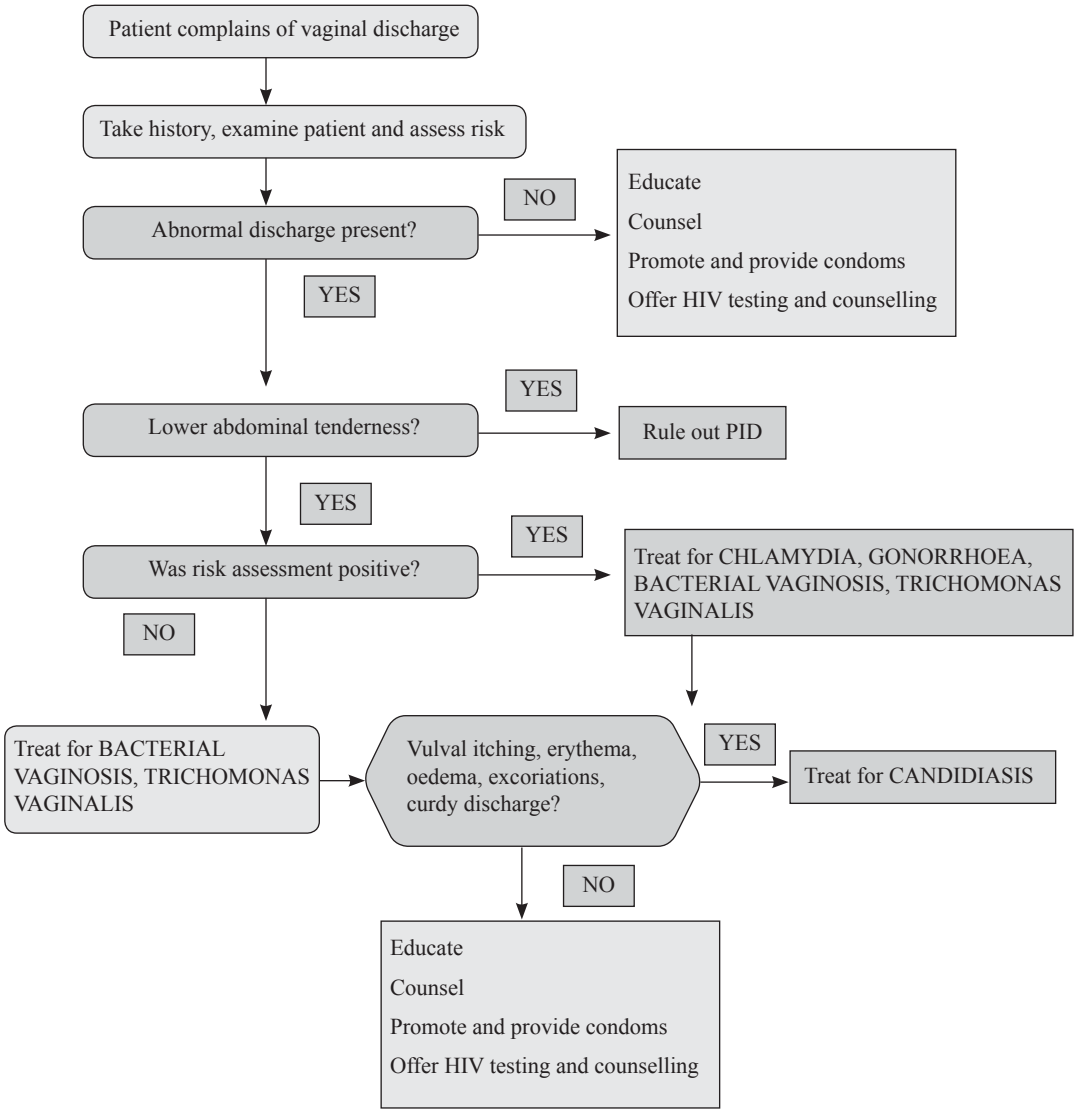
Drug treatment for the three commonest vaginal infections[1]

Vaginal infection	Treatment regimes	Alternate regimes	Management of partner	HIV co-infection	Pregnancy and lactation	Follow up
Bacterial vaginosis	Metronidazole 500mg BD PO for 7 days OR Metronidazole gel 0.75% 5g intravaginal OD for 5 days OR Clindamycin cream 2% 5g intravaginal for 7 days	Tinidazole 2 g OD PO for 2 days OR Tinidazole 1g OD PO for 5 days OR Clindamycin 300mg BD PO for 7 days OR Clindamycin ovules 100mg OD for 3 days	Routine treatment not recommended	Same regimes	Metronidazole 250 mg orally three times a day for 7 days OR Clindamycin 300mg orally twice a day for 7 days	Not needed routinely
Candidiasis	**VVC** Clotrimazole 1% cream 5 gm intravaginally for 7-14 days **Severe VVC** 7–14 days of topical azole OR 150 mg of oral Fluconazole 2 doses (72 hrs apart) **Recurrent VVC** 7–14 days of topical therapy OR oral Fluconazole (100,150 or 200mg) every third day for a total of 3 doses followed by weekly for 6 months	**VVC** Intavaginal azole creams- Miconazole Ticonazole Butoconazole Terconazole OR Fluconazole 150mg PO single dose	Not an STI Routine treatment not recommended	More prolonged treatment may be needed	Topical azoles for 7 days	Not needed routinely
Trichomonas vaginalis	Metronidazole 2g PO single dose OR Tinidazole 2g PO single dose	Metronidazole 500mg BD PO for 7 days	STI Treatment given Abstinence/ barrier contraceptives till cure	Metronidazole 500 mg PO BD for 7 days	Metronidazole 2g PO single dose	Test of cure after 3 months

Drug treatment in case of cervicitis should include combined treatment of chlamydia and gonorrhoea[1]

Non pregnant women		
Treatment of chlamydia	PLUS	**Treatment of gonorrhoea**
Azithromycin 1 g PO single dose OR Doxycycline 100 mg PO BD for 7 days		Ceftriaxone 250 mg IM single dose OR Cefixime 400 mg PO single dose
Pregnant women		
Azithromycin 1g PO single dose OR Erythromycin 1g PO BD or 500 mg QID for 7 days	PLUS	Ceftriaxone 250mg IM single dose OR Cefixime 400mg PO single dose

Syndromic management on outpatient basis4



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Abnormal Uterine Bleeding: Updated PALM COEIN Classification

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Introduction

The International Federation of Gynecology and Obstetrics (FIGO) in 2011 published a pair of systems and a set of clinical recommendations to guide clinicians in evidence based management of abnormal uterine bleeding (AUB). This publication included two systems- Terminology and Definitions (FIGO-AUB System 1) and the PALM-COEIN system of classification of causes of AUB in reproductive years (FIGO-AUB System 2). This classification system proposed by FIGO Menstrual Disorders Group (MDG) is an attempt to standardize the terminology, investigation, diagnosis and management of AUB in non-gravid women of reproductive age.[1]

The PALM-COEIN system was the first systematic classification system of underlying causes of AUB based on clinical and imaging based division of causes into structural pathologies – the PALM group (**P**olyp, **A**denomyosis, **L**eiomyoma, **M**alignancy and **H**yperplasia). This group of pathologies can be imaged and/or defined by histopathology. The other group denoted as COEIN (**C**oagulopathy, **O**vulatory dysfunction, **E**ndometrial, **I**atrogenic, and **N**ot yet classified) are non-structural causes as they cannot be identified by imaging but need a detailed clinical history, physical examination and sometimes supporting laboratory investigations to diagnose them.[1] In 2018 FIGO has updated the recommendations for both the systems and slightly modified the PALM COEIN system.[2]

Apart from the PALM-COEIN system, FIGO has also defined nongestational acute and chronic AUB and redefined the normal limits of menstrual parameters. Acute AUB is defined as an episode of heavy bleeding that is of sufficient quantity to require immediate intervention to prevent further blood loss. Chronic AUB is defined as bleeding from the uterine corpus that is abnormal in duration, regularity, volume or frequency in absence of pregnancy and has been present for most of the previous six months. The evaluation of acute AUB is similar to evaluation of chronic AUB after assessment of hemodynamic status and assuring stability of the patient.[1]

Table 1: FIGO-AUB System 1[2]

Parameter	Normal	Abnormal
Frequency	Absent (no bleeding) = amenorrhoea	
	Infrequent(>38 days)	
	Normal (≥24 to ≤38 days)	
	Frequent (<24 days)	
Duration	Normal (≤8 days)	
	Prolonged (>8 days)	
Regularity	Normal or "Regular" (shortest to longest cycle variation: ≤7-9 days)	
	Irregular (shortest to longest cycle variation: ≥8-10 days)	
Flow volume	Light	
	Normal	
	Heavy	
Intermenstrual Bleeding (IMB) (Bleeding between cyclically regular onset of menses)	None	
	Random	
	Cyclic (Predictable)	Early cycle
		Mid cycle
		Late cycle
Unscheduled Bleeding on Progestins ± Estrogen gonadal steroids (COCs, rings, patches, injections)	Not applicable (not on gonadal steroids)	
	None (on gonadal steroids)	
	Present	

Table 1 shows the updated FIGO AUB system 1.[2] Amenorrhoea has been included in the frequency category. In fluid volume definition of symptom of heavy menstrual bleeding as proposed by NICE has been adopted that is excessive menstrual loss which interferes with woman's physical, social, emotional and material quality of life.[3] Terms such as menorrhagia, metrorrhagia, oligomenorrhea, and dysfunctional uterine bleeding have been abandoned. Intermenstrual bleeding has been added in the revised publication. The definition of regularity has been modified from shortest to longest variation of up to 20 days to 7–9 days, excludes the occasional long or short periods and varies with age age: 18–25 y of age, ≤9 d; 26–41 y, ≤7 d; and for 42–45 y, ≤9 d. Any deviation from these normal parameters is AUB. However, a patient should be labelled 'AUB' only after ruling out pregnancy by history and urine/serum β human chorionic gonadotropin.

FIGO System 2 – PALM COEIN

In the revised 2018 publication the basic core classification of nine categories PALM COEIN remains the same. The summary of changes have been presented in Table 2.[2] The FIGO MDC is working on subclassification systems for polyp and adenomyosis which will be published later.

Table 2: Changes in the PALM COEIN system[2]

System 2	Changes
AUB-A	Refined sonographic diagnostic criteria
AUB-L	Inclusion of Type 3 as a submucous leiomyoma Type definitions and distinctions Distinction between Types 0 and 1; 6 and 7 Distinction between Types 2 and 3; 4 and 5
AUB-C	No longer includes AUB associated with pharmacologic agents that impair blood coagulation
AUB-I	Now includes AUB associated with all iatrogenic processes including use of pharmacological agents used for anticoagulation and those thought to interfere with ovulation
AUB O	Diagnostic threshold changes based upon the revisions of System 1, described above No longer includes ovulatory disorders associated with drugs known or suspected to interfere with ovulation
AUB N	The name of the category has been changed from "Not Yet Classified" to Not Otherwise Classified There is a brief discussion of a potential new cause of AUB the so-called uterine "niche" or isthmocele following lower segment cesarean section

Documentation of AUB as per PALM-COEIN etiological classification:

The status of each potential contributor of AUB is documented as being present (1) or absent (0) in the acronym. Multiple pathologies are also documented simultaneously, eg. adenomyosis, hyperplasia and coagulopathy in a single patient may be cited as $P_0\ A_1\ L_0\ M_1 – C_1\ O_0\ E_0\ I_0\ N_0$. This approach may be relevant for research settings. Mentioning abbreviation for each identified cause after letters 'AUB' is another alterative and simplified way of documentation in clinical settings and above example may be cited as 'AUB-A, -M, -C'. A leiomyoma may be documented as $P_0\ A_0\ L_{1(SM)}\ M_0 – C_0\ O_0\ E_0\ I_0\ N_0$ or AUB-L (SM – type0/1/2) if submucosal or as $P_0\ A_0\ L_{1(O)}\ M_0 – C_0\ O_0\ E_0\ I_0\ N_0$ or AUB-L(O-type 3/4/5/6/7/8) if other than submucosal.[4]

Brief description of PALM-COEIN classification:

Polyps ($P_1\ A_0\ L_0\ M_0 – C_0\ O_0\ E_0\ I_0\ N_0$ or AUB-P)

Endometrial polyps are epithelial proliferations arising from the endometrial stroma and glands. The majority are asymptomatic. The contribution of polyps to AUB varies widely ranging from 3.7% to 65%. Polyps are categorized as either being present or absent.

Adenomyosis ($P_0\ A_1\ L_0\ M_0 – C_0\ O_0\ E_0\ I_0\ N_0$ or AUB-A)

Two-dimensional transvaginal ultrasonography (TVUS) has been found to have similar sensitivity and specificity to magnetic resonance imaging (MRI) for diagnosing adenomyosis. The morphological uterus sonographic assessment (MUSA) group have suggested eight TVUS criteria for diagnosis of adenomyosis and the presence of two or more is highly associated with its diagnosis. The criteria's are: i) asymmetrical myometrial thickening ii) myometrial cysts iii) hyperechoic islands iv) fan shaped shadowing v) echogenic subendometrial lines and buds vi) translesional vascularity vii) irregular junctional zone vii) interrupted junctional zone. Junctional zone identification and evaluation is best accomplished with three-dimensional ultrasonography.

Leiomyomas ($P_0\ A_0\ L_1\ M_0 – C_0\ O_0\ E_0\ I_0\ N_0$ or AUB-L)

In PALM-COEIN system, primary classification represents presence or absence of one or more leiomyomas, regardless of the location, number and size. The secondary classification includes further subdivision into submucous or 'SM' with at least 1 submucous leiomyoma or others 'O'. This aids the clinician to distinguish myomas that contact the endometrium as these are most likely contributing to AUB.

The tertiary classification of myomas is based on the Wamsteker system for submucous leiomyomas (SM-Type 0, 1 and 2)[6] and expanded to include tumors that just contact the endometrium without distorting cavity (Type 3), intramural myomas that have myometrium between boundaries and endometrium & serosa (Type 4) and subserous tumors that are categorized according to the relative proportions of the tumors within the myometrium (Types 5, 6, & 7). Type 8 leiomyomas are those that do not involve the myometrium such as cervical, broad ligament and parasitic leiomyomas. When a leiomyoma abuts or distorts both the endometrium and serosa, it is categorized first by the submucosal classification, then by the subserosal location, with these two numbers separated by a hyphen. This tertiary classification is useful for clinicians especially while planning myomectomy. In the 2018 revision Types 0 and 7 are distinguished from Type 1 and 6 by having a stalk diameter that is 10% or less than the mean diameter of the leiomyoma. Type 2 and 3 leiomyoma are distinguished on hysteroscopy

based upon the lowest filling pressure needed to visualise the endometrial cavity. Type 4 and Type 5 are differentiated based upon distortion of the serosa (Type 5) as determined by ultrasonography or MRI. The revised system also proposed a minimal data set for describing leiomyomas which includes an estimate of total uterine volume based on imaging as well as an estimate of the number of leiomyomas (1, 2, 3, 4, or greater than 4). If imaging is not available, the minimum data set should include an estimate of uterine size on clinical examination as equivalent to a gravid uterus of"X" weeks. When transvaginal ultrasonography or MRI are available, the location (anterior, posterior, left, right, or center), in the vertical plane as upper half, lower half, or both and the estimated volume of up to four individual leiomyomas should be recorded. When more than four leiomyomas are present, the volume of the largest should be recorded, as a minimum and also if other leiomyomas are thought to be important for clinical decision making based on location, the volume of these should also be recorded.

Malignancy (P_0 A_0 L_0 M_1 – C_0 O_0 E_0 I_0 N_0 or AUB-M)

AUB-M includes both malignancy (leiomyosarcoma, endometrial cancer) and atypical endometrial hyperplasia.[11] They are further "sub-classified" by the appropriate WHO or FIGO system.

Coagulopathy (P_0 A_0 L_0 M_0 – C_1 O_0 E_0 I_0 N_0 or AUB-C)

Abnormal uterine bleeding associated with any of systemic disorders of haemostasis is designated as AUB-C.

Ovulatory Disorders (P_0 A_0 L_0 M_0 – C_0 O_1 E_0 I_0 N_0 or AUB-O)

Ovulatory disorders leading to AUB are common in peri-menarcheal and perimenopausal women and endocrinopathies such as polycystic ovarian syndrome, hypothyroidism, and hyperprolactinemia as well as other factors including mental stress, obesity, anorexia, weight loss, and extreme exercise. Anovulatory cycles may contribute to AUB by unopposed estrogen effects on the endometrium causing marked proliferation and thickening resulting in HMB along with an altered frequency of menstruation. Women with AUB-O have menstrual cycles as described in system 1.

Endometrial Causes (P_0 A_0 L_0 M_0 – C_0 O_0 E_1 I_0 N_0 or AUB-E)

AUB due to primary endometrial dysfunction is termed AUB-E. It may be due to increased local production of vasodilators (prostaglandin E2, prostacyclin), decreased local production of vasoconstrictors (endothelin-1, prostaglandin F2a), excessive synthesis of plasminogen activator or accelerated lysis of endometrial clot.[6] Laboratory testing for such abnormalities are not currently available to clinicians. AUB due to endometrial inflammation or infection is also included in AUB-E[1]. According to FIGO consensus, there are no tests to be performed in this situation. AUB-E remains a diagnosis of exclusion of other identifiable abnormalities in women with seemingly normal ovulatory cycles[1] and is also suspected when a structural abnormality is an unlikely cause of AUB such as Type-6 leiomyoma.[7]

Iatrogenic (P_0 A_0 L_0 M_0 – C_0 O_0 E_0 I_1 N_0or AUB-I)

Iatrogenic causes of AUB include exogenous therapy leading to unscheduled endometrial bleeding and is associated with the use of exogenous steroids (ie, estrogen / progestin therapy gonadotropin-releasing hormone agonists and antagonists, aromatase inhibitors, selective estrogen receptor modulators, selective progesterone receptor modulators), intrauterine systems /devices or other systemic/ local pharmacological agents e.g. anticonvulsants - valproic acid and antibiotics - rifampin and griseofulvin, anticoagulant drugs such as warfarin, heparin, low molecular weight heparin, nonsteroidal pharmaceuticals that contribute to ovulatory disorders, such as those that affect dopamine metabolism, including phenothiazines and tricyclic antidepressants. AUB-I is typically associated with continuous systemic or intrauterine delivery routes The use of an intrauterine device may cause a low-grade endometritis which may also contribute to AUB.

Not Otherwise Classified (P_0 A_0 L_0 M_0 – C_0 O_0 E_0 I_1 N_0 or AUB-N)

Abnormal bleeding associated with the remainder of rare or ill-defined causes are categorized as AUB-N. Examples include arteriovenous malformations[8], caesarean scar defects or Isthmocele[9], endometrial pseudo aneurysm, myometrial hypertrophy and chronic endometritis (not precipitated by an intrauterine device). Besides, there may exist other disorders, not yet identified, that would be defined only by biochemical or molecular biological assays. As further evidence becomes available, such abnormalities maybe allocated a separate category, or they could

be assigned to one of the existing categories in the classification system.

To conclude, the PALM-COEIN system is designed as a practical system for use by clinicians worldwide for an easy and consistent evaluation of AUB.

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AUB: Evaluation and Management

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Introduction

Abnormal uterine bleeding is defined as bleeding from the uterine body that is abnormal in frequency, duration and amount arising in the absence of pregnancy. In 2011, FIGO devised the much-awaited standardised nomenclature for AUB known as PALM-COEIN. Based on the etiopathogenesis of AUB and it includes both structural (PALM) and non-structural causes (COEIN).

Abnormal uterine bleeding is one of the most frequent symptom in women in the reproductive age group attending gynaecological OPD. In India, the prevalence of AUB is reported to be around 17.9%.[2] Usually, the first episode of irregular bleeding in a non-pregnant lady is not of much concern.But, in a minority of women, it can be the only symptom of an alarming condition i.e. malignancy. So, proper and meticulous evaluation of all the cases of abnormal uterine bleeding should be the norm.

In 2011, FIGO devised the much-awaited standardised nomenclature for AUB known as PALM-COEIN. Based on the etiopathogenesis of AUB and it includes both structural (PALM) and non-structural causes (COEIN).

Evaluation of a case of AUB

History

It is important to rule out pregnancy in any woman presenting with AUB in the reproductive age group. Other key points include:

- Normal cyclicity, amount and duration of menstrual flow prior to onset of complaints.
- Duration of complaint and abnormality one is suffering from.
- Associated complaint of pain or lump abdomen, vaginal discharge, fever.
- History suggestive of underlying disorder of hemostasis which includes:
  - History of heavy menstrual flow since menarche.
  - Excessive or prolonged bleeding during surgery, dental workor childbirth.
  - Frequent epistaxis or bruising (>once /month), frequent gum bleeding or family history of bleeding diathesis.
- History of use of contraceptives, medicines like anticoagulants, tamoxifen.
- History suggestive of thyroid disorder
- History of diabetes mellitus, hypertension.
- Family history of malignancy.

Examination

It is important to record the following findings:

- Vitals including pulse rate, blood pressure, respiratory rate especially in cases of acute AUB.
- Body Mass Index.
- State of pallor, presence of cyanosis, clubbing, icterus, pedal edema and lymph nodes.
- Thyroid enlargement.
- Acne, hirsutism (Ferriman-Gallwey score).
- Breast examination.
- Abdominal examination to look for palpable masses and presence of free fluid.
- Speculum examination to look for source and amount of bleeding, nature of vaginal discharge and state of cervix and vagina. Take PAP smear if indicated.
- Vaginal, rectal or combined examination to confirm the abdominal and speculum findings.

Investigations

The laboratory evaluation and other investigations are guided by the findings of history and examination.

Blood Tests

- Urine pregnancy test or serum Beta HCG to rule out pregnancy related event.
- Complete blood count including haemoglobin, haematocrit and platelet count to assess the status of anaemia and coagulability.
- Coagulation profile including bleeding time, clotting time, partial thromboplastin time, activated partial thromboplastin time, von Willebrand factor assay, ristocetin factor assay and factor VIII activity is indicated in women with positive screen for coagulopathies in consultation with a haematologist.
- Thyroid stimulating hormone and liver function test if clinically indicated.

Imaging

- Ultrasound pelvis : preferably done after menses that helps in detecting structural abnormalities including thickened endometrium, status of myometrium, fibroids, adenomyomas, adenomyosis or adnexal masses. Trans-abdominal or trans-vaginal ultrasound can be done depending on the feasibility, but trans-vaginal is preferred.
- Ultrasound- doppler should be done in cases of suspected arteriovenous malformation, malignancy, and to differentiate between fibroids and adenomyomas.
- Three-dimensional ultrasound is preferred for mapping intracavitary and myometrial fibroids. (Non-invasive alternative to hysteroscopy)
- Saline infusion ultrasonography is indicated to differentiate thickened endometrium from endometrial polyps in settings where hysteroscopy is not available.
- Hysteroscopy helps in identifying intracavitary lesions and taking directed biopsies. Outpatient hysteroscopy may be the initial investigation in women where intracavitary lesion or endometrial pathology is suspected on the basis of history & examination.
- Magnetic Resonance Imaging is indicated prior to conservative surgery for fibroids in order to map the exact number and location of the fibroids and differentiating them from adenomyomas. It can also be done in cases of suspected malignancy.

Evaluation of Endometrium

It is indicated in:[3]

- All women above 40 years of age.
- In women less than 40 years of age who have high risk factors like irregular bleeding, obesity, hypertension, polycystic ovarian syndrome, diabetes, endometrial thickness more than 12mm on ultrasound, use of tamoxifen for hormone replacement therapy or breast cancer, family history of malignancy of ovary, breast, endometrium or colon.
- In women with persistent AUB that is unexplained or unresponsive to treatment.

Endometrial aspiration is the procedure of choice for obtaining endometrial sample for histopathological diagnosis. In cases with increased endometrial thickness on USG and histopathology report showing inadequate sample or atrophic endometrium, hysteroscopy is indicated to rule out polyps. Dilatation and curettage is not recommended.

Management

In women presenting with acute AUB, she should initially be assessed for the signs of hypovolemia & hemodynamic instability and promptly resuscitated before evaluating the likely aetiology of AUB. The management strategy is to control the present episode of heavy bleeding and to reduce menstrual blood loss in subsequent cycles. Table 1 lists the various treatment regimens for acute AUB.[4] Blood transfusion is indicated in women with severe anaemia (Hb<7 gm%).

Table 1: Medical treatment regimens for acute AUB

Drug	Suggested Dose	Dose Schedule
Conjugated equine estrogen(CEE)	25 mg I/V	Every 4-6 hours for 24 hours
Combined oral contraceptive(COC)	Monophasic combination containing 35 mcg of ethinyl estradiol	Three times a day for 7 days
Medroxyprogesterone acetate	20 mg orally	Three times a day for 7 days
Tranexamic acid	1 gm orally or 10mg/kg I/V (max 600 mg/dose)	Three to four times a day for 5 days

Intrauterine insertion of a Foley's catheter and tamponade by inflating its bulb with saline has been shown to control the bleeding effectively in select cases. In patients where I/V CEE has been given, COCs or oral progestins should be initiated simultaneously and tapered gradually.

Once the acute episode of bleeding has been controlled, further treatment of AUB depends on the aetiology based on the PALM-COEIN classification. The details of treatment for AUB-A and AUB-L will be discussed in details in subsequent articles in this edition. The rest are discussed here.

- AUB-P

  Hysteroscopic polypectomy followed by its histopathological examination (HPE) is the definitive treatment option.If the HPE report confirms a benign lesion and the patient is not desirous of fertility, Levonorgestrel- Intrauterine system (LNG-IUS) may be considered. If the HPE report is suggestive of malignancy, the woman should be managed as a case of AUB-M.

  AUB- A: This is discussed in the article on Adenomyosis

  AUB –L : This is discussed in the article on uterine fibroids

- AUB-M

  In patients with endometrial hyperplasia without atypia, LNG-IUS insertion is the first line therapy.

Continuous oral progestins (medroxyprogesterone 10–20 mg/day or norethisterone 10–15 mg/day) can also be used if the woman declines LNG-IUS(to be given for minimum 6 months). Follow up endometrial biopsy(EB) is indicated after 6 months and at least two consecutive 6-monthly negative biopsies should be obtained prior to stopping surveillance. In women with high risk factors for relapse, surveillance with annual EBs should be continued. Hysterectomy should be done in women not willing for follow up; those showing non-regression, relapse or progression to atypia during follow up; and those with persistence of bleeding symptoms.[5]

In women with endometrial hyperplasia with atypia which has a high risk of progression to endometrial cancer, total extra facial hysterectomy is the treatment of choice.Laparoscopic route for hysterectomy is preferred over an abdominal route because of benefits such as a shorter hospital stay, less postoperative pain and quicker recovery. If the woman aspires to preserve her fertility or is at high risk for surgery, first-line therapy with the LNG-IUS should be recommended. Oral progestins are second-best alternative option with follow up biopsy every 3 months till 2 negative biopsies are obtained followed by long term follow up with 6-12 monthly biopsies till hysterectomy is undertaken.[5]

In women with diagnosed endometrial malignancy, standard treatment protocol should be followed.

- AUB-C

  Up to 13% of women with AUB have von Willebrand disease and around 20 % of women may have an underlying coagulation disorder.[6] Disorders like leukemia, liver failure and medications including chemotherapeutic agents and anticoagulants may also be associated with AUB. In women diagnosed with coagulopathy as the cause of AUB, tranexamic acid is the first line treatment (in the dose of maximum 1gm 6 hourly) followed by oral contraceptives or LNG-IUS as a second line therapy. In cases of persistent heavy bleeding in women with von-Willebrand disease, desmopressin can be given in consultation with haematologist. It can be given via intranasal, intravenous or subcutaneous route. Recombinant factor VIII, von Willebrand factor or specific factors may also be required in cases of uncontrolled heavy vaginal bleeding not responding to usual medical treatments. NSAIDs are strictly contraindicated in these cases, owing to their adverse effects on platelets and liver functions.

- AUB-O

  Oral contraceptives for a total duration of 6-12 months are considered the first line of therapy in women with ovulatory dysfunction. Alternatively, cyclic progestins from Day 5 to 26 or norethisterone for 21 days can also be prescribed in patients having contraindications to the use of COC. Cyclical luteal phase progestins are not recommended. LNG-IUS can also be offered. The success of any of the available treatment options should be assessed after a year to evaluate the need to continue or discontinue the treatment or to decide for hysterectomy.

- AUB-E

  In women with endometrial cause of AUB, treatment options available are similar to that for AUB-O

- AUB-I

  It is usually associated with the use of continuous oestrogen or progestin, gonadotropin-releasing hormone (GnRH) agonists, aromatase inhibitors, selective oestrogen receptor modulators (SERMs) and more rarely selective progesterone receptor modulators (SPRMs). If medicinal intake is suspected to be the cause of AUB, every attempt should be made to switch to a better alternative if available or plan insertion of LNG-IUS.

- AUB-N

  LNG-IUS is recommended as the first line therapy to ameliorate bleeding and COC can be tried as second line option, when LNG-IUS is contraindicated or not approved by the woman. Non-hormonal options like NSAIDs or tranexamic acid can also be given for cyclical AUB. Conservative surgical options such as endometrial ablation may be considered in low risk women when medical therapy fails. GnRH agonist with add back therapy are recommended in women where above treatment options have failed or contraindicated. In women with arterio-venous malformation, uterine artery embolization is the treatment of choice. Hysterectomy stands as the last resort when all other treatments have failed. The route of hysterectomy (vaginal/abdominal/laparoscopic) should be decided by the treating doctor in discussion with the patient.

Conclusion

Abnormal uterine bleeding is common condition which can be debilitating for a woman. Therefore, it is imperative that a prompt evaluation should be carried out and treatment instituted.

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UROGYNAECOLOGY WORKSHOP

Organised by Department of Obstetrics and Gynaecology, AIIMS, New Delhi

Under Aegis of AOGD Urogynaecology Committee,
FOGSI Urogynaecology Committee & Dept of Obst. & Gynae, AIIMS

Venue: **Gynae Seminar Room (3rd Floor)** *Date:* **26 September, 2019 (Thursday)**

Surgeons: Prof. Neerja Bhatla, Prof. J B Sharma, Dr. Garima Kachhawa, Dr. Reeta Mahey, Dr. Rajesh Kumari

Cases (Subject to availability of cases): TVT-O, Burch colposuspension, TLH with bilateral uterosacral plication, CPT repair, RVF repair, Manchester repair with lap ligation, Vaginal hysterectomy with pelvic floor repair.

08:30 AM - 9:00 AM	:	**Registration**
9:00AM - 10:30AM	:	**Live Demonstration of Urogynaecological Surgeries**
	:	Hall Incharge:Dr. Tanudeep Kaur
	:	**Chairpersons**: Prof. Sunesh Kumar, Prof. Neerja Bhatla, Dr.Sunita Malik, Dr. Manju Puri
10:30AM - 10:45 AM	:	Tea
10:45AM - 1:00 PM	:	**Live Demonstration of Urogynaecological Surgeries (ctd.)**
	:	Hall Incharge : Dr. Kavita Pandey
	:	**Chairpersons**: Dr KK Roy, Dr. Vatsla Dhadwal, Dr. Neeta Singh, Dr. Tarini Taneja, Dr. Gowri Dorairajan
1:00PM - 1:30PM	:	Lunch
[1:00PM - 1:20PM]	:	Role of Haemostats in bleeding in Urogynae surgery
[1:20PM - 1:30PM]	:	Role of Fosfomycin in UTI
1:30PM - 2:00 PM	:	**Guest lecture**: Dr. Neeta Thakre
	:	Pelvic floor health for prevention of SUI and POP
	:	**Chairpersons**: Prof. Sunesh Kumar, Prof. Amlesh Seth, Dr. Neena Malhotra, Dr. Aparna Sharma
2:00 PM - 4:00PM	:	**Live Demonstration of Urogynaecological Surgeries (ctd.)**
	:	Hall Incharge:Dr. Bharti Uppal
	:	**Chairpersons:** Dr. Vidhushi, Dr. Juhi Bharti, Dr. Rishi Nayyar
4:00PM - 4:20PM	:	Urodynamics in SUI & OAB
	:	Dr. Amlesh Seth
	:	**Chairpersons:** Dr. Amita Jain, Dr. Jyoti Meena, Dr. Seema Singhal Dr. Karishma Thariani

Prof.Sunesh Kumar
Organising Chairperson

Prof. J.B. Sharma
Organising Secretary

Dr. Rajesh Kumari
Joint Organising secretary

Adenomyosis

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Introduction

Adenomyosis is a benign but challenging gynecological condition causing substantial morbidity and may be present for many years without correct diagnosis. Adenomyosis was first described in 1860 by German pathologist Carl von Rokitansky and is defined as enlarged uterus following areas of the endometrium, both the endometrial glands and stroma, located deep within the myometrium resulting in hypertrophied and hyperplastic myometrium[2]. Adenomyosis commonly occurs together with endometriosis and the two conditions coexist in approximately 20% of affected patients. It affects 20–30% of women undergoing hysterectomy, independent of the indication for surgery. There is also an association with increasing age, multiparity, spontaneous and induced abortions and endometrial hyperplasia.

Pathogenesis

Adenomyosis is diagnosed when ectopic endometrial glands are found within myometrium of uterus. According to one popular theory[2], there is a downward invagination of the endometrial basalis layer into the myometrium due to myometrial weakness or altered immunity which leads to disruption of endometrial-myometrial interface.

Diagnosis

Gold standard in the diagnosis of adenomyosis is histological examination that is presence of endometrial tissue more than 2.5 mm below the endomyometrial junction or a junctional zone thickness of more than 12 mm[3]. Adenomyosis is classified as diffuse adenomyosis or focal adenomyoma when the ectopic endometrium forms a circumscribed nodular collection. The posterior uterine wall is most frequently affected. Levgur et al created the following grading system to describe the depth of adenomyotic foci: deep (>80%), intermediate (40%–80%), and superficial (<40%).[4]

Preoperative diagnosis can be made from classical presentation of dysmenorrhea, dyspareunia & heavy menstrual bleeding with uniformly enlarged globular uterus after excluding uterine fibroid & endometriosis.

Investigations

Trans abdominal ultrasound scan:

Features seen on transabdominal scan are uterine enlargement or asymmetric thickening of anterior & posterior myometrial walls. Due to poor image resolution of myometrium with transabdominal scan it cannot reliably diagnose adenomyosis

Transvaginal ultrasound scan:

USG features of adenomyosis include three or more sonographic criteria: heterogeneity of myometrium, increased echogenicity, presence of linear myometrial striations and anechoic lacunae or myometrial cysts[5]. Poor delineation of the endomyometrial junction also raises the probability of adenomyosis. Doppler can help to differentiate adenomyoma from fibroid in doubtful cases.

Three- dimensional Ultrasound:

3-D USG allows better imaging of Junctional Zone (JZ). The Junctional zone is often visible as a hypoechogenic subendometrial halo which is composed of longitudinal and circular, closely packed smooth muscle fibers[6].

Magnetic Resonance Imaging:

Gold standard imaging modality for assessing the junctional zone in the evaluation of adenomyosis is MRI. MRI clearly distinguishes focal and diffuse adenomyosis from leiomyomatosis. The common features on MRI include[7];

- Thickening of the JZ, JZ thickens ≥ 12mm or irregular junctional thickness with a difference of >5 mm between the maximum & minimum thickness.
- Islands of ectopic endometrial tissue identified as punctate foci of high signal intensity on T1 weighted image.
- An ill-defined of low signal intensity in the myometrium on > 2 weighted MR images.

Hysterosalpingography

Occasional findings of spiculations (1-4mm) from endometrium towards myometrium or a uterus with “tuba erecta” finding may be suggestive of adenomyosis[8]. HSG is seldom used for diagnosis of adenomyosis.

Hysteroscopy

Hysteroscopic findings associated with adenomyosis are hypervascularization, irregular endometrium

with endometrial defect, strawberry pattern or cystic haemorrhagic lesions[9]. Limited data is available on diagnostic accuracy of these features.

Shear Wave Elastography

Application of shear wave elastography to measure myometrial stiffness during transvaginal scan may improve diagnostic accuracy of adenomyosis[10].

Hysteroscopic & laparoscopic myometrial biopsy

Popp et al[11] observed that sensitivity of single myometrial hysteroscopic biopsy ranges from 8-18.7% for diagnosing adenomyosis whereas specificity was 100% among 680 specimens in 68 surgically removed uterus. Jeng et al [12conducted] a prospective, nonrandomized study of 100 patients, sensitivity of laproscopic myometrial biopsy was 98% and the specificity was 100%. The study suggested that laparoscopy guided myometrial biopsy is an important tool in diagnosis of diffuse adenomyosis.

Management

Management depends on type, extent of disease & presenting symptoms. Hysterectomy is the mainstay but there are alternative medical treatments & conservative surgeries for younger population.

Medical Management

Principles of medical management of adenomyosis are similar to that of endometriosis which is aimed at reducing production of endometrial estrogens[14]. The main objectives of medical treatment are:

- Inhibition of ovulation
- Cessation of menstruation
- Achievement of a stable steroid hormone milieu.

Agents used for medical management create hypoestrogenic, hyperandrogenic or hyperprogestogenic environment. Their mechanism of action and adverse effects are summarized in table below (Table-1).

Conservative procedures

- Excision of adenomyosis (Adenomyomectomy)
- Radiofrequency ablation (Transcervically or laproscopic or USG guided)
- Lap Myometrial electrocoagulation
- Endometrial ablation or resection using H-incision technique.

The indications for conservative surgery include dysmenorrhea and hypermenorrhea that are difficult to control with medication, infertility and recurrent miscarriages, and a desire to preserve fertility or the uterus. Magnetic resonance imaging (MRI) examination is performed in order to grasp accurately the location and extent of the uterine adenomyosis and the position of the uterine cavity in order to determine the site, direction, and depth of the incision to be made into the uterus. The various conservative surgeries are summarized in Table 2.

Conservative surgery followed by GnRH agonist therapy has proven to be effective for control of symptoms[15].

Table1: Medical management of Adenomyosis

Drugs	Mechanism of action	Side effects
Progestins	Decidualization & atrophy of endometrial tissue	Irregular bleeding, nausea/vomiting, mood swings, hot flush
Gestrinone	Progestational withdrawal effect & inhibition of ovarian steroidogenesis	Androgenic side effects, risk of metabolic syndrome
Selective ER Modulator (Tamoxifen, Raloxifene)	- Differential ER expression in given target tissue - Differential expression & binding to ER coregulator proteins	Hot flush, leg cramp, hyper coagulation status
Aromatase inhibitors	Inhibition of estradiol synthesis	Vasomotor symptoms,gential atrophy, bone loss
Danazol	Androgenic & hypoestrogenic environment	Androgenic side effects (weight gain, muscle cramps, acne, hirsutism), hepatotoxicity
Selective PR Modulator (Ulipristal acetate, Mifepristone)	- Differential PR expression in given target tissue - Differential expression & binding to PR co-regulator proteins	Headache, nausea, fatigue, dizziness
GnRH agonists	Down regulation of gonadotropin releasing hormone	Osteoporosis, vasomotor symptoms, mood swings
LNG IUS (Mirena)	Local effect on endometrium	Variable bleeding & spotting
Oral contraceptives	Decidualization & atrophy of endometrial tissue & decrease of retrograde menstruation	Nausea/vomiting, headache, irregular bleeding, hypercoagulation status

Table 2: Conservative Surgical Options in Adenomyosis

Excisional surgical Techniques	Method
1. Transverse H-incision technique	H- shaped incision on anterior uterine wall & serosa is widely separated from the underlying myometrium. The adenomyoma tissue is removed using an electro-surgical scalpel or scissors. A tension-less suturing technique is used to apposition the myometrial edges and close the wound in one or two layers.
2. Wedge resection of uterine wall (open or laparoscopic)	Part of seromuscular layer with adenomyoma removed by wedge resection after a sagittal incision in the uterine body.
3. Asymmetric dissection of uterus	Uterus dissected in asymmetrical fashion, removing adenomyotic lesion using loop electrode and a high frequency cutter. From the incision, the myometrium is dissected diagonally as if hollowing out the uterine cavity. While inserting the index finger into the uterine cavity, the adenomyosis lesion is excised to >5 mm of the inner myometrium. The lesion is then excised to >5 mm of the serosal myometrium. Afterwards, the uterine cavity is sutured and closed, followed by uterine reconstruction.
4. Triple flap method	Triple procedure:a) Adenomyomectomy b) reconstruction of a uterine cavity which can sustain subsequent pregnancy in which an endometrial uterine muscle flap is prepared by metroplasty c) reconstruction of a uterine wall resistant to rupture. The uterine muscle on the serosal side is used to fill the large uterine wall muscle defect.
5. Laparoscopic Adenomyomectomy	The laparoscopic surgical method includes a longitudinal or transverse incision of the uterine wall along the adenomyoma. This is followed by resection of the adenomatosis using a monopolar needle or a laser knife. Sero-muscular layer is the closed in two or more layers or using the double-flap method and removal of the adenomyotic mass may be accomplished using a in bag morcellation.
6. Hysteroscopic adenomyomectomy	Possible for adenomyoma <5 cm or when it is protruding into the uterine cavity

*Surgeries 1-4 are by laparotomy.

Non Excisional Techniques (Uterine sparing)

- Hysteroscopic non excisional techniques-
  - Operative hysteroscopy
  - Rollerball endometrial ablation
  - Transcervical resection of endometrium
  - Endomyometrial resection
- HIFU (High Intensity Focused Ultrasound)-High intensity ultrasound focused on target lesion leading to coaugulative necrosis and shrinkage of lesion.
- UAE (Uterine artery embolization)- It causes decrease in uterine volume & symptomatic relief.

Although powered instruments seem to contribute to reduced blood losses and shortened operative times, such use also results in hardening and discoloration of the incised tissue surface due to heat denaturation, making the boundary between the abnormal and normal tissues less clear. No literature comparing the efficacy and/or safety of various cutting energies has been found. However, there are opinions suggesting that in general lasers cause least thermal denaturation.

Hysterectomy

Definitive treatment for intractable symptomatic adenomyosis is hysterectomy. Increased risk persistent pelvic pain should be explained to patients undergoing hysterectomy for adenomyosis. There is no role of conservative management in women more than 40 years[16].

Adenomyosis & Fertility

Women with adenomyosis have lower implantation rate per embryo transfer, lower clinical pregnancy rate and higher spontaneous abortion rate as compared to women without adenomyosis. Management of women with adenomyosis associated subfertility is very controversial as there is no consensus about conservative surgery. It is unclear who will benefit from medical treatment alone & who will benefit from conservative surgery.

- Routine infertility workup is done for women presenting with adenomyosis associated infertility.
- A long protocol GnRH agonist suppression is given for women with normal ovarian function for spontaneous conception.
- Immediate IVF is advised to women with low ovarian reserve.
- Surgery is not taken in to consideration before ART.
- Repeat long protocol GnRH agonist based IVF/ICSI treatment if natural conception is not possible.
- Indication of conservative surgery if there are severe symptoms & repeat failure of long protocol GnRH agonist based IVF/ICSI therapy.
- Long protocol IVF/ICSI is attempted after 3 months of surgery.

Ina systematic review, Ten studies evaluated exclusive assisted reproductive techniques for infertility related to adenomyosis and showed that the long stimulation

protocol had better outcomes compared to short stimulation protocol in pregnancy rate (43.3% vs 31.8%; P = .0001), live birth (43.0% vs 23.1%; P = .005), and miscarriage (18.5% vs 31.1%; P < .0001).[17]

Obstetric complications of conservative surgery like spontaneous miscarriage, preterm birth, intrauterine growth restriction, preeclampsia, obstetric hemorrhage, spontaneous rupture of unscarred uterus in labor should be discussed with the patient before surgery, however only a few of them are reported in literature. Elective cesarean section seems good choice after conservative surgery for adenomyosis for a better patient safety. Post-adenomyomectomy improvements in dysmenorrhea and hypermenorrhea vary but are recognized. The postoperative pregnancy rate also varies between 17.5% and 72.7%.[18]

A literature review suggested that the risk of uterine rupture after uterine adenomyosis is 6.0% (46). Regardless, the risk of uterine rupture due to pregnancy, after removal of a uterine adenomyosis, is >1.0%[19], compared to 0.26%[20] in pregnancies following myomectomy.

Although there is no consensus regarding the necessary contraceptive period before attempting the subsequent pregnancy, what matters is the resumption of the blood flow and that could vary depending on the individual case.[18]

Conclusion

Management of adenomyosis should be individualized depending on the symptoms & desire for fertility. Focal adenomyosis lesion can be treated laparoscopically. However, diffuse adenomyosis must be treated by laparotomy or preferably by laparoscopically-assisted laparotomy.[18] Open surgery is safer than laparoscopy in that it can thoroughly excise the lesions to prevent the recurrence and to properly reconstruct the defect created by the surgery in order to prevent uterine rupture due to subsequent pregnancy.[18]

Future research on definitive diagnosis and treatment is still needed.

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Events Held

- OI Workshop on in Medical Colleges and 28th July, 2019 at LHMC under the Quality Improvement Sub Committee AOGD



- Masterclass in Gynecologic Oncology on 11th August, 2019 at India International Centre, Delhi by UCMS & GTB Hospital under the aegis of AGOI, FOGSI & AOGD Oncology Committees and AOGIN



- Breaking Silos Across: Adolescent to Menopause on 10th & 11th August, 2019 at Hotel Lalit by MAMC Dr Sudha Prasad

- Independence Day Celebration CME on 12th August at Madhuban Hotel by FOGsd under the aegis of AOGD.



- CME on **"Revisiting Basics of Infertility"** on 17th August at Ram Manohar Lohia Hospital.

  CME on "Revisiting Basics of Infertility" was conducted by RML Hospital with Dr Indu Chawla under aegis of Infertility Committee AOGD and Reproductive endocrinology committee on 17th august 2019. There were lectures and panel discussions on various aspects of Basic Infertility. It was attended by over 100 delegates including postgraduate students and was well appreciated.




- Hysteroscopy Live Workshop cum Training Program was organised by Swami Dayanand Hospital, Delhi on 18th August, 2019 under the Aegis of Endoscopy Committee – AOGD.




- Combined activity by Infertility committee AOGD & Quality Improvement Committee AOGD with Delhi Gynae Forum (North) on "Delivering the best to the Infertile Couple- Quality Improvement Initiative" on 29th August at Fortis Hospital Shalimar Bagh

  The two committees joined hands under Dr Manju Puri and Dr Surveen Ghumman with the aim of bringing quality improvement into Infertility Practice. About 40 Gynecologists attended and lectures were based on do's and don't's in basic infertility management with enthusiastic discussion

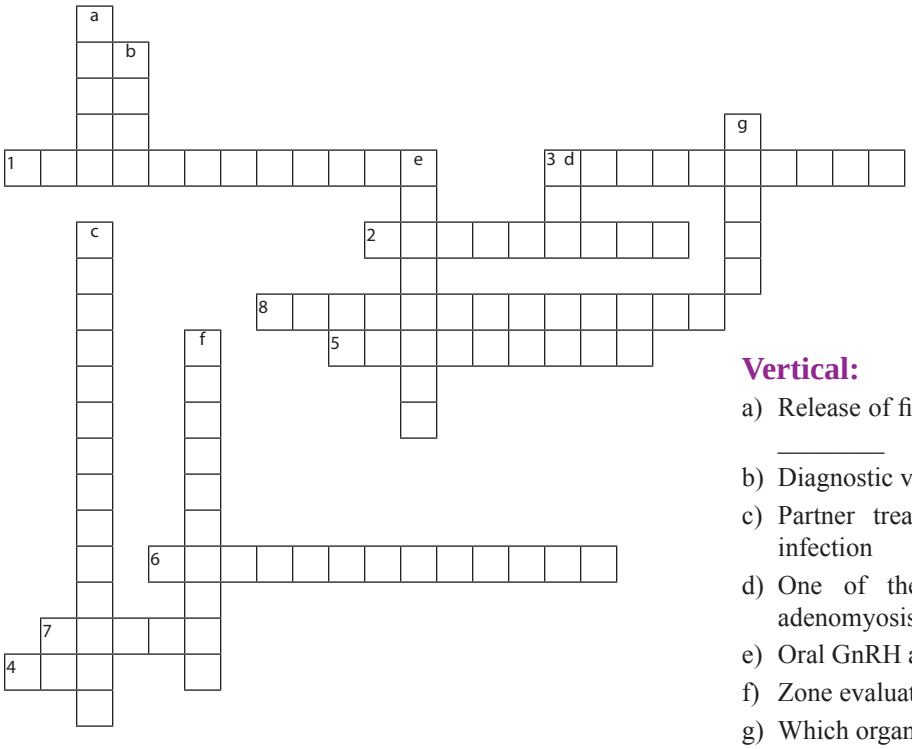




The Maze of Knowledge

Garima Patel, Vidushi Kulshrestha
Department of Obst. Gynaec. and Urogynaecology, AIIMS, New Delhi

CROSSWORD



Vertical:

a) Release of fishy odour on adding KOH called as _____
b) Diagnostic vaginal cells in bacterial vaginosis
c) Partner treatment required in which vaginal infection
d) One of the uterine sparing treatments for adenomyosis
e) Oral GnRH antagonist
f) Zone evaluated on MRI in adenomyosis
g) Which organ toxicity occurs with ullipristal

Across:

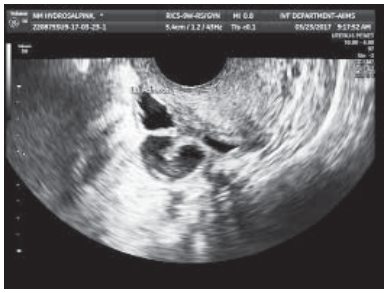
1. One of the oral drugs causing PAEC
2. Classification system FOR Submucosal fibroidS
3. Oral drug which is effective in reducing size of fibroid
4. Gold standard imaging modality to diagnose adenomyosis
5. FIGO classification system of AUB
6. Most common coagulation disorder associated to puberty menorrhagia
7. Triple flap method for adenomyosis is described by _____
8. Disulfiram like reaction causing drug

PICTORIAL QUIZ

Q1. Identify the condition

Name the characteristic imaging feature for its diagnosis

_____



Watsapp your answers to **9211656757.**
Names of first three correct entries will be mentioned in the next issue

Refer page 43 for previous answer key.

41th Annual Conference AOGD

Date: **28th - 29th September 2019 I *Venue:* Eros Hotel, Nehru Place, New Delhi**

28th September 2019 (Day 1) Hall 1

Session 1	Obstetrics :	Speaker	Chairpersons
9:00-9:15	Recurrent pregnancy Loss	Dr Mala Arora	
9:15-9:30	Stage Based management of FGR	Dr Sangeeta Gupta	
9:30-9:45	Managing Rh Negative Pregnancy	Dr Aparna Sharma	
9:45-10:30	Hyperglycemia in Pregnancy : Case based Management	Dr Ashok Kumar	**Panelists** Dr Chitra Setya Dr Madhavi Gupta Dr Manju Khemani Dr Pikee saxena Dr Smiti Nanda
Session 2	**Plenary : 10:30-1:30**		
	Key Note Addresses		
10:30-10:50	Cervical Cancer Elimination: The beginning of the End	Dr Neerja Bhatla	**Chair Persons** Dr VL Bhargava Dr N B Vaid Dr Swaraj Batra
10:50-11:10	Recurrent Pregnancy Loss Through the lens of Immunology	Dr Sudha Prasad	
	AOGD Past President Oration		
11:15-11:45	Maternal Mortality and Near Miss : Steps for reduction	Dr Abha Singh	**Chairpersons :** Dr SN Mukherjee Dr Reva Tripathi Dr Sunesh Kumar Dr Archana Verma
	Inauguration		
11:45-12:30		Ms Meenakshi Lekhi Director, AIIMS Dr Neena Raina Dr Sunesh Kumar Dr Ashok Kumar Dr Vatsla Dadhwal	
12:30-1:30	Competition papers	**Judges** Dr Chitra Raghunandan Dr Anupam Kapoor Dr Vanita Suri Dr Smiti Nanda	
Session 3	**Fetal Medicine**	**Speakers**	**Chairpersons**
1:30-1:45	Role of USG in Multiple gestations	Dr Vandana Chadda	Dr Manisha Kumar Dr Shreyasi Sharma Dr Manu Goyal
1:45-2:00	Establishing services for caring of multiple pregnancies	Dr Soma Mukherjee	
2:00-2:15	Screening in twins	Dr Anita Kaul	
		Moderator	**Panelists**
2:15-3:00	Approach to common fetal anomalies: Panel	Dr Vatsla Dadhwal	Dr Poonam Tara Dr Akshtha Sharma Dr Seema Thakur Dr Anu Thukral Dr Prabudh Goel
Session 4	**Urogynaecology**	**Speaker**	**Chairperson**
3:00-3:15	OAB: Evaluation and Management	Dr Amita Jain	TBA
		Moderators	**Panelists**
3:15-4:00	Panel : Current diagnosis and Management of SUI	Dr J B Sharma Dr Rajesh Kumari	Dr Ranjana Sharma Dr Achla Batra Dr Rishi Nayyar Dr Geeta Mediratta Dr Monika Gupta Dr Karishma
Session 5	**Debates**	**Speakers**	**Moderators**
4:00-4:20	Freeze all for all	For: Dr Nymphea Against: Dr Tanya Bakshi	Dr Nivedita Sarda Dr Madhusudan De Dr Sujata Agarwal
4:20-4:40	NIPT can replace Invasive testing	For : Dr Chanchal Against: Dr Reema	
4:40-5:00	Steroid after 35 weeks	For: Dr Sumitra Against: Dr Nidhi Khera	
5:00-5:20	Induction at 39 weeks	For: Dr Taru Gupta Against: Dr Jyoti Meena	

28th September 2019 (Day 1) Hall 2

Session 1	Reproductive Endocrinology and Infertility	SPEAKERS	Chairpersons
9:00-9:15	Fibroids and fertility	Dr Surveen Ghumman	Dr Jyoti Malik Dr Jigyasa Dr Ila Gupta
9:15-9:30	Social Egg Freezing : Myths Vs Realities	Dr Neeta Singh	
9:30-9:45	Premature ovarian insufficiency	Dr Sonia Malik	
9:45-10:30	Panel: PCOS and fertility -can we improve outcome	**Moderator** Dr Neena Malhotra Dr Reeta Mahey	**Panelists** Dr Anjali Tempe Dr Sunita Arora Dr Anupama Bahadur Dr Richa Katiyar Dr Leena Wadhwa Dr Kavita Khoiwal
Session 2	**Plenary 10:30-1:30**		
	Key Note Addresses		
10:30-10:50	Cervical Cancer Elimination: The beginning of the End	Dr Neerja Bhatla	**Chair Persons** Dr VL Bhargava Dr N B Vaid Dr Swaraj Batra
10:50-11:10	Recurrent Pregnancy Loss Through the lens of Immunology	Dr Sudha Prasad	
	AOGD Past President Oration		
11:15-11:45	Maternal Mortality and Near Miss : Steps for reduction	Dr Abha Singh	**Chairpersons :** Dr SN Mukherjee Dr Reva Tripathi Dr Sunesh Kumar Dr Archana Verma
	Inauguration		
11:45-12:30		Ms Meenakshi Lekhi, Director, AIIMS Dr Neena Raina Dr Sunesh Kumar Dr Ashok Kumar Dr Vatsla Dadhwal	
12:30-1:30	Competition papers	**Judges** Dr Chitra Raghunandan Dr Anupam Kapoor Dr Vanita Suri Dr Smiti Nanda	
Session 3	**Reproductive Endocrinology and Infertility**	**Speaker**	**Chairpersons**
1:30-1:45	Hirsuitism: Case based Management	Dr Garima K	Dr Jyoti Agarwal Dr Seema Varshney Dr Anita Rajohria
1:45-2:00	Male infertility : what a gynecologist should know	Dr Pankaj Talwar	
2:00-2:15	Fertility Preservation-For Whom-when	Dr Nalini Mahajan	
Session 4	**Ethics**	**Speaker**	**Chairperson**
2:15- 2:45	Ethics in Medical Practice	DMC Speaker	Dr Subodh Garg Dr Reva Tripathi Dr Shashilata Kabra
2:45-3:00	Landmark Medicolegal Cases in Gynaecology and Obstetrics	Dr Anita Sabharwal	
Session 3	**One caesarean; a Ton of Complications**		
3:00-3:15	Scar ectopic	Dr Bijoy Nayak	Dr Jyoti Bhaskar Dr Preeti Tyagi Dr Shashi Prateek
3:15-3:30	Reducing caesarean section rates	Dr Renu Mishra	
3:30-3:45	Morbidly Adherant Placenta : Diagnosis and management	Dr Amita Suneja	
Session 5	**Special Situations in Gynaecology**		
3:45-4:00	Postmenopausal Bleeding	Dr Vanita Suri	Dr Poonam Goel Dr Seema Sehgal Dr Chandra Mansukhani
4:00-4:15	Medical Management of fibroids	Dr Pratima Mittal	
4:15-5:00	Panel : Case Based Approach to AUB	Dr Nutan Agarwal Dr Vidushi Kulshreshta	Dr Sanjeevni Khanna Dr Harsha Khullar Dr Mitra Saxena Dr Kavita Bhatti Dr Aruna Nigam Dr Ramandeep Dr Sumanlal

41th Annual Conference AOGD

Date: **28th - 29th September 2019 I *Venue:* Eros Hotel, Nehru Place, New Delhi**

29th September 2019 (Day 2) Hall 1

Session 1	Critical Care in obstetrics		Chairpersons
9:00-9:15	Assessment of Critically ill Women – Obstetric Triage	Dr Kiran Agarwal	Dr Ratna Biswas Dr Sonal Bathla Dr Geeta Kinra Dr J B Sharma
9:15-9:30	Surviving Sepsis Campaign	Dr Kiran Guleria	
9:30-10:15	Maternal Collapse: Case scenarios	Dr Jyotsna Suri	Dr Kanwal Gujral Dr Jaishree Sunder Dr Achla Batra Dr Himsweta Srivastava Dr Madhu Ahuja Dr Sunita Goyal Dr Rachna Agarwal
Session 2	**Intrapartum**		
10:15-10:30	Defining normal and abnormal labor	Dr Asmita Rathore	Dr Rashmi Vyas Dr Suman Mediratta Dr Meenakshu Ahuja
10:30-10:50	Optimising labor and Birthing positions and RMC	Dr Evita Fernandez	
10:50-11:05	Interpreting CTG	Dr Rinku Sengupta	
Session 3 11:00-1:00	**Plenary**		
	Brigadier Khanna Oration		
11:05-11:35	Evolution and Advances in Minimally invasive Gynecology : My Journey Through Endoscopy	Dr Alka Kriplani	Dr SB Khanna Dr Kamal Buckshee Dr SS Trivedi Dr Sudha Salhan
	Keynote Addresses		
11:35-11:55	Obstetrics Care Through Prism of Quality	Dr Manju Puri	Dr Urmil Sharma Dr Ranjana Sharma Dr Sanjeevni Khanna Dr Neerja Goel
11:55-12:15	Medical Education : From Past to Future	Dr Sunesh Kumar	
12:15-1:15	QUIZ	Dr KK ROY Dr Vidushi Dr Juhi Bharti	
1:15-2:00	Basic Life Support	Dr Nishkarsh Dr Gyanendra	
2:00-2:15	Slogan Competition	Dr Sonal Bathla Dr Anita Rajohria	
Session 4 2:15-3:00	**Cutting Edge Technology**		
2:15-2:30	Vaginal Rejuvenation	Dr Ragini Agarwal	Dr Achla Batra Dr Anjila Aneja Dr Urvashi Miglani
2:30-2:45	The robot in Gynecology	Dr Rooma Sinha	
2:45-3:00	Sentinal Lymph Node Biopsy	Dr Rupinder Sikhon	
Session 5 3:00	**Recent advances: Guidelines Practice points /Algorithms**		
3:00-3:10	PE : What's new	Dr Latika Chawla	Dr Sangeeta Gupta Dr Shakuntala Kumar Dr Yukti Wadhawan
3:10-3:20	Preterm Labor	Dr Kamna Datta	
3:10-3:20	Anticoagulation in pregnany	Dr Shehla Jamal	
3:20-3:30	Menopausal Hormone therapy	Dr Monika Madan	
3:30-3:40	PMS	Dr Pakhee Agarwal	
3:40-3:50	Peripartum Mood Disorders	Dr Swati Agarwal	
3:50-4:00	Rational use of IVIg	TBA	
	Discussion		
Valedictory	**4:00-5:00PM**		

29th September 2019 (Day 2) Hall 2

Session 1	Gynaecological Oncology	Speaker	Chairpersons
9:00-9:15	ERAS	Dr Manash Biswas	Dr VL Bhargava Dr Sunesh Kumar Dr Gauri Gandhi Dr Raksha Arora
9:15-9:30	HIPEC in Ovarian Cancer: Standard of Care or experimental approach	Dr Seema Singhal	
9:45-10:00	Current indications of menopausal hormone therapy in cancer survivors	Dr Swasti	
10:00-10:45	Panel on Management of gynaecological cancers in younger women	**Moderator** Dr Shylashree TS	**Panelists** Dr Kanika Gupta Dr Satinder Kaur Dr Sarika Dr Shruti Bhatia Dr Vijay Zutshi Dr Y M Mala Dr Jyoti Meena Dr Nilesh Rohatgi
10:45-11:00	Controversies in Gynaecological Oncology : MIS in Carcinoma Cervix	For: Dr Nikita Trehan Against: Dr Shalini Rajaram	Dr Neerja Bhatla Dr Swaraj Batra
Session 3 11:00-12:10	**Plenary**		
	Brigadier Khanna Oration		
11:05-11:35	Evolution and Advances in Minimally invasive Gynecology : My Journey Through Endoscopy	Dr Alka Kriplani	**Chairpersons** Dr SB Khanna Dr Kamal Buckshee Dr SS Trivedi Dr Sudha Salhan
	Keynote Addresses		
11:35-11:55	Obstetrics Care Through Prism of Quality	Dr Manju Puri	**Chairpersons** Dr Urmil Sharma Dr Ranjana Sharma Dr Sanjeevni Khanna Dr Neerja Goel
11:55-12:15	Medical Education : From Past to Future	Dr Sunesh Kumar	
12:15-1:15	Quiz		
1:15-2:00	Drills and Role Plays Eclampsia Shoulder dystocia	Dr Juhi Bharti Dr Jyoti Meena Dr Neha Gupta Dr Anubhuti	
Session 4	**Safe abortion Practices**		
2:00-2:15	Slogan Competition		Dr Anita Rajohria Dr Sonal Bathla
2:15-3:00		**Moderator**	**Panelists**
	Consortium on safe abortion Panel Discussion	Dr Suneeta Mittal	Dr Sumita Ghosh Dr Sangeeta Batra Dr Priya Karna Dr Rakhi Singh Dr Sunita Malik Dr Nirmala Agarwal
Session 5 3:00-4:30	**Video Session**	**Presenters**	**Moderators**
3:00-3:10	Robotic sacrohysteropexy	Dr Dinesh Kansal	Dr KK Roy Dr Malvika Sabharwal Dr Bijoy Nayak
3:10-3:20	Difficult TLH	Dr BB Dash	
3:20-3:30	Uterine reconstruction post myomectomy & adenomyomectomy	Dr Puneeta Bhardwaj	
3:30-3.40	Endometriosis	Dr Neema Sharma	
3:40-3:50	TBA	Dr Shivani Sabharwal	
3:50-4:00	Difficult Vaginal hysterectomy	Dr Rajesh Kumari	
Valedictory	**4:00-4:30 pm**		

41th Annual Conference of AOGD 2019

Date: **28th - 29th September 2019**

Venue: **Eros Hotel, Nehru Place, New Delhi**

Organised by:

Department of Obstetrics and Gynaecology AIIMS, New Delhi

Theme:

Enlightening the path for the Next Generation

Pre-Conference Workshops

26th September 2019

Ist Trimester USG - Quality Control
Dr Manisha Kumar (LHMC)
ME Hall, SJ Auditorium, LHMC

Urogynaecology
Dr J B Sharma (AIIMS)
Gynae Seminar Room (3rd Floor)

Ovulation Induction and IUI
Dr Surveen Ghumman (Max Hospital)
Auditorium, Wels Block, Max Superspeciality Hospital, Saket

Preventive Oncology
Dr Savita Samsunder (SJH)
& Dr Susheela Gupta (Fortis Hospital)
Old LT1, behind New OPD Building, Safdarjung Hospital, New Delhi

27th September 2019

Endometriosis (video workshop)
Dr Kuldeep Jain (KJIVF Centre)
India Habitat Centre, New Delhi

Obstetric Skills
Dr Reva Tripathi (HIMSR)
Medical Education Hall

Endoscopy
Dr Richa Sharma (GTB Hospital)
Library Hall, UCMS-GTBH, Delhi

Saving Mothers
Dr Mala Srivastava (SGRH)

Medico-legal aspects in Obs & Gynae
Dr Asmita (MAMC)
New Lecture Theatre, Ground Floor, Maulana Azad Medical College, New Delhi

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41st Annual Conference of Association of Obstetricians and Gynecologists of Delhi

28th - 29th September, 2019

Venue: **Eros Hotel, Nehru Place, New Delhi**

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Speciality .....

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1. High Risk Pregnancy & Fetal Medicine ( )
2. Cutting Edge technology in Obstetrics and Gynaecology ( )
3. Preventive Oncology ( )
4. Miscellaneous ( )

Guidelines for abstract submission on aogd.org

Last date for Abstract Submission for Free Communication and Poster: 15th August 2019

Preconference workshops (Tick any one)

26th September 2019

1. Ist Trimester USG - Quality Control ( )
2. Urogynaecology ( )
3. Ovulation induction and IUI ( )
4. Preventive Oncology ( )

27th September 2019

5. Endometriosis (video workshop) ( )
6. Obstetric skills ( )
7. Endoscopy ( )
8. Saving mothers ( )
9. Medico-legal aspects in Obs & Gynae ( )

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Management of Fibroids

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Fibroids are the most common pelvic tumor. They are monoclonal tumors arising from smooth muscle cells of the myometrium. It is estimated that 60% of reproductive-aged women are affected, and 80% develop the disease during their lifetime.[1] 80% of fibroids remain asymptomatic and are incidentally diagnosed. The presenting symptoms may be described into three main categories:

- Menstrual complaints: heavy menstrual bleeding, dysmenorrhoea
- Pressure symptoms: pelvic pressure and pain
- Infertility

Asymptomatic fibroid usually does not require any treatment and regular follow-up may suffice. Exceptions include a significant submucosal leiomyoma in women planning conception and in women with ureteral compression leading to moderate to severe hydronephrosis. Such women may benefit from prophylactic treatment, which may prevent miscarriage or urinary tract obstruction. Lack of predictors for progression of myoma limit prophylactic therapy. Intervention depends upon following factors:

- Type and severity of symptoms
- Size of myoma
- Location of myoma
- Patient age
- Reproductive plans and obstetrical history

This article defines the most commonly used medical therapy in management of symptomatic fibroids.

Medical Management of Fibroids

Medical therapy may prove symptom relief in some women, where bleeding is the predominant symptom. 75% of women get improvement over one year but have a high failure rate.[2] In general, among women receiving medical therapy trial almost 60% required surgery[3].

- Indications of medical therapy include:
- Peri-menopausal women
- Mild symptoms
- Mildly enlarged uterus
- Unfit or not willing for surgery

Medical management may be helpful in patients where the primary symptoms are due to condition associated with leiomyoma such as anovulation and not per say because of fibroid.

Hormonal Therapy

Hormonal therapy benefits women with predominant menstrual symptoms. Bulk symptoms may not be relieved effectively with Progesterones or levonorgesterol intrauterine device.

Levonergestrol Intrauterine device (LNG IUD)

It is widely used to treat menorrhagia related to fibroids and has been FDA approved. Observational studies and systemic reviews show decrease in heavy menstrual bleeding and improvement in anaemia with the use of LNG-IUD[4,5]. The improvement in hematocrit and quality of life has been demonstrated with use of LNG IUS, however there was no change in size of fibroid. It may also be useful as a contraceptive method in women not desirous of conception.

Once inserted may be used for upto 5 years. Its local action decreases the systemic side effects and has a better compliance. An intracavitary leiomyoma is a relative contraindication to insertion of LNG-IUD because of high expulsion rate.

Progesterone (Oral, Injection, Implants)

Progesterones cause endometrial atrophy causing reduced blood loss and increased hemtocrit. Progesterone use in cohort study has also been shown to reduce the formation of leiomyoma[6].

Progesterone Receptor Modulator

Fibroids express more oestrogen and progesterone receptors than the background myometrium. Fibroids are known to increase in size in secretory phase under action of progesterone. Progesterone increases mitotic activity and cellularity in the fibroid. Progesterone antagonist may therefore limit the growth of fibroids. Progesterone receptor modulator are increasingly being used as the first line in treatment of fibroids. A meta-analysis comparing use of progesterone receptor modulator with or gonadotropin receptor modulator revealed a beneficial response to use of Progesterone receptor modulator in terms of menstrual bleeding and improvement in quality of life.

Ullipristal acetate- Most commonly used selective progesterone receptor modulator (SPRM). It is a synthetic steroid from 19-norprogesterone. It is tissue specific and binds to uterus, cervix, ovaries and hypothalamus. It is used in a dose of 5 mg or 10 mg effectively to induce amenorrhea within 10 days of initiation of treatment. The major benefit of ullipristal acetate over GnRH agonists is that it induces amenorrhea almost 2 weeks earlier with a lesser risk of bone loss.

There have been concerns regarding the use of ullipristal acetate with liver toxicity. Liver toxicity has been reported to an extent of liver transplant.[7] This limits the rampant use of SPRM. It is recommended to have a liver function test before the onset of therapy and after initiation of treatment. Since the effect on endometrium is unknown, the drug is usually given cyclically with intermittent menstrual shedding.

Mifepristone is the most studied progesterone antagonist. It is a synthetic 19-norsteroid progesterone antagonist. It exhibits inhibitory effect on growth of fibroid. It reduces uterine volume by 26 to 74 percent[8] Mifepristone has been shown to reduce menstrual blood flow and improve quality of life. However the size of fibroid may increase after cessation of treatment.[9] There have been side effects of transient elevation of serum aminotransferases.

A unique pattern of endometrial changes has been observed termed as c-associated endometrial changes. The characteristic findings are dilated weakly secretory endometrial glands with few mitotic figures, and stroll effect ranging from compaction to uniform edema, termed as progesterone receptor modulator associated endometrial changes (PAECs). PAECs occurs nearly in 50% of the patients. True endometrial hyperplasia and atypical hyperplasia have not been observed, however no woman has developed endometrial carcinoma [10]. These endometrial changes are reversible 1-2 months after cessation of treatment.

Gonadotropin-releasing hormone agonists-

Most effective treatment for medical management of fibroid. It is FDA approved in preoperative management of fibroids. Initially causes flare up of response followed by downregulation. They are effective in reducing menstrual blood loss, decreasing uterine volume, and reducing intra-operative blood loss. However on stopping the treatment there is rapid resumption in symptom. Also, Long term treatment may cause hypoestrogenic symptoms, which may require add back therapy after long term treatment.

These side-effects limits long term use of gonadotropin- releasing hormone agonist. It may be used preoperatively in women:

- Who wish to postpone surgery or are surgically unfit
- Decreasing menstrual blood loss and improvement of anemia
- Reducing size of leiomyoma to facilitate minimally invasive surgery
- To reduce intraoperative blood loss by reducing vascularity of the fibroid

Gonadotropin- releasing hormone antagonist-

They are equally effective when compared to GnRH agonist, may however be beneficial in their rapid onset of action and lack of initial flare-up response. They are likely to be well tolerated and with less severe side effects. Elagolix has recently been approved by the FDA.

Antifibrinolytic agents-

They are useful in the treatment of heavy menstrual bleeding. Tranexemic acids widely used and is FDA approved for treatment of heavy menstrual bleeding.

Nonsteroidal anti-inflammatory drugs

There use is limited to decreasing dysmenorrhoea and has not been associated with decreased blood loss.

Androgenic steroids

Androgenic steroids like danazol may be effective in treatment, but with frequent side effects.

Danazol is a 19-nortestosterone derivative with androgenic effects. Since it induces amenorrhoea it reduces the blood loss related to leiomyoma but not the uterine size. Side effects include weight gain, muscle cramps, decreased breast size, acne, hot flashes, acne, hirsutism.

Surgical Management

The definitive management of fibroid is surgical.

Hysterectomy

In women who have completed childbearing, hysterectomy is indicated as a permanent solution for symptomatic fibroids. The only indication of a completely asymptomatic fibroid is an enlarging fibroid after menopause. The choice and type of hysterectomy, whether it is performed by abdominal, laparoscopic or vaginally is based on expertise, experience and surgeons training. The least invasive approach feasible should be used.

Myomectomy

It is an alternative to hysterectomy for women who wish to retain their uterus. Intraoperatively there is a higher chance of blood loss and greater operative time. The route of myomectoy may be abdominal, laproscopic, robotic. Hysteroscopic myomectomy may be chosen in submucosal fibroid projecting into the uterine cavity.

Women should be counselled about the risks of requiring hysterectomy at the time of myomectomy. Fibroids are associated with a recurrence rate of 15% and 10% of women undergoing a myomectomy will eventually require hysterectomy within 5 to 10 years.

Conclusion

To conclude, medical therapy for fibroid may be temporary and symptoms may re-appear after the therapy has been stopped. Upto 60% of patients on medical therapy may require surgery within 2 years[3]. Never-the less medical therapy may be beneficial in women with menstrual complaints incorrectly attributed to fibroid which may be present due to coexisting pathologies like anovulation.

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Endometriosis

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Endometriosis is defined as presence of endometrial glands and stroma outside the uterine cavity, first described by Rokitansky in 1860. It affects about 7-12% of women in reproductive age group. The incidence is high in patients suffering from infertility about 25-35%. It is also high in patients with chronic pelvic pain (30-55%). There are varied symptoms of the disease. The classical triad for endometriosis is dysmenorrhoea, dyspareunia and sub-fertility. Other symptoms are heavy menstrual bleeding, dysuria, dyschezia, abdominal pain, chronic pelvic pain. It may involve bladder, rectum, gastro-intestinal tract leading to haematuria, hematochezia and constipation.

The most common site of involvement is ovary, utero-sacral ligaments, pouch of Douglas, pelvic peritoneum, tubes, recto-vaginal septum, posterior surface of uterus. The rare sites of involvement are pulmonary, sub-diaghramatic area, paracolic gutters, scar site of episiotomy, hysterotomy, cesarean section.

There are various theories for etiology of endometriosis:

1. Sampson's theory of retrograde menstruation
2. Celomic metaplasia theory
3. Stem cell theory
4. Lymphatic and vascular spread theory
5. Genetic theory
6. Immunological theory
7. Hormonal and inflammation theory

Diagnosis is mainly by strong clinical suspicion based on symptoms as there is typical history of progressive dysmenorrhoea where the pain increases in duration, severity gradually becoming chronic pelvic pain. Physical examination has poor sensitivity, specificity, and predictive value in the diagnosis of endometriosis. Clinical examination may reveal tenderness in fornices, adnexal mass in presence of chocolate cyst, restricted mobility of uterus and thickening of recto-vaginal septum, nodularity in the posterior vaginal fornix, and visible vaginal endometriotic lesions. Imaging modalities include ultrasound, in which mainly transvaginal scan is helpful. Transrectal USG also is useful when transvaginal cannot be performed and to detect recto-vaginal endometriosis. USG will detect ovarian endometrioma, haematosalpinx, where it will show the homogenous ground-glass appearance of the endometrioma. Hydronephrosis secondary to ureteric endometriosis may be detected by transabdominal USG. Minimal and mild endometriosis is difficult to be diagnosed on ultrasound. MRI is said to be better for diagnosis of moderate to severe and deep endometriosis. It should not be ordered as primary investigation for diagnosis of endometriosis.

Role of serum bio-marker CA-125 is controversial. It may be high (>35mIU/ml) suggesting the presence of endometriosis, its rupture but normal value of CA-125 does not exclude endometriosis.

Laparoscopy is the gold standard in diagnosis and management of endometriosis. It is both diagnostic and therapeutic. Visual inspection of endometriotic spots on laparoscopy is also not confirmatory. It has to be proven histologically by presence of glands and stroma both but negative biopsy does not rule out endometriosis. The endometriotic patches may appear as red, pink, bluish-purple, velvety lesions or white powder burnt patches. One should be aware of different appearances of endometriotic spots so as to identify them all and properly treat them in the same sitting of surgery. DIE (deep infiltrating endometriosis) may be missed even at laparoscopy and if diagnosed, it requires expertise to remove it.

When endometriosis is diagnosed, the gynaecologist should document a detailed description of the appearance and site of endometriosis. The staging should be done as per ASRM/ESHRE or revised AFS classification given in 1997. Recent classification is ENZIAN which takes into consideration DIE and Endometriosis Fertility Index (EFI).

Endometriosis causes infertility due to immunological, ovulatory dysfunction, alteration in endometrial receptivity and tubal factors in severe cases. Endometriosis causes ovulatory infertility by altering folliculogenesis and ovulation due to inflammation associated with endometriosis. Endometriosis causes immunological infertility due to increased production of ROS by macrophages and poly morphonuclear cells associated with endometriosis which causes increased oxidative stress. Decreased expression of integrins and increased production of cytokines are noted. Endometriosis causes decreased sperm quality and function due to inflammatory toxic effects of the peritoneal fluid and activated macrophages upon the sperms. Endometriosis affects endometrial receptivity by causing progesterone resistance, dysregulation of progesterone receptors and by increased E2 production

secondary to elevated aromatase enzymes.

Endometriosis is a chronic, recurrent, progressive disorder which affects the quality of life rather than decreasing the survival. Management options are both medical and surgical. It is based mainly on symptoms, patient's age and desire for fertility. Medical management is mainly for patients suffering from pain, dysmenorrhoea, dysuria. It is also used for prevention and treatment of recurrence and if patient refuses surgery. But if the patient has main complain of infertility, then one has to go for surgical management.

Medical Management:

There are many groups of drugs being used for medical management of endometriosis. These are described below:

a). Non-steroidal anti-inflammatory drugs (NSAIDs): The pain pathogenesis is through prostaglandin pathway so COX I and COX 2 inhibitors are first line therapy in endometriosis associated pelvic pain and dysmenorrhoea. Mefenamic acid and ibuprofen are most commonly used and they are effective in almost 50-60% cases when given thrice daily.

b). Combined oral contraceptive pills: These are mainly used in women who are not trying for conception. They suppress endogenous release of gonadotropins, reduce menstrual flow and progesterone component decidualizes endometriotic implants. They can be used as continuous or cyclic regimen. Continuous regimen for 6 months is more effective in controlling pain and dysmenorrhoea in about 40-50%.

c). Progestins: They cause atrophy of the endometriotic implants and pseudo-pregnancy state. It can be given for 3-6 months continuously and leads to 60% reduction in pain. Various preparations are used such as medroxyprogesterone acetate in 20-80mg daily dose, norethisterone 10-20mg daily, Injection Depot medroxyprogesterone acetate 150mg every 3 months for 6-9 months, dienogest 2mg daily for 6-9 months. Dienogest is a fourth generation synthetic progesterone which has recently been proposed as treatment of choice for this condition. Long term progesterone delivery system in the form of LNG-IUS (levonorgestrel intrauterine system) is also beneficial in these patients as amenorrhoea is achieved in 88-92% of patients after 9-12 months. It delivers 20mcg of progesterone daily for five years. It has less systemic side effects and more effective in causing local atrophy of endometrium.

d). GnRH agonists: they cause pituitary desensitization and thereby leading to inhibition of ovarian steroidogenesis. They lead to pseudomenopause and also called medical oophorectomy. Common preparations available are leuprolide acetate 3,75 mg, triptorelin 3.75mg, goserelin 3.6mg. They are given as monthly injections for 6 months. If one has to give it for longer duration than 6 months then add-back therapy is used to prevent hypoestrogenic side effects and decrease in bone mineral density.

Add-back therapy includes conjugated equine estrogen (0.3 to 0.625mg) combined with norethisterone acetate (2.5mg-5mg).

e) GnRH antagonist: cetrorelix in dose of 0.25mg daily or weekly dose of 3mg for 3 months can also be used.

f). Aromatase inhibitors: these agents lead to hypoestrogenism which is responsible for suppression of endometriotic implants. Anastrozole (2mg) or letrozole (2.5mg) is use for 6 months continuously.

g). Selective progesterone receptor modulator (SPRM): they bind to progesterone receptors and exert varying effects on different tissues. Ulipristal acetate and mifepristone are used for this condition for 3-6 months.

h). Gestrinone: It is 19-nortestosterone derivative having anti-estrogenic and anti-progestin activity. It is used in the dose of 2.5 mg twice weekly for 6 months. Danazol was also used for endometriosis in dose of 400-800mg daily doses but it is not used as it has got many androgenic side effects and has gone into disrepute.

i). Others: TNF-alpha inhibitors, MMP (matrix metalloproteinase) - inhibitors, pentoxifylline, raloxifene etc are experimental.

Surgical Management:

Laparoscopic ablation or excision and adhesiolysis improves pregnancy rate in stage I and II endometriosis when compared to diagnostic laparoscopy alone. Operative laparoscopy in stage III and IV endometriosis has shown to improve pregnancy rates as compared to expectant management. It restores the anatomy of tubes and ovaries and also decreases the inflammatory milieu within the peritoneum and uterus for better implantation rates. When endometrioma or chocolate cyst is present, then cystectomy is preferred to drainage and fulguration as it improves pregnancy rate and also associated with reduced recurrence rates. Cyst wall should be removed completely and cautery should be done to the base of the cyst.

Surgery can also be done by laparotomy as well as laparoscopically. If the patient's age is advanced and she has completed her family with no desire to retain uterus, then hysterectomy with bilateral salpingo-oophorectomy can be offered to woman. Laparoscopic uterine nerve ablation (LUNA) is also an option for endometriosis-associated pain but it has very low efficacy (30-40% only). In DIE, one has to go for radical surgical excision of all deep seated endometriotic lesion with extensive bowel and ureteric dissection. Pre-sacral neurectomy can also be done for transection of presacral nerves but it is obsolete in current practice.

Treatment of Infertility associated with endometriosis: Laparoscopy with treatment of the endometriotic lesions is must. This is followed by ovulation induction and intrauterine insemination in stage I and II diseases. While stage III and stage IV disease patients should undergo ART (assisted reproductive technique) with IVF-ET (in-vitro fertilisation- Embryo transfer) or ICSI (intracytoplasmic sperm injection).

Recurrence: Endometriosis is one disease which is known for high rates of recurrence. The disease recurs in almost 20% of patients in 2 years and 40% recurrence is seen after 5 years. There is high morbidity and surgical complications are also more in recurrent cases. Resistant and repeated cases ultimately require hysterectomy and bilateral salpingo-oophorectomy.

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Calendar of Monthly Clinical Meetings 2019-20

Months	Name of the Institute
25th October, 2019	ESI Hospital
29th November, 2019	MAMC & LN Hospital
27th December, 2019	Sir Ganga Ram Hospital
31st January, 2020	Dr RML Hospital
28th February, 2020	UCMS & GTB Hospital
27th March, 2020	LHMC
24th April, 2020	Apollo Hospital

AOGD Quiz

Entries are invited for participation in the
Quiz on

"General Gynaecology"

For participation send your details at aogdabstract2019@gmail.com.
Preliminary round will be held at Gynae Seminar Room
on Friday, 27th September, 2019 at 3:00pm.

Walkathon

Walk an Safe Abortion on 29th September, 2019 to assemble at Eros Hotel at 6:30am.

Forthcoming Events

Next Monthly Clinical Meeting on 25th October, 2019 (4:00-5:00 pm) at ESI Hospital.

Evidence Based Practice: An overview

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Evidence based Practice (EBP) combines individual clinical expertise with best available research evidence to extend high standard of care to patients.

Best evidence is the unbiased view of truth. Bias is any systematic error that can produce a misleading difference between study results and truth. It is a challenge to get unbiased true results. Well designed and executed studies largely provide the unbiased answers. Evidence, such as those obtained by personal experience are also useful but are not included under "evidence-based medicine."

EBP in Indian context

- There is a vast variation of practice between hospitals, individual units/doctors even within one hospital. Ensuring uniform standards of care for patients is a challenge.
- Aggressive marketing and industry-driven treatments with the attendant impact on treatment practices & costs is often observed
- There is increasing patient demand with easy access to information. One is expected to answer their queries and offer the latest evidence on the proposed therapy as well as alternative options.

Advantages of EBM

1. Optimizes the quality of health care
2. Supports uniform standard of care
3. Minimizes risky clinical practice
4. Makes it easier to audit practice
5. Ensures medicolegally safe practice

Steps in Evidence Based Practice (Fig 1)

Fig 1: Evidence Based Practice

Step 1: The search for the best answer to a clinical query begins with a tight definition of the question. *The clinical question (PICO) should reflect the following*

- Which individual or group of patients(P) is being studied?
- What drug/surgical/other intervention (I) is under consideration?
- What are the alternative interventions available or compared (C)with?
- What is the result/outcome(O) of intervention that is being studied

Step 2: *Search for best quality evidence*.

Of the various sources available, not all are up-to-date and reliable. A good source should give us the systematic reviews available on the topic. It should be easily accessible, comprehensible and clinically relevant. In the Pyramid of Evidence (Fig 2), Systematic review occupies the top position. Randomised controlled trials are next only to systematic review.

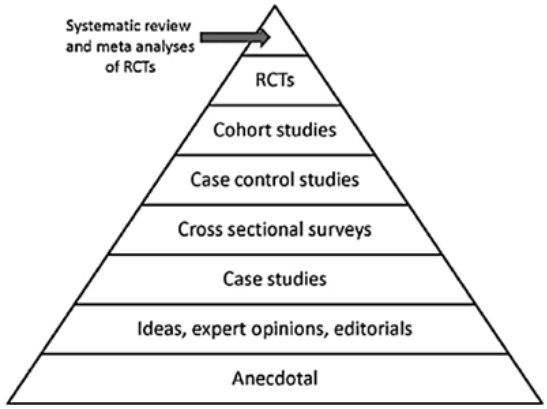

Fig 2. Pyramid of Evidence

Textbooks take 3-5years to be published and hence may be out of date. However, they are good source of information for basic questions on anatomy and a good source to understand pathophysiology of a condition; however, the may not give the best latest advice regarding management of the same.

Journals—peer-reviewed journals with good quality research papers are better than those featuring descriptive or expert reviews. Some journals (with good impact factor) only consider good quality Randomised Controlled Trials (RCTs) for publication as evidence as they are considered the gold standard for evidence. However, today, one needs to be aware of the Predatory Journals with substandard publications.

Colleagues & Senior Faculty—it is common to discuss and seek answers; however, they are not always accurate and sometimes harmful. In fact, some clinicians may have very good bedside manners, appear confident in clinical judgement and technical skills, but this does not guarantee that their approach has evidence base or has been appraised.

In the hierarchy of evidence, statements by the 'medical expert' are considered the least valid form of evidence. All experts are now expected to reference their statements to scientific studies

Guidelines—excellent evidence-based guidelines are available from institutions like Royal College of Obstetricians and Gynaecologists (RCOG-UK), National Institute for Clinical Excellence (NICE-UK). However, it is incorrect and indeed unfair to translate the same to Indian scenario. There is a dearth of good quality evidence-based guidelines in India.

Internet search—this is the quickest, and effective method to search for evidence in trained hands. The clinical question that one has framed is typed as key search words into search engine and one is presented with huge volume of relevant literature within a fraction of a seconds.

However, literature search is an art which can be mastered through training. The web is full of information. Accessing the right database using the appropriate key word results in successful access to relevant and credible results within few minutes.

Popular search databases or websites include

- PubMed (www.ncbi.nlm.nih.gov/pubmed)
- Ovid (ovidsp.ovid.com)
- Cochrane (www.cochrane.org or www.thecochranelibrary.com)
- CDC (www.cdc.gov)
- WHO (www.who.int)
- NHS Evidence (www. evidence.nhs.uk)
- Google scholar (scholar.google.com)
- Web of Science/Knowledge (wok.mimas.ac.uk)
- RCOG (www.rcog.org.uk/guidelines)
- ACOG

Step 3 It is important *critically appraise the evidence* in terms of its validity and applicability to the chosen population. Critical appraisal skills can help clinicians choose more wisely which information they use, favouring the sources with explicit standards.

The focus of critical appraisal is judging both internal validity and generalizability (external validity). Biases (Fig 3) creep in sometimes unknowingly and is reflected on detailed review of the publication.

Internal validity — Answers the question whether the results of clinical research are correct for the patients studied. Threats to internal validity include bias and chance (random error) inherent in all observations. The probability of chance producing erroneous results can be minimized by having a large sample size for study. Sound idea of bias is required to verify the results of study. Brief details of types of bias are listed in Fig 3.

External validity — External validity refers to the question of whether the results of the study apply to patients outside of the study, particularly the specific patient (or population) Study patients are typically highly selected, unlike patients in usual practice. Often, they have been referred to academic medical centres, meet stringent inclusion criteria, are free of potentially confounding conditions or disorders, and are willing to adhere to the rigorous demands of study protocols. As a result, they may be systematically different from the patients most doctors see in practice.

Fig 3 : Types of Bias[1]

Type of bias	Description
Selection bias.	Systematic difference between baseline characteristics of the groups that are compared.
Performance bias.	Systematic differences between groups in the care that is provided, or in exposure to factors other than the interventions of interest.
Detection bias.	Systematic differences between groups in how outcomes are determined.
Attrition bias.	Systematic differences between groups in withdrawals from a study.
Reporting bias.	Systematic differences between reported and unreported findings.
Publication bias	Systematic error due to non - publication of studies with negative or neutral findings

A set of guidelines endorsed by the *International Committee of Medical Journal Editors* can facilitate the critical appraisal of individual studies based on the type of study:

Study Type	Appraisal Checklist
Systematic reviews and meta-analyses	Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) and PRISMA Protocols (PRISMA-P)
Randomized controlled trials	Consolidated Standards of Reporting Trials (CONSORT) and Standard Protocol Items: Recommendations for Interventional Trials (SPIRIT)
Observational studies	Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)
Diagnostic and prognostic studies	Standards for Reporting of Diagnostic Accuracy (STARD) and Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis (TRIPOD)

Step 4 *Applying the evidence in practice* — There is often a gap between recommendations from the best available evidence and actual practice. The reasons for the gap are numerous, including uncertainty whether results of large studies apply to individual patients, lack of awareness or misunderstanding of the evidence, and failure to organize care in a way that facilitates use of evidence. The access to evidence/guidelines is another factor for lack of its usage. Contextual evidence needs to be freely available and shared.

Step 5 *Assess the impact, Audit*

Auditing the clinical practice ensures the standard of care and also helps to evaluate the impact of the new/ altered management strategy. This is an important step in evidence based healthcare practice and requires to be built into the system

Grading and levels of evidence by the professional bodies is done after going through all these steps. Finally, after collation and critical appraisal, the robustness of evidence is identified and incorporated in guidelines. Hence, training and skill to practice all the stated steps is extremely important not only in one's clinical practice but also for the experts involved in formulation of guidelines & recommendations. In India, there has been a sudden increase in publication of guidelines in the past few years. In a study conducted by the author (under publication) the quality of the guidelines was found to be poor in terms of evidence base, transparency with respect to conflict of interest and there was no updating or revision plan in any of the guidelines.

Important Points

- The volume of evidence available to guide clinical decisions continues to grow at a rapid pace due to improvements in understanding of research design.
- Despite the advances in research methods, quite a few published study results are false or draw misleading conclusions
- Newer methods for analysing data have led to a better understanding of how to produce valid clinical research.
- Many clinicians, even those in good standing, do not practice medicine according to the best current research evidence.
- Behaviour change usually requires a combination of interventions and influences, including time for rethinking practice habits.

Individuals/institutions interested to have training on any/all aspects of EBP may feel free to contact the author.

References

1. https://handbook-5-1.cochrane.org/chapter_8/table_8_4_a_a_common_classification_scheme_for_bias.htm

Answer: August Issue

Crossword

Horizontal clues:

1. Meckle Gruber
2. Pericallosal
3. Truffle
4. Pluto
5. Digoxin
6. Sliding
7. Warfarin
8. Dandy walker
9. PR
10. MOMs

Vertical clues:

1. MPS
2. Collin's
3. Stippling
4. Binder's

Pictorial Quiz

1. Holoprosencephaly

2.1. Congeital heart block
2.2. SLE/ Sjogren's syndrome

3.1. Radio frequency ablation needle
3.2. Twin reversed arterial perfusion / TTTS / twin to twin transfusion syndrome

SOP: Approach to Pelvic Inflammatory Disease

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Women with lower abdominal pain/pelvic discomfort

History — Look for risk factors.
- Sexual history - assessing for new sexual partners & consistent use of condom.
- Onset and character of pelvic pain.

Speculum examination-
- vaginal discharge
- cervical mucopurulent discharge

P/V examination
- cervical motion, uterine, or adnexal tenderness.
- tubo-ovarian mass

Laboratory tests — Supportive role
- UPT- to rule out pregnancy.
- Microscopy of vaginal discharge – to detect pathogen
- Nucleic acid amplification tests (NAATs) for *C. trachomatis* and *Neisseria gonorrhoeae*)
- HIV screening and Serologic testing for syphilis- both partners
- Cervical discharge gram stain- gram negative intracellular diplococci (suggestive of *N. gonorrhoeae*)

Additional evaluation for diagnostic uncertainty:
- Acutely ill: fever, peritonitis, or a pelvic mass
- Atypical symptoms:(abnormal site or duration of symptoms) **or**
- Do not improve in 72 hrs after empiric antibiotic therapy,
- Persisting pain after completing therapy.

Pelvic Imaging: Ultrasound
- Thickened, fluid-filled fallopian tubes
- Cogwheel sign may be present
- Endometritis: fluid or gas in endometrial canal, heterogeneous thickening, or indistinctness of the endometrial stripe.
- Tubo-ovarian abscess - a complex thick-walled, multilocular cystic collection in adnexa with internal echoes.

Laparoscopy — useful in:
- Failed outpatient treatment for PID
- Not improving or worsening after 72 hours of inpatient T/t, which suggests alternate diagnosis.

Presumptive diagnosis- any of three **minimal criteria** on P/V:
- Cervical motion tenderness
- Uterine tenderness
- Adnexal tenderness

Additional criteria to support clinical diagnosis
- Oral temperature >38.3°C
- Abnormal cervical or vaginal mucopurulent discharge
- Abundant numbers of WBCs on saline microscopy of vaginal secretions
- Cervical infection with *N. gonorrhoeae* or *C. trachomatis*
- *Elevated - C reactive Protein/ESR*

Specific criteria
- Histologic evidence of endometritis in biopsy.
- Pelvic imaging -thickened, fluid-filled tubes/ oviducts with or without free fluid, or tubo-ovarian complex.
- Doppler studies- Tubal hyperemia
- Laparoscopic findings- tubal erythema, edema, adhesions; purulent exudate or cul-de-sac fluid; and abnormal fimbriae.

Differential Diagnosis
- ✓ Ectopic pregnancy
- ✓ Ruptured ovarian cyst/torsion
- ✓ Endometriosis
- ✓ Cystitis
- ✓ Appendicitis
- ✓ Diverticulitis
- ✓ Irritable bowel syndrome
- ✓ Functional pain

Mild to moderately severe acute PID

Outpatient management

Oral therapy

Regimen A
T. Levofloxacin 500 mg OD x14 days

OR
T. Ofloxacin 400 mg OD x14 days **WITH OR WITHOUT** T. Metronidazole 500 mg BD x 14 days.
Levofloxacin is as effective as ofloxacin & may be substituted.

Regimen B
Ceftriaxone 250 mg IM, single dose **PLUS** T. Doxycycline 100 mg BDx14 days **WITH OR WITHOUT** T. Metronidazole 500 mg BDx14 days

OR
Cefoxitin 2 g IM single dose and T. probenecid, 1g administered concurrently in a single dose **PLUS** T. Doxycycline 100 mg BDx14 days **WITH OR WITHOUT** T. Metronidazole 500 mg BDx14 days

OR
Other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime), **PLUS** T. Doxycycline 100 mg BDx14 days **WITH OR WITHOUT** T. Metronidazole BD x14 days.

Indications for hospitalization & parenteral antibiotics:
- Inability to follow or tolerate an outpatient oral medication regimen
- No clinical response to oral antimicrobial therapy
- Severe clinical illness (high fever, nausea, vomiting, severe abdominal pain)
- Complicated PID with pelvic abscess (including TO abscess)
- Surgical emergencies cannot be excluded (eg, appendicitis or ovarian torsion)
- Pregnancy

In patient management

Parenteral Therapy (if no response to oral therapy in 72 hrs can be given either on outpatient or inpatient basis)

Regimen A
Cefotetan 2 g IV BD OR Cefoxitin 2 g IV QID PLUS T. Doxycycline 100 mg orally or IV every 12 hrs. Parenteral therapy may be discontinued 24 hrs after a patient improves clinically. **T. doxycycline 100 mg BDx14 days should be continued**

Regimen B
Clindamycin 900 mg IV every 8 hrs PLUS Gentamicin loading dose IV or IM (2 mg/kg of BW) f/b by maintenance dose (1.5 mg/kg) every 8 hrs.
Continue oral therapy- T. doxycycline 100 mg BD or T. clindamycin 450 mg QIDx14 days after 24 hrs of clinical improvement.

Follow up (within 48 to 72 hours)
- Diagnostic evaluation for complications (eg, pelvic abscesses)
- Alternate diagnoses- appendicitis, ovarian torsion, diverticulitis
- Outpatients warrant hospitalization and parenteral therapy.
- Already on parenteral therapy, re-evaluate therapy for appropriate coverage

Counseling
- **Medical adherence & compliance**
- **Sexual abstinence** until therapy completion
- **Contacts tracing-** Male partner examined & treated-h/o sexual contact in last 60 days prior to the patient's onset of symptoms- Ceftriaxone **(250 mg)** i.m **plus** either T. Azithromycin (1 gram) - single dose or T. Doxycycline (100 mg) BDx7 days

Screening and prevention of STIs — HIV and syphilis.

Suggested reading

1. CDC. Sexually transmitted diseases treatment guidelines. MMWR 2010;59(RR12):1-110.
2. 2018 United Kingdom National Guideline for the Management of Pelvic Inflammatory Disease. BASHH guidelines.
3. Heinonen PK, Leinonen M. Fecundity and morbidity following acute pelvic inflammatory disease treated with doxycycline and metronidazole. Arch Gynecol Obstet. 2003;268:284-8.
4. Sweet RL. Treatment of acute pelvic inflammatory disease. Infect Dis Obstet Gynecol. 2011;2011:561909.

Journal Scan

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Effectiveness of Dienogest in Improving Quality of Life in Asian Women with Endometriosis (ENVISIOeN): Interim results from a prospective cohort study under real life clinical practice

Techatraisak K, Hestiantoro A, Ruey S, Banal-Silao MJ, Kim MR, Seong SJ, Thaufik S, Ahlers C, Shin SY, Lee BS

Objective: Dienogest has been shown to substantially improve endometriosis-associated symptoms such as debilitating chronic pelvic pain, and in turn, health-related quality of life (HRQoL). To date, there is no data on patient-reported outcomes reflecting the real-world practice in Asia where endometriosis is a relevant health, social and economic burden. This non-interventional, multi-center, prospective study aims to investigate the influence of dienogest on HRQoL.

Materials and Methods: Asian women received dienogest (2mg/daily) and were followed for 24months. The effectiveness of dienogest to improve HRQoL and endometriosis-associated pelvic pain (EAPP) was assessed by patient-reported outcomes. HRQoL, especially the "pain" domain as primary endpoint, was evaluated with the Endometriosis Health Profile-30 (EHP-30) questionnaire. The numeric rating scale served to determine changes in the severity of EAPP. Within the presented interim analysis (data cut-off: 2017-11-27), the mean changes in EHP-30 and EAPP scores from baseline to 6months upon availability of the data were evaluated. Treatment-emergent adverse events (TEAEs) and bleeding profiles were documented.

Results: Dienogest therapy decreased EHP-30 scores in all assessed domains (score 0–100, lower scores indicate better HRQoL). Primarily, the "pain" domain was improved in 78.4% of patients. EAPP was reduced (score 0–10, lower scores reflect less pain), highlighted by a mean reduction of the pain score by −4.5 points. Patients with a higher EAPP score at baseline had an increased response to dienogest (−6.2 points mean change) compared to patients with low baseline EAPP severity (−1.4 points mean change). Both surgically and clinically diagnosed patients described comparable pain reduction, as well as women with or without prior treatment. Drug-related TEAEs were documented for 31.5% of patients, with amenorrhoea (5.9%) and metrorrhagia (5.1%) being the most common events. The bleeding pattern was changed upon dienogest, characterized by decreased normal bleeding (84.2 to 28.8%) and increased amenorrhea (3.2 to 42.9%) at 6months.

Conclusion: The data indicates an amelioration of HRQoL and EAPP upon dienogest therapy. No new safety signals were observed. Therefore, its use as first-line therapy for long-term management of debilitating and chronic endometriosis-associated pain represents an interesting option that remains to be further investigated.

Editor's Comment: Endometriosis is a chronic disease that affects approximately 10% of all women in reproductive age and up to 50% of infertile women. It is characterized by a progressive course with worsened symptoms if no appropriate therapy is applied. Symptoms mainly include chronic pelvic pain, dyspareunia, dysmenorrhoea and infertility. Quality of life studies reveal that these symptoms can influence several aspects of woman's life such as work, education, relationship, social support, especially with worsening symptoms. The impact of the disease on psychosocial parameters can lead to a significant reduction in health-related quality of life (HRQoL), thus, effective treatment of chronic pelvic pain is essential. Dienogest is an oral progestin with short half life and high oral bioavailability offering unique pharmacologic benefits in significantly reducing the size of endometriotic lesions. The interim analysis evaluated the pain domain of HRQoL as primary goal, as well as safety of drug as secondary goals after 6 months of follow up. Dienogest, 2mg once daily, demostrated not only a reduction in endometriosis associated pelvic pain comparable with GnRH agonists but also improved other psychosocial parameters like emotional well being, self-image, relationship with children, dyspareunia etc. With no new safety signals and a good satisfaction and compliance rate, dienogest may be an interesting option in Asian women for long term management of endometriosis.

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The Impact of Hormonal Replacement Treatment in Postmenopausal Women with Uterine Fibroids: A State-of-the-Art Review of the Literature

Moro E, Degli Esposti E, Borghese G, Manzara F, Zanello M, Raimondo D, Gava G, Arena A, Casadio P, Meriggiola MC, Seracchioli R

Leiomyomas are the most common benign uterine tumours with an unclear etiology. With menopause, the size of leiomyoma shrinks easing the burden on the symptomatic woman who often require no treatment. This state-of-the-art review aims to clarify the influence of different kinds of HRT on fibroid growth and fibroid-related symptoms after menopause, verifying whether a specific treatment method could be more appropriate in this instance.

Objectives: Hormonal replacement therapy (HRT) is effective in treating many debilitating symptoms of menopause. However, its use in women with uterine fibroids is widely debated, based on the susceptibility of these tumours to sexual steroids. This review aims to ascertain the effects of HRT on leiomyomas development and growth in postmenopausal women.

Materials and Methods: Electronic databases (i.e., MEDLINE, Scopus, ClinicalTrials.gov, EMBASE, Sciencedirect, the Cochrane Library at the CENTRAL Register of Controlled Trials, Scielo) were searched from January 1990 until May 2019. All English-written studies evaluating the impact of various HRT regimens on uterine leiomyomas were selected.

Results: Seventeen papers, considering a total of 1122 participants, were included. Fifteen of these were prospective trials, of which nine were randomized controlled trials. The remaining two works were a retrospective observational trial and a retrospective case series respectively. Five studies evaluated the effects of tibolone, also comparing it with various estrogen/progestin combinations, while two were about raloxifene. Thirteen studies compared different combinations of estrogens/progestins, the most common being transdermal estrogens (used in nine studies) and medroxyprogesterone acetate at different doses of 10mg,5mg and 2.5 mg (used in 10 studies).

Conclusion: For women with uterine fibroids, the choice of the most appropriate HRT regimen is crucial to avoid leiomyomas growth and the symptoms possibly related to it. Available data are conflicting, but suggest that uterine fibroids might be influenced by HRT, without representing an absolute contraindication to hormonal replacement therapy. Women with uterine fibroids subjected to HRT should be periodically examined and hormonal treatment should be discontinued if leiomyomas appear to increase in size. Moreover, the minimal effective dose of progestin should be employed.

Editor's Comments: Leiomyomas affect upto 80% of reproductive age women and cause morbidity in up to 30% of them. These are hormone dependent masses with their growth being influenced by the endogenous ovarian steroidogenesis and exogenous administration of estrogen and progesterones. This is because of an increased expression of estrogen and progesterone receptors on the fibromatous tissue as compared to the normal myometrium. Based on this, the use of hormone replacement therapy in post menopausal women has been widely debated and they have been considered as a relative contraindication to prescription of HRT. Specific therapy is only reserved for a subgroup of women with larger fibroids which continue to grow even after menopause and this group is represented by total hysterectomy. Given the unevenness of available data, it could be argued that uterine myomas might be influenced by HRT, without representing an absolute contraindication to treatment. Indeed, several authors encourage the use of HRT in postmenopausal women with fibroids. However, these patients should be regularly subjected to a thorough follow-up, including transvaginal ultrasound for the monitoring of myomas size, and HRT should be discontinued if an increase in size of uterine fibroids is documented.

Clinical Proceedings of AOGD Clinical Meeting held at Army Hospital-Research & Referral, New Delhi on 30th August, 2019

Challenging the Frontiers of Uterine Size in Total Laparoscopic Hysterectomies

Anupam Kapur, Shazia Khan

Case

A 47yr old P2L2 lady with previous 02 caesareans, presented with menorrhagia and dysmenorrhea of 4 months duration. Her cycles were regular with passage of clots and flow of 7-8 days with usage of 8-9 pads per day. On examination her BMI was 31 Kg/m^2. A per abdominal examination revealed a healed Pfannensteil incision and an abdominopelvic mass of 26-week size with restricted mobility. A per vaginal examination confirmed the findings. Ultrasound showed multiple intramural and submucosal fibroids with largest being 5X 4 cm and uterine size of 16x15x16cm. Her PAP smear ruled out any intraepithelial lesion. Endometrial biopsy showed Secretory endometrium. Her hematological, biochemical and serological investigations were normal. She was counselled regarding various surgical modalities and about need for morcellation for specimen retrieval if laparoscopic hysterectomy was attempted. She was sensitized about chances of potential occult uterine leiomyosarcoma. Patient chose Minimally invasive surgery and decision was taken to do a laparoscopic hysterectomy for her.

After adequate pre op bowel preparation and consent patient was taken up for Total Laparoscopic Hysterectomy. Patient was placed in lithotomy position, hands by side, parts cleaned and draped, bladder catheterized and Mangeshikar's Uterine manipulator inserted. Meanwhile surface marking of mass was done to enable correct port placement. Pneumperitoneum was created using Veress needle at Palmer's point. Initial intra-abdominal pressure was set at 15mmHg. Abdomen entered at Lee Huang point using a 10mm Endotip trocar and a 30 ^{0} scope. Once adequate pneumoperitoneum was achieved the IAP was reduced to 11mmHg. 02 secondary ports were taken on bilateral lateral abdominal wall at the level of uterotubal insertion. Another 02 ports were taken 8cm above the lower ports on each side. The upper pedicles were tackled using bipolar and scissors.The anterior and posterior sheaths of broad ligament separated. By staying in the loose areolar tissue of broad ligament we opened the retroperitoneal space. The anterior sheath of broad ligament was lifted and cut angling towards the uterovesical fold of peritoneum. The posterior sheath was dissected and cut angling towards the uterosacrals. The uterovesical peritoneum was held with harmonic and lifted and cut. A lateral approach through window of Treitz was done to avoid bladder damage as it was expected that bladder might be adhered anteriorly. Bladder flap was created by cavitatory action of harmonic and carbodissection. The uterines were skeletonized and by using bipolar and scissors dessicated and cut. The ureters fell off laterally. The colpotomy cup was pushed inwards and anterior & posterior colpotomy done using monopolar hook. The specimen was morcellated and removed. The vault was endosutured using 1-0 vicryl and hemostasis checked. The specimen weighed 1.8kg and total surgical time was 110mins.

Discussion

Hysterectomy is the second most common surgical procedure done on women. Evidence supports the opinion that when feasible vaginal hysterectomy is associated with better outcomes & cost effectivity vis a vis TLH. The potential barriers to performing TLH are not absolute contraindications but rather limitation. Uterine size till late was a limitation for attempting TLH for large uteri. However use of a 30 ^{0} scope, a good uterine manipulator, correct port placements with suitable supraumblical entry point and a novel technique like the Switch over method and specimen retrieval through power morcellation can push the boundaries for TLH for large Uteri.

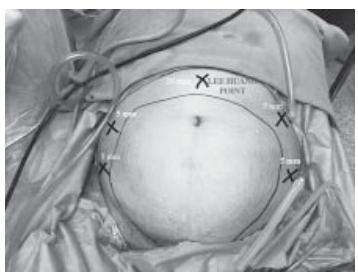

Fig 1: Port Placement

Fig 2 : Morcellated specimen- uterus, cervix and fibroid

Oocyte cryopreservation: A new beginning

Satyabrato Chatterjee

Introduction

Cryopreservation of human oocytes has significantly improved by refined new vitrification techniques. Oocyte cryopreservation techniques would provide would provide a number of benefits. First it would help carrier-oriented ladies as age at which people are marrying is rising. second it would prevent ethical and legal issues associated with embryo cryopreservation in certain countries were embryo cryopreservation is banned. Finally, it gives the option of fertility preservation for patients who are receiving anticancer treatment or oophorectomy.

Case Report

32yrs old w/o serving soldier presented with complaints of inability to conceive she as married for past seven years and cohabitating with her husband for past 3 years. She had regular menstrual periods and her past and family history had no significant positive history. Her vitals were stable and systemic examination was normal. The infertility workup of the wife showed normal ovarian reserve with TVS and AMH of 3.4ng/ml but the husbands semen analysis showed oligoasthenoteratozoospermia (Count: 5 mill/ml

Motility :5%/5%/95% Morphology (Normal forms): 2%). Plan was to put the husband on antioxidant for 2 months but repeat semen analysis showed marginal improvement. Patient was taken up for Long protocol IVF plus ICSI and embryo transfer. The patient was stated on OCPs in month of January and Inj Lupride was started I ml on day 21 of her cycle down regulation was checked on $D_{2\,by}$ E_2 & LH and TVS. The Stimulation was started on 1 Feb 2019. Husband moved on emergency operational duties without informing on 08 Feb 2019. The options available to us was to abandon the cycle or go for Donor semen or Oocyte vitrification. After extensive counselling and discussion with the patient we opted for oocyte vitrification. Ovum pickup was done on 12 Feb 2019. 12 oocytes were retrieved and eight M_2oocytes were vitrified. Husband returned back from his mission in the month of March

Pt was started on frozen embryo transfer on March D2 of her Menstrual cycle (3 /3/19). Patient's endometrium (8.5mm) was ready on 15 March /2019. Semen sample taken the same day and oocytes thawed. 6 oocytes were recovered and ICSI was done. On 18 Mar 2019 three 8 cell stage Grade 1 embryo transferred. Serum B hCG done on 4 Apr 2019 was 2116IU/ml and TVS showed Twin IUGS. The present status is twin pregnancy at 24 POG and anomaly scan normal.

Discussion

In assisted reproductive technology cryopreservation of embryos and oocyte has become important to store supernumerary embryos. During the steps of cryopreservation there is risk of various types of injury among them formation of intracellular ice appears to be most damaging.

Over the past decade there has been several advances in vitrification technologies such that it can provide high clinical efficiency along with better clinical outcome. Although oocyte cryopreservation has a lot of advantages clinical outcomes remain unsatisfactory due to low pregnancy and implantation rates resulting in decreased survival and poor embryo development. The main reasons are oocyte is a single cell survival is judged as all or none. Multicellular embryos can compensate their loss by as much as half of their total cell loss. Membrane permeability of oocyte is another significant reason. In cryobiology it is important to achieve acceptable permeation of cryoprotectants dehydration and rehydration. The types of injuries seen during oocyte cryopreservation are Chilling injuries, Serious deformations during vitrification and thawing and Hardening of zona pellucida. The most important stage in vitrification is exposure of oocytes to vitrification solution before rapid cooling in liquid nitrogen. In order top prevent intracellular ice formation a longer period of exposure is desirable. However, if exposure is too long embryo suffer from toxicity of the solution. Therefore, the optimal exposure time for successful vitrification must be compromise between preventing ice crystal formation and preventing toxic injury.

Conclusion

Since vitrification was introduced as an alternative approach for cryopreservation of human gametes and embryos vitrification with recent improvements has become more reliable strategy because it can lead to high clinical efficiency and better clinical outcomes.

Role of Preconceptional Genetic Counselling in Today's Era – A Case Series of Global Developmental Delay

Reema Kumar Bhatt, Rohin Kumar

Introduction

Over the past few decades genetic testing for inherited diseases has expanded significantly. Human genome project resulted in our increasing knowledge about causative genes and rapid advances in genetic screening technologies such as next generation sequencing made it easier to diagnose these disorders. The advent of rapid next generation sequencing has been transformative for

prenatal diagnosis. Here we present three cases where next generation sequencing helped us to clinch the diagnosis in children being treated and labelled as just global developmental delay or cerebral palsy to enable us to offer prenatal diagnosis for ongoing pregnancy.

Case series

Case 1

A G3P2L2 lady in a non consanguineous marriage with two daughters aged 7 yrs and 9 yrs old both under treatment for global development delay with microcephaly and seizures in one child. The couple was referred at 06 week POG for periconceptional counselling. The case was worked up and tests expediated in consultation with geneticist . An array of tests for index cases were ordered as per the protocol for work up of case of global developmental delay which were normal. On clinical suspicion of creatine deficiency disorder, cerebrospinal fluid was done which showed low levels of creatine for both children . This finding was further augmented by a Magnetic Resonance Spectroscopy which showed absence of creatine peaks suggestive of creatine deficiency disorder. Based on this clinical phenotype a clinical exome sequencing was ordered for both children which revealed both children to be homozygous for Cerebral Creatine deficiency disorder type 2. Both parents were found to be carriers of the same .We were just in time with the diagnosis for prenatal diagnosis, the foetus was found heterozygous for the mutation. The pregnancy was continued ,a healthy baby was delivered who is doing well. The condition is treatable and other two children have also started to show improvement.

Case 2

A G2P1L1 lady was referred from a peripheral hospital 06 week POG for periconceptional counselling, with 5 year old child under follow up for Global developmental delay, Intellectual disability, Motor delay, Seizures and Self mutilation. The index case was evaluated by an array of tests indicated for Global developmental delay which were all normal.. A whole exome sequencing revealed the index case to be homozygous for X linked recessive mental retardation type 96.Prenatal diagnosis was offered and fetus was also found to homozygous for the same mutation. The pregnancy was terminated after counselling the parents. Had this diagnosis not made in time the second child would have been affected by similar condition.

Case 3

A consanguineous couple with 06 yrs old male child under follow up for global developmental delay, severe intellectual disability and mainly cerebral palsy. The couple was referred in pre-conceptional period with limited evaluation of index case. MRI revealed thin corpus callosum with delayed myelination. Other investigations routinely done for global developmental delay in a male child were all normal. A clinical exome, followed by a whole exome sequencing, both of which were normal. However Whole genome sequencing revealed the child to be homozygous for spastic paraplegia type 47. Prenatal diagnosis by Chorionic Villi Sampling was offered at 12 week period of gestation which revealed the fetus to be homozygous for the same gene. The couple was counselled and the pregnancy was terminated.

Conclusion

The aim of presenting these cases was to put forward the importance of prenatal diagnosis and the needs of the patients that has been created by rapid advances in genomics and to highlight the increasing responsibilities of obstetricians and primary care providers with regard to prenatal diagnosis. Every case with previously affected child is not just global developmental delay or Cerebral Palsy and entails detailed evaluation of the the index case with the help of clinical geneticist in peri conceptional period and not after conception because the turn around time for genetic tests is much more. Next generation sequencing needs to be used judiciously and cautiously. Targeted sequencing involves sequencing specific regions on gene based on clinical phenotype which s most cost effective. Whole exome sequencing is also cost effective involving sequencing of whole exome, Whole genome sequencing, sequences whole genome it is expensive but covers everything and can identify all kinds of various variants including SNPs, INDELs nd SV which may sometimes be difficult to interpret so at all times these tests need to be offered with the help of clinical geneticist.

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