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Editor's Choice

Ovarian Reserve Following High Intensity Focused Ultrasound In the Management of Leiomyomas

Currently the treatment protocol using magnetic resonance guided (MRg) / ultrasound guided (USg) high intensity focused ultrasound (HIFU) for women with symptomatic fibroids and adenomyosis is gaining popularity worldwide. It is considered superior to many other non invasive or invasive procedures. Levels of Anti Mullerian Hormone (AMH) as a biomarker for ovarian reserve were evaluated following treatment with HIFU. This was done specially in women to assess ovarian reserve and response to a particular stimulation protocol. Concerns were raised when the levels were observed low in some early studies.

Anti Mullerian Hormone (AMH) is a peptide, produced by the granulosa cells of the preantral and small antral follicles up to 6 mm in diameter. Serum AMH levels are found to correlate strongly with the number of antral follicles in the ovary. With this observation, the levels of AMH is taken as an indicator of ovarian reserve.[1,2] Serum levels of AMH is cycle independent and can be measured at any phase of menstrual cycle. The intercycle and intracycle variations in AMH levels are insignificant. Any woman using combined oral contraceptives or GnRH analogues for a long period (> 1 year) has been found to have low levels of AMH. The main interest in measuring AMH before any assisted reproductive cycle (ART) is to assess: (a) ovarian follicular pool reserve (b) ovarian responsiveness to a particular stimulation protocol and (c) prediction of any excess ovarian response to ovarian hyperstimulation syndrome (OHSS).

Since the establishment (July, 2013) of *International Society for Minimally Invasive and Non invasive Medicine (ISMINIM)*, more than 13 countries in the world, are making continued progress in the field of non invasive treatment. Earlier study reports raised the concern as regard to the effect of HIFU on ovarian reserve.[3,4] Principally the technique focuses ultrasound energy to cause targeted tissue necrosis during the treatment period. The procedure is done with conscious sedation. Ablation of an average size myoma takes around 60 to 90 minutes. Once the HIFU treatment is conducted with precise imaging guidance, only the leiomyoma is ablated without affecting the smooth muscles of the uterus. After treatment, the fibroid is absorbed. The normal uterine shape and the cavity is restored gradually. It is stated that the myometrium maintains uterine wall integrity and tension during pregnancy and labor.

Moreover there is no adverse effect on the function of ovary and endometrium.[5,6] With the present state of knowledge, there is no extra risk for the patient to conceive after the HIFU treatment. Other advantages are: it is a non invasive method with rapid recovery time and quick return to daily activities. More importantly, it preserves the uterus. However, some patients may need repeat treatment or some other alternative method of treatment as the symptoms may remain either uncontrolled or poorly controlled. As with any other method of treatment, not all women with fibroids or adenomyosis could be benefitted with this treatment. There are few contraindications to this form of treatment. Contraindications to MRI itself is an independent factor. Leiomyoma that are much vascular respond poorly to this treatment.

Long term data regarding the duration of symptomatic relief following treatment are awaited. Initial study reports suggest that MRI / US guided HIFU is safe and effective. It can be used as an alternative for hysterectomy for women not responding to medical therapy. Treatment with HIFU for the treatment of symptomatic fibroid and adenomyosis has been approved by FDA (2008).

Concerns were expressed as regard the effect of HIFU on ovarian reserve when evaluated with the levels of serum anti mullerian hormone before and after the treatment. More published reports are now available to evaluate the changes.[6]

Overall used median treatment time 140.5 minutes, median ablation (sonication) time 24 minutes, median energy delivered was 400 124.10 joules. This was correlated with serum AMH levels before and 6 months after HIFU. There was no significant difference in AMH levels between two time points ($P > 0.05$). Neither any woman became amenorrhoeic nor any one presented with symptoms of menopause. It was concluded, HIFU is a safe and effective treatment of a woman with symptomatic fibroids and adenomyosis and this does not affect ovarian function or reserve.[7]

It is observed that initial studies included many elderly women with age more than 40 years. Hence the result was affected adversely. This may not be applicable to women in the younger age. Currently median HIFU treatment energy in most studies are 364 713.8 joules for fibroids and 317 847.2 joules for adenomyosis respectively. The clinical use of HIFU as the non invasive treatment of symptomatic fibroids and adenomyosis is considered a priority in many countries.

Reports of pregnancy following treatment with HIFU have been currently observed and found to be encouraging. Eighty pregnancies have been reported in 78 patients following treatment with HIFU. Preliminary reports of pregnancy outcome following HIFU is satisfactory.[8,9] However, it is essential that pregnancy and labor management must be done with intensive monitoring till sufficient data are available with more safety outcome measures.

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PCOS - A Metabolic Epidemic??

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Overview

Our first knowledge about Polycystic ovarian syndrome came from a case series report by Dr. Irving F. Stein and Dr. Michael Levental in 1935. They demonstrated a triad of polycystic ovaries, hirsutism and oligo/amenorrea in a series of women and successfully treated them by wedge resection of ovary thus giving an idea that the disease is ovarian in origin.[1] After that, the subject received tremendous attention by researchers all over the globe and about 28,000 research articles were published on this topic in subsequent 80 years, majority of them in the last 15 years. Thanks to all these researches, the perception regarding the pathophysiology of the disease has changed and it is now considered a multisystem, metabolic and endocrine disease with reproductive implications.

Is the incidence of PCOS on the rise?

The prevalence of PCOS according to diagnostic criteria of NIH, Rotterdam and AE- PCOS Society were 6%, 10% and 10% respectively as described in a meta-analysis.[2] Indian studies reported a higher prevalence ranging from 2.2 to 26%.3 So, about 1 in 5-17 women are suffering from this condition. The high prevalence may be due to unhealthy life style including fast food, lack of exercise and sedentary habits. It may also be due to increased awareness and improved accessibility to medical care.

Metabolic Syndrome and PCOS

There are different criteria for diagnosis of Metabolic syndrome (Table 1). (WHO-World Health Organization; NCEP ATP III-National Cholesterol Education Programme Adult Treatment Panel III; IDF- International Diabetic federation.)

The metabolic syndrome, although varies in diagnostic criteria, has 4 major components: obesity, Insulin resistance/Impaired glucose tolerance/Type II diabetes mellitus, dyslipidemia, and hypertension. PCOS is intimately related to all these components as follows.

Table 1: Definitions of MBS for women, according to WHO, NCEP ATP II and IDF criteria

WHO	NCEP ATP III	IDF
T2D or IFG or IGT or insulin resistance plus ≥ 2 of the following: • BMI > 30kg/m^2 or WHR > 0.85 • HDL < 1.0 mmol/L (<40 mg/dL) • TG ≥ 1.7 mmol/L (150 mg/dL) • BP ≥ 140/90 mmHG or use of blood pressure medication • microalbuminuria > 20 pg/min • Alb/Crea ratio ≥ 30 mg/g	≥3 of the following: • WC ≥ 88 cm • HDL < 1.3 mmol/L (<50mg/dL) • TG ≥ 1.7 mmol/L (150 mg/dL) • BP ≥ 135/85 mmHg or use of blood pressure medication	Central obesity defined as WC above the ethnicity specific cut-off plus ≥ 2 of the following: • TG ≥ 1.7 mmol/L (150 mg/dL) or specific treatment • HDL< 1.3 mmol/L (<50mg/dL) or specific treatment • BP ≥ 135/85 mmHg or use of blood pressure medication • fasting plasma glucose ≥ 5.6 mmol/L (100 mg/dL) or previously diagnosed T2D

BP=blood pressure; HDL=high density liporprotein cholesterol; IGT=impaired gluse tolerance; T2D=type 2 diabetes; TG=triglyceride; WC=waist circumference; WHR=waist to hip ratio.

PCOS and Obesity

35-60% women with PCOS are obese and 28% of all obese women have PCOS. Obesity is the cause and not the effect of PCOS. There are 3 distinct mechanism by which obesity is involved in the pathogenesis of PCOS- first, by increasing aromatization of androgen to estrogen in the adipose tissue leading to hyper estrogenism and chronic anovulation; second, by decreasing Sex hormone binding globulin (SHBG) thus increasing the free estrogen and androgen level and third, by causing impaired glucose tolerance leading to hyperinsulinemia which cause increased androgen production in the ovary.

Hence, a weight reduction even to the extent of 2-5% is beneficial in restoring ovulation and regularizing menstrual cycle and improving metabolic consequences.

Insulin Resistance/Impaired Glucose Tolerance/Type II Diabetes Mellitus and PCOS:

50-75% of women with PCOS have insulin resistance, 35% have IGT and 10% have DM.

Prevalence of PCOS is 6 fold higher in women with Type II DM.

Insulin resistance lead to compensatory hyper insulinemia which acts on the ovary through IGF 1 receptors resulting increased androgen production in the ovary. Apart from that, insulin causes decreased production of SHBG from liver and thus increasing free androgen and estrogen level. This hyper androgenemia in turn aggravates Insulin resistance further. So, this becomes a vicious cycle.

Dyslipidemia and PCOS:

70% of PCOS cases have at least one abnormal lipid parameter[4] and it may be due to Insulin resistance, increased androgen and increased estrogen.

Hypertension and PCOS:

Hypertension is 3 times more common in PCOS. Although there is no direct evidence of increased incidence of Cardio-vascular disease, dyslipidemia increases the risk of premature atherosclerosis in PCOS.

Apart from these PCOS is related to the following multi-system dysfunction:

Liver Disease

Non Alcoholic Fatty Liver Disease (NAFLD) / Alcoholic Steatohepatitis (NASH):

Incidence of NAFLD is 15-57.8% (based on elevated transaminases) and 60-70% (on USG) among obese PCOS and 39% among lean PCOS.5

Dermatological Problems

PCOS is associated with acne in 67.5%, hirsutism in 62.5%, seborrhea in 52.5%, alopecia in 30% and Acanthosis nigricans in 22.5% cases.[6]

PSYCHOSEXUAL PROBLEMS: Depression, anxiety, eating disorders, sexual problems, low self esteem are common but undiagnosed entities associated with PCOS leading to Reduced Quality of Life (QoL).[7]

OBSTRUCTIVE SLEEP APNOEA (OSA): Although the incidence is high in obese PCOS, it remains often undiagnosed. OSA is a risk factor for HT, stroke, CV disease, pulmonary HT.

PCOS and Cancer:

Risk of Endometrial cancer is increased by 2.89 fold in women with PCOS due to effect of unopposed estrogen and risk increases in women with less than 4 period/year.[8] However there is no additional risk for breast and ovarian cancer.

MANAGEMENT GUIDELINE:[9] Although, women with PCOS come to the gynecologist for menstrual and infertility related problems, but the duty of the gynecologist is beyond regularization of her menstrual cycle and inducing her ovulation. They should Screen for metabolic and multisystem diseases, counsel and make them aware regarding the long term health hazards, treat and refer to the concerned specialists as required.

SCREEN: It is necessary to clinically assess OBESITY by BMI and Waist –Hip ratio and Hypertension by regular BP measurement.

USG upper abdomen and liver function tests, Oral Glucose tolerance test, and Lipid profile are very important screening tests.

TVS for endometrial thickness and endometrial sampling is necessary in older women with PCOS who have infrequent periods in order to rule out endometrial hyperplasia and cancer.

COUNSEL: One need to elicit history of OSA (sleep disturbance, fatigue, snoring) and Psychological Problems (depression, lack of interest etc). It is very important to make the women aware regarding long term health hazards of PCOS.

TREAT: First line of therapy is life style modification, by Diet control and Exercise. Low calorie diet and avoidance of fast food is necessary. Brisk walking for at least 30 minutes /day is to be advised.

REFER: A multidisciplinary approach in managing metablolic and multisystem disorders with metabolic specialist and endocrinologist, gastroenterologist, pulmonologist, nutritionist and psychiatrist may be required according to individual need.

NB: As PCOS has become a major health hazard in recent times, another review article on PCOS has been added in this issue. We invite debates and discussion on this subject in the section "letter to Editor"

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Original Article: Obstetrics

Evaluation of Modified Dose versus Standard Pritchard Regimen of Magnesium Sulfate in Eclampsia

Vandana Verma,[1] Vinita Das,[2] Anjoo Agarwal,[3] Amita Pandey,[4] Vaishali Jain[5]

Background

Pre-eclampsia complicated by generalized tonic clonic convulsion is termed eclampsia. Eclampsia is most common in last trimester and becomes more frequent as term approaches. In more recent years there has been an increasing shift in the incidence of postpartum eclampsia, presumably due to increased access to prenatal care, earlier detection of preeclampsia and prophylactic use of Magnesium Sulfate. In a pregnant woman once eclampsia has ensued, the risk to both mother and fetus is appreciable. Control of fits and blood pressure forms the mainstay of management in these women. Although several drugs have been tried to control fits in eclampsia, Magnesium Sulfate is the drug of choice. Different regimens for administration of Magnesium Sulfate have been tried and the most commonly recommended one is the Pritchard regimen.[1] But the dosage used in this regimen is based on height and weight parameters of western women. Indian women have a lesser weight, height and BMI from their western counterparts. So this dosage frequently causes toxicity of Magnesium Sulfate. In order to avoid the toxicity shown in Pritchard regimen and to overcome the recurrence of fits seen in many low dose regimen studies, we developed a modified Medium dose regimen of Magnesium Sulfate for the treatment of eclampsia in Indian women and planned the present study to evaluate it. In this regimen we gave the loading dose as 10 gm (4 gm I.V.I. + 3 gm I.M.I in each buttock) and the maintenance dose as 3 gm I.M.I. in alternate buttock, at 4 hourly interval till 24 hours after delivery or last fit whichever is later.

Aims and Objective

To compare the efficacy and toxicity of Magnesium Sulfate and perinatal outcome in both the regimen of Magnesium Sulfate.

Material and Methods

It was a randomized controlled trial over a period of one year on 75 patients of eclampsia in the Department of Obstetrics and Gynecology, King George Medical University, Lucknow. 75 patients were randomly selected and distributed in 2 groups (group 1-36 cases received medium dose regimen, Group 2-39 controls received Pritchard regimen). Randomization was done by using computerized random numbertable and allocation concealment was done by using sealed envelope of these numbers. Women already taken anticonvulsant drug in referred cases, cases presenting with serious complication, cases with uncertainty of diagnosis were excluded. All the included patient's relaties were informed about the purpose and protocol of study and consent was taken. Approval of the ethical committee of the institute was obtained.

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Protocol for medium dose regimen: Loading dose of 10 gm of Magnesium Sulfate (4 gm 20% Magnesium Sulfate solution slow I.V. + 3 gm 50% Magnesium Sulfate solution I.M. in each buttock), followed by 3 gm 50% Magnesium Sulfate solution given alternatively in each buttock at 4 hourly interval, to be continued 24 hours after delivery or last fit whichever is later.

Protocol for standard Pritchard regimen: Loading dose of 14 gm of Magnesium Sulfate (4 gm 20% Magnesium Sulfate solution slow I.V. + 5 gm 50% Magnesium Sulfate solution I.M. in each buttock). This is followed by 5 gm 50% Magnesium Sulfate solution given alternatively in each buttock at 4 hourly interval, to be continued till 24 hours after delivery or after last fit whichever is later.

All the patients' data were entered on MS Excel sheet and analysed by statistical package of social sciences and statistical significance was set at $P \leq 0.05$.

Results

Seventy five subjects with eclampsia were registered in this study. Thirty six subjects were allocated to group I (Cases) and 39 were allocated to group II (Controls). Twenty eight (77.8%) subjects in study group had antepartum eclampsia while in control group this proportion was 79.5%. No case with intrapartum eclampsia was recorded in the present study.

On observation 58.3% subjects of study group and 48.7% subjects of control group were in age groups <25 years. In the study group, 58.3% subjects were nullipara and remaining 41.7% were multipara. However, in control group, the subjects were almost equally distributed paritywise. Majority of subjects in both the groups were of normal (BMI 19-25) weight. Only 5.6% subjects in study group and 10.3% subjects in control group were underweight.

32.1% subjects of study group and 51.6% subjects of control group had a gestational age of <37 weeks at the time of admission. 77.8% subjects of study group and 79.5% of Control Group patients gave history of <10 fits before admission.

Only 1 (2.8%) of study group and 1 (2.6%) of control group subject had recurrence of fits following initiation of Magnesium Sulfate therapy. Patient in the study group did not respond to an additional 2g I.V. (half loading dose) of Magnesium Sulfate therapy & needed Phenytoin for control of fit.

No patient in study group had loss of deep tendon reflexes whilein the control group 5.1% patients had loss of deep tendon reflexes. No maternal mortality took place in study group. Two maternal mortalities took place in control group (5.13%). One mortality occurred due to cerebrovascular accident and the other due to severe aspiration pneumonitis. Both women expired on third post natal day and both were on ventilatory support since one and two days respectively.

There were 18 (64.3%) live births in study group and 21 (67.7%) live births in control group. 60.7% neonates in study group and 64.5% neonates in control group who were <2.5 kg of weight. Neonatal ICU care was required in 55.6% neonates of study group and 23.8% neonates of control group. Apgar score of baby at 1 minute after delivery was found to be <6 in 15 (71.4%) neonates in study group and 16 (88.9) neonates in control group. There was 1 neonatal death in each group.

Discussion

The highest incidence of eclampsia was found in the age group of <25 years (47.45%) in this study similar to the study conducted by Chaudhary P et al.[2] at Maternity Hospital, Kathmandu on 47 cases of eclampsia, out of which 66% were in age group <25 yrs. In study conducted by R. Nautiyal et al at Department of Obstetrics & Gynecology at Himalayan Institute of Medical Sciences, Dehradun, the mean age of women presenting with eclampsia was 25.5 yrs.[3]

Majority of cases of eclampsia in present study were nulliparous as in retrospective study conducted by Rashida Begum et al. at Dhaka Medical College in which 75% cases of eclampsia were primiparous.4

In the present study majority of women (78.6%) had antepartum eclampsia. The distribution of cases having antepartum and postpartum eclampsia was almost similar in the study and control groups. Similarly the study done by Bangal VB and Sharma A found higher incidence of antepartum eclampsia.[5,6] Chowdhury et al reported 65% cases of antepartum eclampsia in their study[7] and in study conducted by Sardesai et al at Department of Obstetrics and

Gynecology, Solapur Medical College 78.67% had antepartum/intrapartum eclampsia.[8]

In present study only 1 case (2.8%) of study group and 1 case (2.6%) of control group had recurrence of fits. Study done by Ekele BA et al conducted in the Department of Obstetrics and Gynecology at Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria, found 7.4% incidence of recurrent fits.[9] In present study no study group subject had loss of deep tendon reflexes and decreased urine output. In the control group only 5.1% cases had loss of deep tendon reflexes. In a prospective study at Dhaka Medical College (1997), by Rashida Begum et al using low doses of Magnesium Sulfate, of the 65 cases, only 5 had diminished knee jerk.

In our study no maternal death occurred in the study group and two maternal death occurred in control group (5.1%). Four Maternal mortality reported by Sardesai Suman in her low dose regime was 2.63%, whereas the maternal mortality reported by collaborative eclampsia trial with Pritchard regime was 3.8% and 5.2%.[10]

In this study there were 35.73% still births in study group and 32.3% still births in control group. All the women who had given birth to stillborn babies presented in the hospital with intrauterine death at the time of admission, 1 neonatal death each occurred in study group (5.56%) and control group (4.76%). Sardesai Suman et al reported 33.90% perinatal mortality in their study.[8]

Conclusion

Medium dose magnesium sulphate regime was effective for the control of eclamptic convulsions. Dose required for control of convulsion with medium dose magnesium sulphate regime was nearly half of standard Pritchard regime. There was no magnesium related toxicity with medium dose magnesium sulphate regime. There is need for additional multicentric case control trials to support the observations of the present study, before we recommend a change from standard Pritchard regime to medium dose magnesium sulphate regime, which suits the Indian women, having relatively low body mass index as compared to their western counterparts.

Table - 1
Distribution of patients according to basic characteristics

Maternal variables	Study Group (Medium dose regimen) (n=36)		Control Group (Pritchard Regimen) (n=39)		(χ2value) P value
Age in yrs	**n**	**%**	**n**	**%**	(χ2=1.453)
<25 years	21	58.3	19	48.7	.484
25-35 years	15	41.7	19	48.7	
>35 years	0	0	1	2.6	
Parity	**n**	**%**	**n**	**%**	(χ2=.376)
Nullipara	21	58.3	20	51.3	0.54
Multipara	15	41.7	19	48.7	
BMI Category	**n**	**%**	**n**	**%**	(χ2=1.549)
Underweight (BMI<19)	2	5.6	4	10.3	0.461
Normal weight (BMI 19-25)	34	94.4	34	87.2	
Overweight/Obese (BMI>25)	0	0	1	2.6	
Type of eclampsia	**n**	**%**	**n**	**%**	(χ2=.033)
Antepartum	28	77.8	31	79.5	0.857
Postpartum	8	22.2	8	20.5	

Table - 2
Maternal complications

S. No.	Complications	Study Group (Medium dose regimen) (n=36)	Control Group (Pritchard Regimen) (n=39)	P value (χ2=1.897)
		n (%)	n (%)	
1.	Magnesium Sulfate toxicity (Loss of Deep Tendon Reflex)	0	2 (5.1%)	0.168
2.	Maternal mortality	0	2 (5.13)	0.168

Table - 3
Perinatal outcome

S.No.	Fetal parameters	Study Group (Medium dose regimen) (n=28)		Control Group (Pritchard Regimen) (n=31)		P value
		n	%	N	%	
1.	Birth wt<2.5 kg	17	60.7	20	64.5	.763(χ2=.091)
2.	Stillbirth	10	35.7	10	32.3	.779(χ2=.078)
3.	NICU care required	10	55.6	5	23.8	.042(χ2=4.172)
4.	Neonatal death	1	5.56	1	4.76	.911(χ2=.013)

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Successful Maternal and Perinatal Outcome in Peripartum Cardiomyopathy – Our Experience

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ABSTRACT

Background: Peripartum Cardiomyopathy is a rare but grave complication of pregnancy. It is one of the less studied and investigated conditions, which is difficult to diagnose and treat, with quite high morbidity and mortality.

The incidence of peripartum cardiomyopathy varies from 1 in 1300 to 4000 pregnancies. The mortality rates ranges from 25-50%. In the last four years, six cases of Peripartum Cardiomyopathy has been diagnosed and managed in our unit. Presentation of these cases were different. Clinical suspicion of the condition helped in early diagnosis and prompt management of the patients. The etiology of peripartum cardiomyopathy is unknown, but inflammatory cytokines, myocarditis, viral, auto-immune and idiopathic causes may contribute.

Cases: In the first case patient had elective LSCS and she collapsed during LSCS and was later diagnosed as a case of peripartum cardiomyopathy.The second case had LSCS for BOH with term pregnancy, peripartum cardiomyoathy in this case also developed after LSCS. The third case was diagnosed peripartum cardiomyopathy antenatally .She had twins with anemia, LSCS was done in view of both fetuses presenting by breech at term. The fourth case had an emergency LSCS under GA but took longer time for reversal- unexplained metabolic acidosis, tachycardia persisted with low O2 saturation. Cardiologist consulted and echo confirmed the diagnosis of PPCM. The fifth case had NVD & PP ligation. On 6th post-op day patient had sudden collapse and diagnosis of PPCM was made. The sixth case had term pregnancy with PIH and dyspnoea. On investigation she was diagnosed as PPCM antenatally and managed accordingly. All the patients were managed conservatively. Five of them are well and alive and their LVEF has improved upto 65%, whereas one patient still has LVEF of 25% even after four years.

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Conclusion: Fortunately maternal peripartum cardiomyopathy is infrequent but serious complications of human pregnancy, that threatens both mother and fetus . Clinicians must be aware of this problem in order to provide prompt diagnosis & management. The successful outcome is possible with an intensive treatment. This condition tends to recur in subsequent pregnancy. A minimal interval of 3 yrs after the recovery of function has shown to be safe for subsequent pregnancies , in consultation with a cardiologist.

Keywords: peripartum, cardiomyopathy, LSCS, conservative, maternal echo.

Introduction

Peripartum cardiomyopathy is a rare but grave complication of pregnancy . It is one of the less studied and less investigated conditions, which is difficult to diagnose and treat, with quite high morbidity and mortality even in best centres.

Hibbard et al from Chicago in 1999[1] has emphasized for uniform criteria that defined peripartum cardiomyopathy. The new definition include heart failure within the last month of pregnancy or 5 months postpartum with the absence of preexisting heart diseases or with no determinable etiology with the echocardiographic criteria of left ventricular dysfunction, with ejection fraction less than 45% or M -mode fractional shorting less than 30% or both and end diastolic dimensions more than 2.7%cm / m^2.

Tateda K. etal in 2000 AD[2] reported that the incidence of Peripartum Cardiomyopathy varies from 1 in 1300 to 4000 pregnancies. The mortality rates of this disorder in the acute and subacute phases range from 25% - 50% The prognosis is especially poor in-patients with cardiomegaly persisting more than 6 month and with low left ventricular ejection fraction.

In last four years six cases of Peripartum Cardiomyopathy had been diagnosed and managed in our unit. The successful maternal and perinatal outcome in this series is presented here.

Case 1

31 yrs old, G2 P 1+0. with previous LSCS term pregnancy admitted for elective caesarean section .

Patient was a diagnosed case of bronchial asthma for last 1 month on medication.

She had LSCS under spinal anesthesia. As soon as the baby was delivered, patient developed severe hypotension not responding to IV fluids, blood volume expander, and mephentine. Patient was intubated, put on intermittent positive pressure respiration and steroids. Since, she was not maintaining her BP and oxygen saturation, she was shifted to ICU. She stabilized in 8-10 hrs, but her tachycardia persisted . She was put on ventimask from BIPAP after 12 hours. Patient was put back on BIPAP support as her oxygen saturation fell to 85%, she was dyspnoeic and her CVP was 22cms.

ECG was done and showed nonspecific ST changes. Echo showed generalized hypokinesia . LVEF 30% severe MR, TR with PAH.

Diagnosis of peripartum cardiomyopathy was made. She responded to ACE inhibitors, digoxin, antibiotics, low molecular weight heparin and lasix and was discharged on 10th post op day in stable condition..

Patient still has LVEF of 30-35% even after four years, and continues to be on treatment for cardiomyopathy.

Case 2

28 yrs old G5P0A4 36+1 weeks amenorrhea with BOH admitted with pregnancy induced hypertension breech presentation and decreased fetal movements, so she had caesarean section done under general anaesthesia .

Through out surgery patient had tachycardia. Patient was extubated but was not maintaining Oxygen saturation, so cardiologist was consulted.

Since her antenatal, preoperative echo was normal, so diagnostic dilemma persisted.

She continued to have tachycardia and low oxygen saturation.

Her ECG was done which showed fresh Right Bundle Branch Block, and then echo was done which showed global left ventricular hypokinesia with LVEF

of 35% with mild MR. Diagnosis of Peripartum Cardiomyopathy was made Patient was shifted to ICCU and managed conservatively with digoxin, antibiotics, fragmin and lasix.

She had improved, and after 3 months her LVEF was 65% and complete resolution of symptoms.

She came after ten months with a pregnancy of six weeks and medical termination of pregnancy was done.

Again she was pregnant after a gap of one year, and this time an elective LSCS was performed with an uneventful antenatal and postnatal period.

Case 3

26 yrs old primigravida with twin pregnancy presented at 26 weeks of amenorrhoea admitted with severe anemia, she was investigated and managed conservatively.

Her echo showed mild MR with LVEF of 65%. This patient at term was posted for elective caesarean section in view of both fetuses presenting by breech .

On the preoperative day she again developed dyspnoea. Her echo was done which showed global left ventricular hypokinesia with LVEF of 30% and the diagnosis of Peripartum Cardiomyopathy was made.

With due precaution caesarean was done, and patient shifted to ICCU as a precautionary step and managed conservatively.

Case 4

36 yrs old G2 P1 with 36+5 wks amen. admitted with leaking P/V. She was booked antenatally and had dry irritating cough with bilateral bronchial breath sounds for last three weeks. She was taken up for emergency LSCS under GA in view of cord prolapse. Surgery was uneventful but the patient took long time to recover from anaesthesia. Unexplained metabolic acidosis and tachycardia persisted with low oxygen saturation. X-Ray chest showed bilateral homogenous density in the upper mid zone .Cardiologist consulted, Echo was done, which showed LVEF of 30%, mild MR TR the diagnosis of peripartum cardiomyopathy was confirmed. She was managed conservatively and discharged in fair condition.

Case 5

29 yrs G3P2 admitted at term pregnancy with labour pains. Had NVD and had PP ligation. She had breathlessness in the post partum, and was managed with nebulisations and bronchodilators. On the 6th post-operative day patient had sudden collapse and was shifted to ICU. Her echo was done – LVEF of < 25%.Diagnosis of PPCM done and managed accordingly.

Case 6

25 yrs old G2P1 presented at 37 weeks with PIH and dyspnoea, she was given treatment for bronchitis, but cough persisted. Cardiologist consulted and echo done. Her echo was done which showed global left ventricular hypokinesia with LVEF of 30% and the diagnosis of Peripartum Cardiomyopathy was made. She was stabilized by anti- hypertensives, digoxin, she went into spontaneous labor and delivered normally

Discussion

Presentation of all these cases were different. In the first case we were taken unawares, our patient was being treated as a case of bronchial asthma, and we took time to diagnose her as a case of Peripartum Cardiomyopathy.

Second case had an antenatal echo done, as the patient complained of breathlessness & palpitation in the antenatal period.

In the post op period since the patient was not maintaining her Oxygen saturation so echo was done and she was diagnosed as a case of peripartum cardiomyopathy.

Third case had anemia at 26 weeks of amenorrhoea with dysnoea, echo was done despite the fact that her dyspnoea which could have been explained by her anemia. But before her elective LSCS she developed dyspnea again, an echo was repeated and the diagnosis of PPCM was made.

We could then take adequate precaution for our patient during LSCS and postpartum period.

The fourth case was diagnosed as chronic bronchitis antenatally. But in the post – operative period her low oxygen saturation persisted. On thorough investigation she was diagnosed as PPCM.

The fifth case was also treated as a case of chronic bronchitis unless she collapsed and was shifted to ICU, & her subsequent echo proved the diagnosis.

The sixth case was diagnosed in the antenatal period with peripartum cardiomyopathy and went into spontaneous labor and delivered normally

Clinical suspicion was important in all the cases. It helped us in early diagnosis and prompt management of the patients.

Discussion:

Peripartum cardiomyopathy is a less recognized, infrequent but serious complication during pregnancy, of unknown etiology and is associated with excess morbidity and mortality in women of child bearing age.[3]

The incidence of peripartum cardiomyopathy varies from 1 in 1300 - 4000 pregnancy

The mortality rates of this disorder in the acute phase and subacute case range from 25% to 50%. The prognosis is especially poor in patients with cardiomegaly persisting more than 6 months, and in patient with low left ventricular ejection fraction.

The etiology of peripartum cardiomyopathy is unknown but viral, autoimmune and idiopathic causes may contribute.[4]

The risk factor includes multiple gestation, pregnancy induced hypertension, multiparity, advanced maternal age, caesarean section and African - American women. Other possible etiologic factors include prolonged tocolysis, proinflammatory cytokines (TNF, IL1, IL6). Abnormalities of relaxin, and ovarian hormone produced during pregnancy, can cause positive inotropic and chronotropic properties and cause excessive relaxation of the cardiac muscles. It is unclear, that whether nutritional deficiency may play a role in the pathogenesis of PPCM.

The cardinal symptom are that of easy fatigability, tachypnoea, orthopnoea, varying degree of dyspnoea and features of pulmonary oedema.

The examination reveals raised jugular venous pressure, pulmonary rales, cardiomegaly and third heart sound.

The murmur of mitral or tricuspid regurgitation may be audible, there may be peripheral oedema.

Since these clinical features are common to many other disease as well as to underlying heart disease, it is important to rule out all other possibilities before diagnosing peripartum cardiomyopathy.

Diagnostic criteria:

ECG - only non specific ST-T wave abnormalities, arrhythmias or an infarct pattern.

Echocardiography: characteristically shows an enlarged heart with global hypokinesia. The ejection fractions are typically 15-25% in symptomatic patients.

Chest X-Ray shows an enlarged heart, elevated hemi-diaphragm with a fluffy pulmonary infiltrate, worse at hilum which becomes less obvious towards the periphery.

Arterial blood gases show: Significant hypoxia, normal ph, low to normal Pco2.

The objective deficit is one of failure to oxygenate, rather than failure to ventilate.

CVP is raised, and CWP (Pulmonary capillary wedge pressure) is raised.

If diagnosed in the antenatal period, prompt delivery depending on the maturity of the foetus should be considered.

Immediate and vigorous diuretic therapy is important, digoxin, anticoagulants, inotropic agents and many hypertensive drugs for after load reduction are somewhat more controversial interventions .

Management of such patients includes multidisciplinary approach.

Bolis et al from Italy 1999[5] reported the case of a women presenting with severe cardiac failure immediately after caesarean section just as in our first case.

The diagnosis of Peripartum Cardiomyopathy was difficult due to the presence of pre-eclampsia and acute pulmonary oedema which occured four hours after delivery.

About 50% - 60% of patients have spontaneous recovery of cardiac function within six months of onset. The remainder either have persistent ventricular dysfunction or deteriorate, to die early or to receive cardiac transplantation.

There is a tendency towards recurrence with subsequent pregnancy.

Hence, a previous history of peripartum cardiomyopathy is a relative contraindication to repeat pregnancy in mothers who have recovered normal cardiac functions, and an absolute contraindication to pregnancy if there is persistent left ventricular dysfunction.

Albanesi et al 1999[6] concluded a study on 34 patients to assess the effect of subsequent pregnancy after Peripartum cardiomyopathy on maternal and fetal outcome. They concluded that subsequent pregnancy are well tolerated after Peripartum cardiomyopathy but not devoid of risk. A minimum of 3 years after recovery of function seem to be safe for subsequent pregnancies.

But our second patient despite all advise was pregnant in quick successions, but we managed to take her safely through term and deliver her.

CONCLUSION

Fortunately maternal peripartum cardiomyopathy is infrequent but serious complications of pregnancy, that threatens both mother and fetus. Clinicians must be aware of this problem in order to provide prompt diagnosis and management. The successful outcome is possible with an intensive treatment which ensures a favorable return of normal left ventricular function. This condition tends to recur in subsequent pregnancy.

A minimal interval of three years after the recovery of function has shown to be safe for subsequent pregnancies, in consultation with a cardiologist.

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Review Article: Gynecology

PCOS and Its Complications in Reproduction

Dr Vandana Walvekar

PCOS (Polycystic ovarian syndrome) is one of the most important and prevalent cause of anovulation characterized by hyperandrogenism, Insulin Resistance (IR) and hyperinsulinemia, and anovulation despite controlled ovarian hyperstimulation (COH),This cohort of symptoms arising at adolescence persisting into adulthood and into the perimenopause of the woman's life has long reaching adverse effects on her reproductive life and metabolic status.

The prevalence of PCOS worldwide is between 5% to 15% which amounts to a very large number of women being affected. In the reproductive age group the incidence can be as high as 26% in certain population e.g. South Asian countries. Thus the problem is serious and its implications even more so.

Let us evaluate: Clinically, a PCOS woman can present as an adolescent/reproductive age group/peri-post menopausal age group with the following:

1. Obese with BMI>30 but a small percentage can even be non obese with normal BMI Central obesity is almost always present
2. Menstrual disorders ranging from oligomenorrhea with occasional menorrhagia and oligovulatory or anovulatory cycles
3. Subfertile and infertile

Adolescent and Adult forms are discussed with its diagnosis and risks in reproduction

Diagnosis: Clinical examination: Weight, BMI, BP check, evidence of hirsutism, gynecological examn leads to normal uterus with enlarged palpable ovaries, Breast may reveal areolar hair or galactorrhea

Investigations: This being a metabolic dysfunction an evaluation of hormonal status is essential iR being the basic abnormality the foll are done:

CBC, urine as routine, FSH/LH ratio is raised, FBS/PLBS/OGTT especially with family history of DM, TSH, PRL prolactin assays to rule out comorbidities, Testostrone/SHBG to evaluate androgen excess and most importantly Fasting Insulin levels.

Pelvic USG clinches the diagnosis with normal uterus, enlarged ovarian volume bilaterally and >10-12 follicles on ovarian periphery: Rotterdam criteria.

Complications and Risks in the Reproduction

Adolescent Phase: Most frequent is acne and hirsuitism of varying degree. Next comes the menstrual irregularities with/without menorrhagia leading to anemia, poor performance at educational facility, depression and personality issues with peers. Serious though these may appear: These can be efficiently managed with hormonal therapy, cosmetic management and counselling.

Adult Phase: This is more serious as the condition has lasted a few years and has long reaching consequences:

1. Reproductive cycle: anovulation, menstrual irregularity leading to subfertility and infertility requiring ART techniques frequently. She may also need surgical interventions of endoscopy and ovarian diathermy/drilling

2. Central obesity carries an additional risk of hypertension and hyperhomocystenemia with potential of atherosclerosis.

Pregnancy: This is not easy to achieve and having conceived is not an easy prenatal period with the risk of

1. Higher rate of spontaneous abortion, age>35 will have the risk of aneuploidy
2. Hypertension early in pregnancy with PIH,FGR: fetal growth restriction, development of GDM with its risks later in life as well as fetal growth: macrosomia, abnormal presentations ,placental deficiency
3. Last but not the least: obese pregnant may have sleep apnea with respiratory complications

Labor and Postpatrum Phase: Not uneventful!!:

1. Higher incidence of preterm delivery due to FGR PIH or macrosomia and PROM
2. Higher incidence of operative delivery LSCS and if vaginal delivery may have trauma due to fetal size, presentation or instrumentation

Long Term Complictions:

1. Obesity and hypertension.
2. Insulin resistance and GDM may lead to Type II Diabetes especially in the obese with central obesity and family history.
3. All this increases the incidence and susceptibility to atherosclerosis and cardiac events like MI and CVS, CNS accidents.

Primary Management Principle Only:

1. Lifestyle changes with 30mt/day exercise
2. Controlled diet with regarding weight reduction awareness
3. Wt. loss in the obese
4. Comorbities to be treated
5. Systematic and diligent treatment of reproductive hurdles of menstruation, ovulation infertility.

This is an ominous occurance in a woman's life must be treated with equal gravity with evidence based treatment plans.

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Case Report: Obstetrics

A Rare Case Of Necrotising Fasciitis of Leg in Twin Pregnancy with Cellulitis in One Twin

Dr. Jayoti Malhotra,[1] Dr. Purvi Khandelwal[2]

ABSTRACT

Background: Necrotising fasciitis is a rare but life threatening complication in pregnancy requiring prompt diagnosis and treatment. Here we report a case of twin pregnancy with necrotising fasciitis of leg, presenting to the emergency department with severe pain and high grade fever at term gestation. One twin also had cellulitis diagnosed after delivery, a rare phenomenon. Early debridement and broad spectrum antibiotics is the key treatment.

Keywords: necrotising fasciitis, pregnancy, obesity, mortality

Introduction

Necrotising fasciitis is a rare, potentially fatal and rapidly progressive infection of soft tissue that spreads along fascial planes. It involves wide spread necrosis of subcutaneous tissue, fascia and other adjacent tissue[1] with mortality upto 76%.[2] It is an acute surgical emergency requiring early surgical debridement of necrotic tissue, broad spectrum i.v antibiotics and multidisciplinary approach along with good ICU care.[3,4] Risk factor for occurrence of necrotising fasciitis in pregnancy are general immune suppression and obesity Co-morbidities like DM, peripheral vascular disease, injuries and other surgical procedure that disrupt skin barrier and septic focus anywhere else in body like perineum or genitourinary tract are also the contributing risk factors.[2,5]

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We report a case of NF of left leg in a multigravida patient with twin pregnancy where one twin also had cellulitis of limb.

Case report: Mrs A.K. 25 years age was admitted through emergency on 23/7/17 in Kurji Holy Family Hospital at 37 weeks 5 days gestation with twin pregnancy in early labor. She presented with severe pain in left leg for 4-5 days and fever for last 2 days. She was G3P2+0 with previous two vaginal deliveries with last child birth 4 years back. She was an unbooked case with only 2 antenatal check-ups in private nursing home. As told by the patient, she was diagnosed with Hep B positive in her last pregnancy and for which she was on treatment.

Clinical examination and treatment

Patient was toxic, flushed look, febrile 102ºC, pulse 120/min, BP 100/60 mmHg. She was conscious and was having severe left leg pain. She was unable to move the leg. Her left leg was swollen below knee, edematous, tender, discolored with multiple eruptions seen at various sites. Right leg edema was also present but

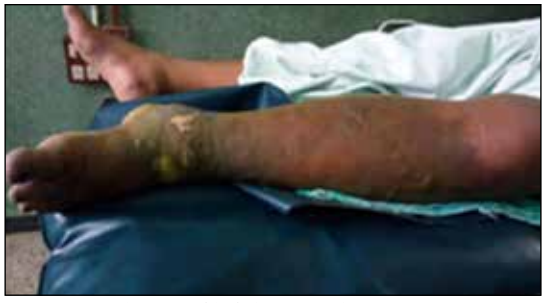

Fig.1: Condition of left leg on admission

Fig.2: Left leg of one of the twin showing cellulitis

Fig.3: At the time of first debridement

Fig.4: Left leg after 43 days showing granulations, ready for grafting

that was normal physiological oedema of pregnancy (Fig.1). There was no history of any recent trauma. She was in early labor at the time of examination. Fetal heart rate of both twins was normal and regular. The laboratory results on admission were as follows: white blood cell (WBC) 32600/dl with mild toxic granulations present on polymorph, Random blood sugar 168mg/dl, s.creatinine 0.9, urea 20, Hb 10.6 gm/dl, C-reactive protein 29mg/dl. Pus and tissue were sent for culture sensitivity and histopathology respectively. Culture report later came out to be positive with streptococcus species and on HPE tissue necrosis was seen and no definite diagnosis could be made.

Initially broad spectrum antibodies were started and Zinly capillary glucose monitoring was done. Engylycacmie status was achived by medication. Surgical opinion was sought in the emergency room and provisional diagnosis of superficial spreading cellulitis with necrotising fasciitis was made. She delivered uneventfully within 8hrs, two male babies weighing 2435gm and 2700gm were born. Twins were dichorionic-diamniotic with normal APGAR. However features of cellulitis were noted in right arm and leg of first twin and i.v. antibiotics were started (Fig.2). Exact cause for fetal cellulitis could not be ascertained although hematogenous spread from mother was suspected.

Discussion

Necrotising fasciitis is a life threatening surgical emergency with case fatality rate > 40% in single centre studies.[6] It involves severe infection of subcutaneous tissue that spreads along deep fascial planes causing vascular occlusion and ischemic necrosis.[1–5] A population based cohort study by Lavi and Phillips Watkin has shown that incidence of pregnancy associated necrotising fasciitis (PANF) hospitalisation is rising by 14% per year.[7]

Predisposing factors for development of NF are immunocompromise situations, HIV, severe anemia, malnutrition, diabetes etc. Pregnancy is a definitive risk factor for development of NF due to immunosupression especially in late third trimester and post partum[8,9] as was the case in our patient. Obesity a well known risk factor,[10] was also found in our patient (BMI =30). Our patient was carrying twin fetuses which further hampered lymphatic drainage from lower limb and predisposed to stasis, edema and infection.

It often clinically presents with severe pain, high grade fever, redness, edema, toxic look, leucocytosis. Without prompt and urgent therapeutic intervention, it may rapidly lead to septic shock syndrome with cyanosis, hypotension and tachycardia, altered level of consciousness, multiorgan failure, and death.[4,5] The inflamed skin appears erythematic with edema and

blistering but its involvement is smaller than the extent of necrosis of the underlying subcutaneous tissue and fascia, making the clinical distinction between simple cellulitis and NF extremely difficult.[1,4,5,11] On admission, our patient had similar signs and symptoms..

The mean age of patients with NF is 38-44 yrs as reported in literature but our patient was exception being only 25 yrs. According to causative organism NF may be caused by aerobic, anerobic, or mixed flora.[1] It can be broadly divided into 3 types –

- Type I, (polymicrobial)
- Type II, (group a streptococcus)
- Type III (gas gangrene)

Our patient belonged to type 2 category.

Management includes early diagnosis; prompt surgical debridement and broad spectrum i.v. antibiotics. MRI may be done to confirm diagnosis in certain cases if in doubt.

Conclusion

NF is a fatal illness that is often difficult to diagnose. Key to success is early recognition and prompt surgical debridement under antibiotic cover. The Laboratory Risk Indicator for NF (LRINEC) is a scoring system to distinguish NF from other severe soft tissue infection based on lab results for 6 variables CRP, WBC, Hb, Na level, S.creatinine, glucose.[12]

However a simple rapid bed side test is required for early recognition and to prevent morbidity and mortality.

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Instruction to authors IJOPARB in each issue. Page No. 40

For any more information, please contact Editorial Board

ijoparb1978@gmail.com

IJOPARB Communication

Letter to the Editor

Article Name: **Anti Mullerian Hormone in Reproductive Biology**
IJOPARB Vol 07 : No 1 : Jan-Mar 2017 : Pg 7-10

Sir,

Your article **"Antimullerian Hormone in Reproductive Biology", published in IJOPARB, Vol. 07, No. 01, Jan-Mar 2017; pg: 07-10** is a very relevant topic. A clear idea of AMH is obtained by reading your article. This is very important and essential for day to day gynecological practice. This is specially important while treating patients with infertility and deciding the mode of therapy. It is well presented, compact and highly informative to keep one updated. However, I have got queries regarding levels of AMH and use of **high intensity focused ultrasound**. Currently this mode of therapy is often used in different gynecological cases like intra mural or submucosal fibroids or adenomiosis. Controversies are seen in the literature as regard the levels of AMH following such therapy.

We would be happy if you could throw some more light on this debated area.

Looking forward to your kind response.

Your sincerely,

Dr. Rathindra Nath Ray
RMO Cum-Clinical Tutor
Department of Obstetrics and Gynecology
Calcutta National Medical College & Hospital
Kolkata - 700014

Editor's Reply:

Thanks Dr. Ray for your comments on the topic of "Anti Mullerian Hormone in Reproductive Biology", published in IJOPARB. There are controversies as regarding the level of AMH in respect of treatment following "High Intensity Focused Ultrasound" use in the treatment of uterine fibroids and adenomyosis. However the current research works in this respect appear to be beneficial. As this is an area of current research, I have decided to discuss this topic in a greater detail. Kindly go through the article in the section "Editor's Choice" of this issue (IJOPARB, Vol 07, No 03, July-Sept-2017; pg 73-74).

Thanking you once again for your interest to IJOPARB.

Regards,

Prof. Hiralal Konar
Editor-in-Chief
IJOPARB

Instruction to Authors

The Journal of Indian Society of Perinatology and Reproductive Biology which is the official publication of the Indian Society of Perinatology and Reproductive Biology (ISOPARB) invites original research articles in gynaecology / obstetrics / related subjects in the following category: Clinical Articles; Review Articles; and Brief Communications (including Case Reports).

All manuscripts should be prepared according to the guidelines detailed below. Any manuscript that has not been formatted as per the ISOPARB requirements will be returned to the author for correction. All manuscripts should be created and submitted in Word format.

1. SUBMISSION

Authors must submit manuscripts by Email ID –
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Hard-copy submissions will not be considered.

Please submit a cover letter to the Editor-in-Chief mentioning the following:

- Each author's name, address, and email address.
- Each author's affiliation and qualifications.
- The name of the author who is to deal with correspondence and proofs.

Once submitted, manuscripts undergo initial screening by the editorial staff and editors and then papers will undergo peer review.

2. Authors must give a separate "Author Guarantee" document mentioning the following:

(1) that all authors have met the criteria for authorship and have participated sufficiently in the work to take responsibility for it;

(2) that all authors have reviewed the final version of the manuscript and approve it for submission to the ISOPARB journal.

(3) that neither this manuscript nor one with substantially similar content by the authors has been published elsewhere or is being considered for publication elsewhere;

(4) that the manuscript has been submitted with the full knowledge and approval of the institutions or organizations given as the affiliation(s) of the author(s);

(5) that the authors have informed the editor in a cover letter and in the manuscript itself of any conflicts of interest; and

(6) that the corresponding author affirms the manuscript to be an honest and transparent account of the study being reported.

In line with ICMJE standards, the criteria for authorship are as follows:

(1) Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND

(2) Drafting the work or revising it critically for important intellectual content; AND

(3) Final approval of the version to be published; AND

(4) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

3. CLINICAL TRIALS AND REVIEW ARTICLES

Clinical trials

Submission of clinical trials must include reference to ethics approval (or explanation of why ethics approval was not received). Authors must consult the CONSORT statement and checklist and submit a CONSORT flow chart as an editable figure in Word/PowerPoi ntformat.

The clinical trial registration is preferable and information should be included at the end of the abstract of the submitted manuscript.

Review articles

Reviews based on the recent and relevant subjects of clinical interest should be considered.

4. LAYOUT OF MANUSCRIPTS

Manuscript text should be in English (US spelling), double-spaced, font size 12, in Arialmmes New Roman font.

First page

The first page of the manuscript should contain the following: (1) title; (2) full names of authors (6 maximum, although listing more authors may be considered on an individual basis if authorship requirements have been met and a request has been included in the cover letter); (3) affiliations of authors (i.e. department, section or unit of

an institution, hospital or organization, city, and country (4) full contact details (postal address. email address) of the corresponding author ; (5) a list of up to 8 keywords for indexing and retrieval:

Footnotes linking author names to affiliations should be listed as 1,2,3 etc..

The first page should also list the type of article: Clinical Article; Brief Communication: or Review Article.

Abstract

Clinical Articles

A structured abstract not exceeding 200 words is required for all full-length clinical articles. It should contain all and only the following headings: Objective; Methods; Results; and Conclusion.

The Objective reflects the purpose of the study: that is, the hypothesis that is being tested. The Methods should include the setting for the study, the participants (number and type), the treatment or intervention, and the type of statistical analysis. The Results include the outcome of the study and statistical significance, if appropriate. The Conclusion states the significance of the results.

Review articles

An abstract not exceeding 200 words is required for all review articles.

Narrative reviews require an unstructured abstract. Systematic review articles should have a structured abstract with the headings; Background; Objectives; Search strategy; Selection criteria; Data collection and analysis; Main results; and Conclusions.

Brief communications

Brief communications should not include an abstract.

Main text

In full-length articles, subject matter should be organized under the following headings, with no subheadings: Introduction; Materials and methods; Results; Discussion; Acknowl edgments; Conflicts of interest; and References.

Brief communications should not have any headings separating the text.

Clinical articles

The main text of clinical articles should not exceed 2500 words, excluding the first-page information, abstract (no more than 200 words), author contributions, acknowledgments, Conflicts of interest, references (no more than 25), figure legends, and tables and figures. Please include the word count in the cover letter and on the first page of the manuscript.

Review articles

Review articles should have no more than 3000-3500 words in the main text and 40 references. Please include the word count in the cover letter and on the first page of the manuscript.

Brief communications

Brief communications should be no more than 400 words, excluding the first-page information, synopsis, keywords, author contributions, acknowledgments, conflicts of interest, references, figure legends, and tables and figures. There should be no more than 4 references and no more than 1table or 1figure.

Power calculations, statistics, and reporting of numbers.

Power calculations

Where appropriate (e.g. for clinical trials), power calculations should be performed as part of the study design, and a statement providing the power of the study should be included in the Materials and Methods. Authors should state how the power calculation was determined, including what type of difference the calculation was powered to detect and on what studies the numbers are based.

Statistics

The statistical tests used and the significance level set should be listed in the methods for all studies that employed statistical analysis. Information regarding the statistical software programs used should be included in the methods: for example, "SPSS version 20 (IBM, Armonk, NY, USA)." This information should not be included in the reference list.

P values should be provided where calculated. The largest P value that should be expressed is $P>0.99$. The smallest P value that should be expressed is $P<0.001$.

For measures of effect (e.g. relative risks, risk ratios, odds ratios), authors should also report confidence intervals (e.g. 95%) so that the precision of the effect estimate can be assessed.

5. Ethics approval and informed consent

Studies of patients, patient records, or volunteers require Ethics Committee approval and informed consent.

Ethics approval

Include a statement in the methods that the research protocol was approved by the relevant Institutional Review Board or Ethics Committee before the study began; if such approval was not needed/obtained, include an explanation. Authors must provide copies of the appropriate documentation if requested.

Informed consent

Include confirmation in the methods that all human participants gave written informed consent before the study began; if consent was not needed/obtained, include an explanation. Authors must provide copies of the appropriate documentation if requested.

6. Acknowledgments

Sources of funding should be acknowledged by the author(s), along with the names of individuals who do not fulfil the criteria for authorshi p, but who have made a substantial contribution to the manuscript.

7. Confllcts of Interest

A conflict-of-interest statement must be included in the cover letter and before the reference list in the manuscript. It should list any relationships (for any author) that may be deemed to Influence the objectivity of the paper and its review, or state that no such relationships exist. Commercial associations, either directly or through immediate family, in areas such as expert testimony, consulting, honoraria, stock holdings, equity interest, ownership, patent-licensing situations or employment that might pose a Conflicts of interest should be stated. Conflicts for other reasons, such as personal relationships or academic competition, should also be stated.

8. References

The number of references should not exceed 25 for clinical articles, 40 for review articles, and 4 for brief communications; in general, they should be limited to the past decade. They must be numbered and listed as they are cited in the article, using Index Medicus abbreviations for journal titles. Cite the names of all authors when there are six or fewer; when there are seven or more, list the first three authors followed by "et al." Include the volume number.

Journal article

[1] Vellacott ID, Cooke EJ, James CE. Nausea and vomiting in early pregnancy. Int J Gynecol Obstet. 1988;27:57-59 .

Book

[2] Speroff L, Glass BH, Kase NG. Clinical Gynecologic Endocrinology and Infertility. Baltimore: Williams and Wilkins; 1982.

Chapter in a book

[3] Disaia PJ, Creasman WT. Invasive Cancer of the Vulva. In: Disaia PJ, Creasman WT, eds. Clinical Gynecologic Oncology. St Louis: C.V. Mosby; 1984:214-219.

Web reference

[4] World Health Organization. WHO Recommended Surveillance Standards, Second Edition [WHO website). 1999. http://www.who.int/csr/resources/publications/surveillance/whocdscsrisr992.pdf.

Text references can be indicated by Arabic numerals in superscript. abc[1]

Tables

Each table should be titled, numbered (with Arabic numerals), and placed on a separate page after the reference list (not embedded within the main text).

All tables must be cited in numeric order in the main text as "Table 1" etc.

Footnotes to tables should be listed as a, b, c etc.

9. Figures and photographs

Figures and photographs should be submitted as jpg format. CONSORT flow charts should be created and submitted as editable Word/ Power Point files. All figures must be cited in numeric order in the main text as "Figure 1" etc.

Figure permission

All authors wishing to use figures (or any material) that have already been published must first obtain the permission of the original author and publisher and/or copyright holders, in addition to giving precise reference to the original work. This permission must include the right to publish in electronic media. Confirmation should be included in the cover letter (the actual permission correspondence from the copyright holder does not need to be submitted).

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If photographs or videos of identifiable people are used, authors must obtain and submit a signed statement of informed consent from the identifiable person(s) or their next of kin. Authors should not try to conceal identity with black bars over eyes etc.

9. Drugs

Give generic names of all pharmaceutical preparations and, where appropriate, include the trade name and manufacturer's name and address. Review drug names and dosages with care. The author is responsible for all recommended dosages.

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Plagiarism entails the "use or close imitation of the language and thoughts of another author and the representation of them as one's own original work." Self-plagiarism, a form of misconduct in which an author reuses his/her previously written text, data, or ideas, wholly or in part, without indicating previous dissemination, will also be considered plagiarism. Verbatim copying of sentences, even if a citation is provided (unless the sentence appears in quotation marks), is considered to be plagiarism.

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If your paper is accepted for publication, you will receive an email informing you of this decision.

12. Copyright

Once accepted and published, all copyright will belong to ISOPARB. No part of the article could be published without permission. All disputes are subjected to Indian Jurisdiction.

13. It is desirable that the, author(s) submitting article in IJOPARB be a member of ISOPARB.

ISOPARB AFG

10 –11 March 2018

Dear Friends

It is our proud privilege to invite you to the **ISOPARB - AFG Annual Conference 2018** (Annual Conference of the Indian Society of Preinatology and Reproductive Biology and the Association of Fellow Gynaecologists, Mumbai), to be held on **10th March 2018** at **Raddisson Sarovar,** Goregaon (West) & on **11th March 3018** at **Hotel The Leela,** Sahar, Andheri (East), Mumbai. During the course of the event, we will have Workshops, Orations, Keynote addresses, Public Forum and also a Banquet & Cultural program.

The Scientific program has been designed with lot of efforts taking into consideration Clinicians, Teachers as well as students. The conference will provide you an ideal opportunity to hear and interact with distinguished faculty from Mumbai and other parts of India.

We take immense pleasure to invite you to participate in this 2 Day Academic event. The Organising Committee will leave no stone unturned in making your participation a pleasant, memorable and fruitful experience.

Looking forward to welcome you at Mumbai.

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