

IFS CONVERSATIONS

Volume 13 & 14 (2020-21)



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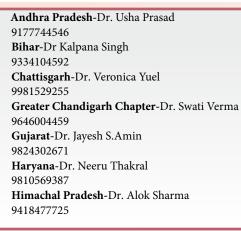
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MESSAGE FROM THE **PRESIDENT DESK**



DR SUDHA PRASAD President - IFS

Dear Friends.

Greeting to all of you from the entire executive team 2020-22 of Indian fertility society First of all I would like to congratulate the entire team of IFS for working so hard during the difficult time of covid .

The sole purpose of getting these conversations is to showcase the various recent academic activities conducted by our extremely enthusiastic and committed members spread over 28 chapters across India and abroad .The topic of this conversation is PCOS

PCOS is a very prevalent reproductive disorder in women. It leads to hormonal imbalance in the reproductive age group which affects the woman's ability to conceive, causes irregular periods and miscarriages, causes acne and facial hair growth due to relative increase of male hormone and is associated with obesity and insulin resistance in a large percentage of women.

The management of PCOS does not end with the treatment of acne or hirsutism or irregular periods or infertility, it goes much beyond that. We need to prevent metabolic syndrome, and it's associated cardiac problems, cancer of the uterus, severe obesity etc. which commonly occur in PCOS women at an older age. It is extremely important to make a timely and correct diagnosis of PCOS when girls or women come to us with their symptoms, and convince them to have a regular follow up; by self-determination and self-discipline in their own life style, women themselves, can overcome this difficult situation, and live healthy lives.

While we have come a long way from PCOS being a poorly understood condition. in 1990 to present day, there is still much to learn and accomplish. The aim of this conversation is to give our readers an updated knowledge on PCOS. I congratulate the editorial team for their excellent hard work and dedication to plan and prepare this news bulletin and wish all readers a very rewarding and pleasant reading

Warms Regards and best wishes,

Indhe franced

Dr. Sudha Prasad President- IFS

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MESSAGE FROM THE SECRETARY DESK



DR NEENA MALHOTRA

Secretary - IFS

Dear Friends,

It is indeed a privilege and pleasure to address you all on this issue of IFS Conversations. I hope you are all in good health and safe. Our editorial team brings you this IFS conversation dealing with various aspects of PCOS.

I would like to congratulate the dynamic team of IFS who have spent their valuable time in compiling this bulletin, the authors and contributors for their efforts in providing in depth information and keeping us all updated with recent advances in the field .

IFS has been doing excellent work by focussing on academic activities all over the country, helping young faculty to learn from experienced senior members. Indian Fertility Society (IFS) has progressed over the few years with nearly 3400 members and 28 chapters. It is an internationally affiliated organization engaged in training and educating clinicians and embryologists by organizing CME, workshops and seminars.

In this conversation we have dealt with "PCOS'. With the increasing incidence of PCOS and metabolic syndrome, management of insulin resistance is a dilemma for all clinicians, this edition shall throw light on role of insulin sensitizers, dispel myths and clarify its indications in background of available scientific evidence. Hope you all will find it very useful.

Warms Regards and best wishes,

Neena Malholie

Dr. Neena Malhotra Secretary - IFS





MESSAGE FROM THE EDITOR'S DESK





Jt. Editor - IFS

DR. SHWETA MITTAL GUPTA Editor - IFS

Dear Memebers of IFS & all Readers,

Hope you all are keeping safe & healthy.

In this bulletin dedicated to PCOS, one of the most commonly faced situation, yet difficult to treat, we bring you a variety of academic bonanza.

"The role of vitamin D deficiency in pathogenesis of PCOS" as well as modulator of insulin deficiency has been elucidly written & discussed by Dr. Prateebha Makhija.

Prof. Dr. Rekha Ratnani has described a **"case report of endometrial carcinoma in a young PCOS women"** and further elaborated on how to preserve fertility in such cases.

Dr. Paulami Dey in detail has described all the **"Different phenotypic presentation of PCOS and the phenotypic approach"**.

Lastly Dr. Neha Mathur wrote about **"Oocyte quality in PCOS women undergoing IVF"**, a yet another important aspect which needs to be understood and dealt with.

Hope all our readers will find all the articles interesting with many important practising tips. May this season bring in a lot of joy and happiness to all. Editorial team.



INVITED ARTICLES

Emerging Concept: Role of Vitamin D Deficiency in the Pathogenesis of PCOS



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Introduction

Polycystic ovary syndrome (PCOS), characterized by clinical and biochemical evidence of hyperandrogenism, menstrual irregularities, ovulatory dysfunction, and polycystic appearance of the ovaries, is the most common endocrinopathy of reproductive-age women [1-4]. Insulin resistance (IR) has emerged as being central to the path physiology of PCOS [5, 6], with resulting hyperinsulinemia as a mechanism contributory to both the ovulatory dysfunction and hyperandrogenism that characterize this disorder [6]. The endocrine and metabolic milieu of PCOS places this population at enhanced lifetime risk for a spectrum of morbidities including poor reproductive outcomes, type 2 diabetes mellitus (DM), cardiovascular disease (CVD), mood disorders including depression and anxiety, as well as endometrial cancer [1, 2].

The role of vitamin D in calcium homeostasis and in the maintenance of skeletal health is well recognized [7]. In addition, a growing body of evidence relates deficiency of vitamin D to a number of nonskeletal sequelae, including obesity, DM, dyslipidemia, hypertension, inflammation, CVD, autoimmune disease, and cancers [7-12]. Genes that are critical for glucose and lipid metabolism are recognized to be downstream targets of vitamin D signaling [7-9]. A relationship between vitamin D deficiency and IR has additionally become apparent in recent years. Given that a role of calcium in oocyte activation and maturation is well understood [13-15] and since PCOS is a state of follicular developmental arrest, abnormalities in vitamin D metabolism and action could be theorized to be linked to the pathogenesis of PCOS. Thus, in PCOS, vitamin D deficiency has emerged as a plausible mechanism to explain some of the metabolic and endocrine features of PCOS. Indeed, multiple studies, observational as well as randomized controlled trials, have explored the relevance of vitamin D in PCOS [16-28]. Accruing data suggest low levels of vitamin D in PCOS [17, 19, 21, 28], and vitamin D deficiency is shown to be linked to PCOS pathophysiology through its associations

with obesity, insulin resistance,

hyperandrogenism, dyslipidemia, inflammation, as well as features of depression and risk for DM and CVD. while impaired folliculogenesis, steroidogenesis, and reproductive compromise are well described in the animal models of vitamin D deficiency, a relevance of vitamin D for human reproductive biology, however, is less well understood. Herein we attempt to provide an overview of our current understanding on the plausible relevance of vitamin D in the pathophysiology of PCOS.

Vitamin D: Mechanism of Action (Fig. 1.1)

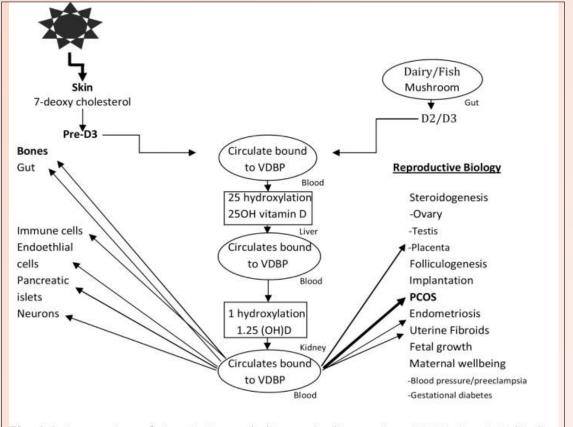


Fig. 1.1 An overview of vitamin D metabolism and salient actions. VDBP vitamin D-binding protein, D2 ergocalciferol, D3 cholecalciferol.

Humans are primarily dependant on endogenous cutaneous synthesis of vitamin D through exposure to solar ultraviolat B (UVB); dietary sources account for less than 20 % of daily requirements of the vitamin. Exposure to solar UVB converts dehydrocholesterol in the skin to previtamin D₃ which is rapidly converted to vitamin D₃ (cholecalciferol). Dietary vitamin D gets incorporated in the chylomicrons and gets transported via the lymphatics to the circulation. Circulating vitamin D is transported to the liver where it undergoes the first step of activation wherein vitamin D-25-hydroxylase catalyzes the conversion to 25-hydroxy vitamin D (25(OH)D), the major circulating form that reflects the overall vitamin D status. Final activation of 25(OH)D occurs in the kidney as well as at target cell level, via 1 -alpha (α-hydroxylase to 1,25dihydroxyvitamin D (1,25(OH)2D), the active vitamin D metabolite that promotes intestinal calcium absorption through its interaction with the cognate vitamin D receptor (VDR). Vitamin D acts as a transcription factor via signaling through the nuclear VDR-retinoic acid x-receptor (VDR-

RXR) complex [7] and exerts actions across a host of tissues, including the skeleton, pancreas, parathyroid glands, and even ovary [29, 30]. Serum levels of calcium, phosphorus, parathyroid hormone (PTH), as well as fibroblast-derived growth factor-23 (FGF-23) are recognized modulators of 1α -hydroxylation of 25(OH)D [7].

Obesity, PCOS, and Vitamin D

There is increased prevalence of body mass excess in women with PCOS as compared to age-matched controls [1, 6, 31, 32]. A recent meta-analysis identified women with PCOS as having an increased prevalence of being overweight, obese, and centrally obese compared to non-PCO controls [32]. An inverse relationship between circulating 25(OH)D levels and parameters of body mass excess such as body mass index (BMI) and waist circumference (WC) is well described across populations including women with PCOS [17, 19-21, 23, 25, 33, 34]. Possible rationale for the lower circulating 25(OH)D levels in obesity may include increased sequestration tendency of obese individuals to seek sunlight [35].

depression in the non-PCOS populations. Women

burden; although the exact underpinnings remain

acne, alopecia, hirsutism), menstrual irregularity,

with PCOS carry a substantial psychological

unclear, altered physical appearance (obesity,

and difficulties in conceiving are recognized as

potential contributors to the prevalent issues of

depression, anxiety disorders, body image

encountered in women with PCOS [61-64].

Reproductive Success in Women with PCOS

Vitamin D as essential for procreative "success,

implantation [65-67). Vitamin D is recognized to

with recognized effects on folliculogenesis,

spermatogenesis, steroidogenesis, and

dissatisfaction, and sexual dysfunction

Vitamin D: Potential Implications for

Vitamin D: A Modulator of Insulin Resistance in PCOS

Insulin secretion is a calcium-dependent process [36, 37] and IR and compensatory hyperinsulinemia are well described in the setting

of PCOS [5, 6]. The precise mechanism of action whereby vitamin D influences insulin signaling appears to involve genomic stimulation of the insulin receptor mRNA via VDR signaling [38, 39]. Vitamin D signaling appears to promote insulin synthesis and release, enhance insulin receptor expression, and also inhibits pro-inflammatory cytokines that are recognized to play a role in the pathogenesis of IR [40]. The latter mechanisms may explain the observed associations between vitamin D deficiency with impaired glucose and insulin metabolism [41-44] and the recognized high prevalence of hypovitaminosis D in populations with type 2 DM [45-47]. Similar trends and associations have been described in women with PCOS in whom limited data suggest similar relationships between IR and vitamin D deficiency in women with PCOS [20, 21, 24, 25].

Vitamin D: Relationship with Hyperandrogenemia of PCOS

Vitamin D status can be hypothesized to modulate circulating androgen levels through interactions with sex hormone-binding globulin (SHBG), and PTH. Recognized as a modulator of circulating levels of free androgens, the hepatic SHBG is the dominant carrier protein that binds circulating androgens including testosterone and androstenedione and, hence, minimizes percentage of free androgen available to act at the target tissue; a decline in SHBG levels, as seen in states of IR, is associated with increase in circulating free androgen levels and, hence, worsening features of hyperandrogenism. Serum levels of 25(OH)D are shown to correlate positively with SHBG levels [48]. Conversely, hyperinsulinemia, as in PCOS, promotes hyperandrogenism through inhibition of hepatic synthesis of SHBG [49]. Hirsute PCOS women were reported to have significantly lower 25(OH)D levels compared to the non-hirsute. Serum PTH levels are intimately related to vitamin D status, and are known to be higher in the obese individuals, secondary to vitamin D deficiency [50-54].

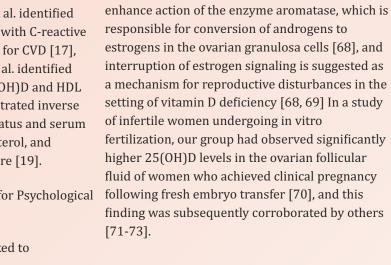
Vitamin D: Implications for Ovarian Physiology and Relevance for PCOS

Animal studies have established the role of calcium in oocyte activation and maturation [13-15]. An increase in intracellular free calcium is responsible for progression of oocyte meiosis [13-15]. Given the known association of PCOS with ovulatory dysfunction [55], studies were conducted to investigate the contribution of altered calcium homeostasis in PCOS pathophysiology [16, 19, 26]. Addition of vitamin D and calcium to along with metformin is more effective in correcting menstrual disorders and follicular growth than either metformin or calcium and vitamin D alone.

Vitamin D: Potential Implications for Cardiovascular Health in PCOS

Epidemiological data identify vitamin D deficiency as a risk factor for enhanced cardiovascular morbidity and mortality [56-58]. Vitamin D receptors are located in the vascular smooth muscle [59] and endothelium [60], and inflammation, dyslipidemia, hypertension, coronary artery disease, cardiac failure, and accelerated carotid atherosclerosis have been described in association with vitamin D insufficiency across populations [56-58]. Limited data are additionally available on the relationship between vitamin D status and CVD risk in the PCOS population [17, 19, 21]. Li et al. identified 25(OH)D levels to relate inversely with C-reactive protein (CRP), a known risk factor for CVD [17], whereas both Li et al. and Hahn et al. identified positive correlations between 25(OH)D and HDL levels [17, 21]. Wehr et al. demonstrated inverse correlations between vitamin D status and serum levels of triglycerides, total cholesterol, and systolic and diastolic blood pressure [19].

Vitamin D: Potential Implications for Psychological Well-Being in Women with PCOS



Vitamin D deficiency has been linked to

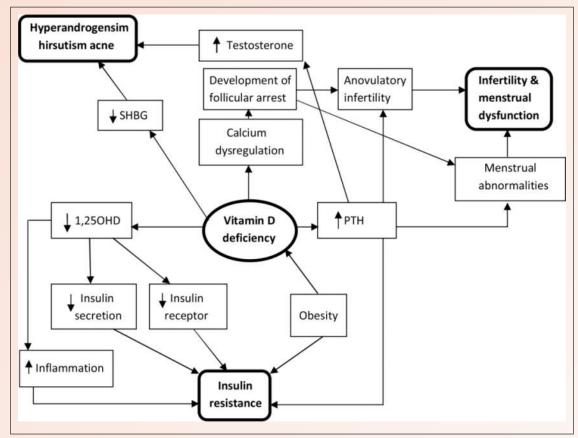


Fig. 1.2 The role of vitamin D deficiency in the pathology of PCOS. Adapted from Thomson RL, Spedding S, Buckley JD. Vitamin D in the aetiology and management of polycystic ovary syndrome. Clin Endocrinol 2012;77:343–350, with permission from John Wiley and Sons. © 2012 Blackwell Publishing Ltd

Summary

A vast body of literature links low vitamin D status to obesity, insulin resistance, menstrual irregularity, depression, and increased CVD risk (Fig. 1.2), and a growing body of literature suggests a relevance of vitamin D insufficiency for the pathophysiology of PCOS. A need for appropriately powered doubleblind randomized controlled trials is underscored by the currently existing data so as to definitively address if vitamin D insufficiency may be a modifiable mechanism in the pathophysiology of PCOS, and if normalization of vitamin D status could mitigate the endocrine, metabolic, and clinical stigmata of PCOS.

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Case Report- Endometrial Carcinoma In A Young Pcos Female With A Successful Pregnancy Outcome Secondary To Medical Management And Art Technique



PROF DR REKHA RATNANI MS(SURG)MD(OBGYN) Fellowship in ART,FMAS Senior laparoscopic surgeon & IVF expert INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a common endocrinopathy characterized by oligo/anovulatiaon and elevated circulating androgens or evidence of hyperandrogenism after,all known potential causes have been excluded. These endocrinologic and metabolic abnormalities expose endometrium to unopposed estrogen for long durations causing infertility and endometrial disorders including hyperplasia and cancer. Endometrial carcinoma in young nulliparous women poses a challenge for the diagnosis and management. This case report illustrates that young subfertile women with PCOS are at risk of developing endometrial carcinoma and medical management combined with ART techniques can be rewarding.

CASE REPORT

A 25 year old married woman presented 2 years after her marriage with irregular cycles.She was known case of PCOS but never prescribed any medication.Her pelvic sonography revealed 3.5 cm thickness of endometrium.On MRI study, uterus was found bulky, both ovaries were polycystic and a heterogenous, non enhancing intermediate signal intensity mass within the expanded endometrial cavity measuring 4.5cm x3.3 cmx3.4cm with peripherally situated T2 hyperintensities resembling endometrial polyp was reported.She was posted for hysteroscopic resection.Intraoperatively it was seen as bunch of multiple polyps appearing more like retained products of conception due to its friable nature.Histopathologically it was reported as a case of well differentiated endometrioid adenocarcinoma FIGO grade 1.She was refered to Tata memorial hospital and after ruling our myometrial invasion in fresh MRI,she was put on medroxyprogesterone which was gradually

increased to 80 mg TDS after all her requires blood tests were within normal range.Three monthly hysteroscopic biopsy was planned and response was assessed based on histopathological findings.After 6 months, dose was decreased to 40 mg TDS and LNG -IUS was inserted.Patient had massive weight gain and mood swings as side effects.After 9 months therapy ,biopsy showed mild atypia hence dose was tapered and after full 18 months ,she showed no feature of atypia and was only on LNG-IUS.Her FSH was 0.23 ,LH was 0.12, and E2 was 85.0 Decision was taken to hyperstimulate her with recombinant FSH in step up protocol.She made 24 follicles with 150 iu and leupride was used as trigger.OPU was smooth and 20 good quality oocytes were in hand.ICSI was done and 13 embryos of grade 1-2.5, with 6-8 cells were frozen and FET was planned.She was put on 8mg estradiol hemihydrate from day 3 of next cycle and later dose was increased to 12 mg daily.Her endometrium failed to grow beyond 6mm and instead of trilaminar pattern it showed fluid collection in the endometrial cavity.Decision was taken for hysteroscopy and it revealed suspicious thickened endometrial patches from which biopsy was taken. It revealed complex hyperplasia with mild atypia.Procedure was abondoned and she was refered back to Tata Memorial hospital.She was put on LNG-IUS and kept on follow up.Patient was councelled and decision for surrogacy taken.FET was done and we got success in third attempt .Beautiful twin babies were born in corona times bringing lot of happiness in her life after four years of struggle with the disease.

DISCUSSION

An association between polycystic ovary syndrome (PCOS) and endometrial carcinoma was first suggested in 1949. However, obesity, hyperinsulinemia, and hyperandrogenism, which are also the features of PCOS, are risk factors for endometrial carcinoma. Lack of clinical suspicion and reluctance to do an endometrial evaluation may delay this rare diagnosis of endometrial cancer in young women. One of the welldocumented effects of estrogen on the endometrium is its growth-stimulating effect, which can produce a progression of changes from benign proliferation to atypical hyperplasia and adenocarcinoma. Anovulation due to unopposed estrogens contributes to this situation. In a normal menstruating women, progesterone induces regular sloughing of the endometrium, thereby removing endometrial tissue that might otherwise become hyperplastic. Furthermore, progesterone can reverse various degrees of hyperplasia and early stages of adenocarcinoma to normal endometrial histology by causing suppression of endometrial glandular growth, through stromal decidualization and leukocytic infiltration to glandular atrophy and stromal focal necrosis. Due to prolonged treatment, connective tissue fibers increase to some degree and may be accompanied by endometrial fibrosis and calcification. Clinical and histological data have demonstrated that all these changes, including fibrosis and calcification, return to normal in a short period after discontinuing the treatment.

Adenocarcinoma of the endometrium is a morbid condition in women under 40 years of age with an incidence of 25%. The disease is often advanced when diagnosed, thereby depriving the woman of the option for fertility sparing conservative approach. In young women with menstrual abnormalities and polycystic ovarian disease and/or infertility, an endometrial evaluation should be performed. Carcinoma endometrium should be kept in mind while evaluating young women with polycystic ovary syndrome for abnormal uterine bleeding. Only strictly selected patients should, therefore, be indicated for longterm progestogen treatment and careful evaluation before and after treatment should be performed.

The standard treatment for endometrial carcinoma is total abdominal hysterectomy with bilateral salpingo-oophorectomy. In young women with low histological grade and early stage of the disease, conservative hormonal therapy has been tried with close follow-up. There are reports of high-dose medroxyprogesterone acetate (600 mg/ day) treatment with endometrial evaluation in every 3 months to assess the effects of medication but studies suggest that 200mg/day gives similar control. If the response is not satisfactory, hysterectomy is advocated. For a successful outcome following conservative approach, a strict clinical staging in the form of physical examination, Hysteroscopy and imaging with ultrasound, CT or MRI, and a cautious evaluation of histological grading by a pathologist are required. Women with endometrial cancer who want fertility preservation should be counseled regarding the possible risk of advanced disease if surgical therapy is delayed. Nevertheless these young patients should be given fair trial of conservative management and if recurrence occurs, the disease free window should be used for hyperstimulation of ovaries and ovum pick up.While preparing the endometrium for FET,keep suspicion index very high for recurrence and counsel the patient for the need of surrogacy for successful outcome.Multidisciplinary approach while treating such young patients help in formulating the treatment plan with successful outcome.DO NOT FORGET that conservative therapy is feasible only in carefully selected young women with endometrial cancer. Recurrence rates are high as quoted by long-term observational studies even after pathologically complete remissions. Therefore, close follow-up is recommended.



Endometroid carcinoma



Successful twin delivery with LNG IUS in situ

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POLYCYSTIC OVARIAN SYNDROME-THE PHENOTYPIC APPROACH



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INTRODUCTION-

Polycystic ovary syndrome (PCOS) was first described in 1935 by Stein and Leventhal in a case series of seven women with amenorrhea, hirsutism, obesity, and ovaries with a gross polycystic appearance. Polycystic ovary syndrome is a common (4% to 21%) disorder among reproductive age women. Depending on diagnostic criteria, PCOS's prevalence was approximately 4%–6.6% in accordance with NIH 1990 criteria and approximately 4%–21% when Rotterdam 2003 criteria were applied.

Polycystic ovarian syndrome (PCOS) is a highly prevalent disorder affecting multiple aspects of a women's overall health, with long-term effects that transcend well beyond the reproductive age. The term "polycystic ovarian syndrome" does not fully or accurately reflect the complexity of this disorder given its very broad spectrum of clinical manifestations and associated morbidities. Patients with PCOS demonstrate reproductive abnormalities, marked insulin resistance, increased risk for type 2 diabetes mellitus, coronary heart disease, atherogenic dyslipidemia, cerebrovascular morbidity, and anxiety and depression. If pregnant, these women have substantially increased odds for the development of gestational diabetes, pre-eclampsia, fetal macrosomia, small-for-gestational age infants, and perinatal mortality.

Hospital admissions for women with PCOS are twice as high as for the general population. Over the last several decades, significant efforts have been made to classify PCOS; however, global consensus regarding a PCOS criterion remains controversial. Unfortunately, existing epidemiologic and/or basic research data have not been sufficient in providing the foundation needed to derive an evidence-based definition of the syndrome. Currently proposed criteria are predominantly based on expert opinion, thereby serving as a point of disagreement among researchers: some experts assert it is a disorder predominantly of androgen excess, whereas others believe that it has a broader spectrum of

presentation. Some progress has been achieved more recently with the introduction of a novel phenotypic approach to the diagnosis. A phenotypic approach to classifying PCOS avoids the drawbacks of currently existing criteria, which may be interpreted as "lumping" all phenotypes together, while providing a simple diagnostic instrument and avoiding the need to decide between multiple different PCOS definitions. In the present article we review the controversy around the PCOS definition; the prevalence of the disorder on the basis of these definitions; the distribution and associated morbidity of the PCOS phenotypes; and important phenotypic differences in PCOS according to population source and referral bias

GLOBAL PREVALENCE OF PCOS

Understanding the global prevalence and phenotype of PCOS is important, considering that geographic factors and ethnic/racial variations can shape the clinical presentation of the syndrome. The first studies to determine prevalence in a medically unselected (unbiased) population were initiated by Azziz and colleagues, who reported PCOS prevalences ranging from 4% to 6.6% using the NIH 1990 criteria among unselected reproductive-age women residing in the southeastern region of the United States. The prevalence of PCOS among different geographic regions ranges from 5% to 10% according to NIH 1990 criteria; from 10% to 15% according to the AE-PCOS 2006 criteria, and from 6% to 21% when the ESHRE/ASRM 2003 criteria were applied. Greater estimates of PCOS prevalence with the Rotterdam 2003 and AE-PCOS 2006 criteria are largely attributed to their more expansive definition and inclusion of additional phenotypes, compared with NIH 1990 diagnostic criteria. and the PCOS phenotypes were defined.

LIMITATATIONS OF STUDIES ASSESSING PCOS PREVELANCE-

1.**UNDER REPORTING-** assessment of the PCOS phenotype is a complex multistep process, which requires multiple clinical and laboratory assessments, pelvic ultrasound, and possibly several visits for some subjects.

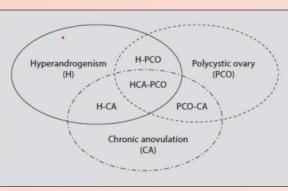
2.INCOMPLETE DATA

3.HETEROGENEITY- Several limitations in the definition of the outcomes (PCOS and its compounds) leading to heterogeneity in prevalence estimates (e.g., the lack of population-defined normative ranges), androgen measures based on total T only, use of insensitive/inaccurate circulating androgen assays, involvement of multiple observers for the evaluation of hirsutism with unknown interobserver variation, effect of transvaginal ultrasound transducer frequency on the cut-off value for antral follicle count and the absence of standardization in the evaluation for the exclusion of mimicking disorders.

However, despite all discussed limitations, the prevalence of PCOS by the NIH 1990 criteria is relatively similar among different ethnic and geographic populations, possibly suggesting that, at least for the "classic" PCOS phenotype, the disorder seems to have originated before the separation of Homo sapiens into racial groups.

PCOS PHENOTYPES

The presentation of PCOS can be subdivided into four phenotypes: **phenotype A:** HA + OD + PCOM; **phenotype B:** HA + OD; **phenotype C:** HA + PCOM; and **phenotype D:** OD + PCOM."**Classic**" **PCOS**



(Phenotypes A and B)

Women with "classic" PCOS (phenotypes A and B) are associated with more pronounced menstrual dysfunction; increased insulin levels, higher rates of insulin resistance, and risk for metabolic syndrome; body mass index and prevalence of obesity; and more severe forms of atherogenic dyslipidemia, increased risk of hepatic steatosis as compared with women diagnosed with non-classic or nonhyperandrogenic PCOS phenotypes (phenotypes C and D).

The highest antimullerian hormone levels are also found in patients with classic PCOS. Menstrual cycle pattern is also more irregular in these women as compared with phenotype D but seems to normalize with ageing.

"Ovulatory PCOS" (Phenotype C)

Patients with "ovulatory PCOS" generally demonstrate intermediate levels of serum androgens, insulin, atherogenic lipids, hirsutism scores, and prevalence of metabolic syndrome, as compared with patients with "classic" and the nonhyperandrogenic PCOS phenotypes.

Higher socioeconomic status is related to a higher prevalence of the ovulatory phenotype. Differences in ovulation patterns between the social groups could in part be explained by differing insulin levels and fat tissue distribution.

"Nonhyperandrogenic PCOS" (Phenotype D)

In the majority of studies, patients with nonhyperandrogenic PCOS had the mildest degree of endocrine and metabolic dysfunction and the lowest prevalence of metabolic syndrome as compared with healthy controls. These women had lower LH to FSH ratios, lower total and free T levels, and higher sex hormone-binding globulin levels, as compared with subjects with classic PCOS. Besides that, the number of women with regular cycles alternating with irregular cycles is highest in women with phenotype D.

Distribution of PCOS Phenotypes

Understanding the distribution of PCOS phenotypes is essential in defining the epidemiology of PCOS in a population. Overall, published data indicate that more than half of PCOS patients identified within the clinical setting demonstrate phenotype A, whereas the other three phenotypes (i.e., B, C, and D) have almost equal prevalence. Overall, it seems that the classic form of PCOS (i.e., phenotypes A and B) constitutes approximately two-thirds of the total of PCOS patients identified within the clinical setting. Few data exist regarding the distribution of phenotypes in women with PCOS identified in medically unbiased (i.e., unselected) populations, which would more accurately reflect the distribution of phenotypes in PCOS in the "natural" state. The few studies suggest that approximately two-thirds of PCOS patients identified among unselected populations could be

classified as having phenotypes B and C, whereas phenotype A and phenotype D are almost equally prevalent. Interestingly, these early data suggest that the least prevalent phenotypes are the most (phenotype A) least (phenotype D) metabolically severe phenotypes.

Comparison of the Different PCOS Phenotypes Based on Clinical Metabolic, and Hormonal Profile, and their Response to Clomiphene

Height, weight, BMI, waist circumference-Phenotype A has significantly higher weight and BMI (P < 0.05) in comparison to phenotypes C and D. Although phenotype B has higher weight and BMI than phenotypes C and D, but the results are not statistically significant (*P* > 0.05). However, there is no significant difference noted in the waist circumference, waist-hip ratio (P > 0.05). **Both clinical and biochemical** hyperandrogenism (Ferriman-Gallwey score, total testosterone, and androstenedione levels) Significantly more in phenotype A as compared with the phenotype C and D. Although phenotype B has higher Ferriman-Gallwey score, total testosterone, and androstenedione levels than phenotypes C and D but the results are not statistically significant (*P* > 0.05).

Menstrual irregularities (cycle length >60 days) significantly more common in phenotype A as compared with phenotype D (P = 0.000). **Ovarian reserve (mean AFC, mean ovarian volume, AMH)** significantly higher in phenotype A (P < 0.05) as compared to the phenotypes B and D. Although phenotype A has a higher ovarian reserve than phenotypes C also, but the results are not statistically significant (P > 0.05).

Fasting insulin and HOMA-IR significantly more in phenotype A as compared to phenotypes B and D (P < 0.05). Phenotype B had higher insulin and HOMA-IR values than phenotypes C and D, but the results are not statistically significant (P > 0.05). **Lipid profile** significantly more deranged in phenotype A with higher LDL and total cholesterol and lower HDL values (P < 0.005) as compared to phenotype D. Although phenotype B has more deranged lipid profile than phenotypes C and D but the results are not statistically significant (P > 0.05).

waist circumference, waist-hip ratio, blood pressure and blood sugar values (fasting, 1-hour postprandial, 2-hour postprandial). FSH, LH, LH-FSH ratio,

17-hydroxyprogesterone (17-OHP) and vitamin D levels no significant difference amongst various PCOS phenotypes (*P* > 0.05).

Clomiphene resistance significantly Higher in full-blown PCOS (phenotype A) as compared to phenotype D .

Referral Bias in Defining the PCOS Phenotype Multiple studies have shown that the difference in the distribution of PCOS phenotype between patients identified in clinical vs. unselected populations suggests that the clinical PCOS cohort may not be truly representative of the disorder in its natural, medically unbiased, state in the general population. The investigators found that the referral cohort of PCOS patients had a higher prevalence of the more severe PCOS phenotypes, greater BMI, more severe hirsutism, and more pronounced hyperandrogenemia, compared with women with PCOS identified in the unselected population. Subjects with PCOS identified in the general population have less severe manifestation of the disorder, higher prevalence of milder

phenotypes, and are different socioeconomically and racially, reflecting the ability to access medical care. Therefore, the use of clinical cohorts for epidemiologic research could possibly produce falsely elevated odds

ratios and pseudo-significant associations.

These data raise important questions *regarding the validity of epidemiologic research using clinical PCOS cohorts*.

SUMMARY-

Despite meaningful limitations of published prevalence studies relevant to sampling and outcome definitions, PCOS prevalence by NIH 1990 criteria remains relatively constant.

Of the various PCOS criteria, NIH's 2012 phenotypic extension of the Rotterdam definition has been shown to be the most convenient approach when conducting research and clinical practice. This approach permits comparisons in epidemiologic studies among different populations and allows researchers to identify high-risk individuals in clinical practice. More epidemiologic data are required among medically unbiased PCOS populations to better understand the natural course of this syndrome, as well as validate any strengths of true associations with comorbid disorders.

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2. Diagnosis, phenotype, and prevalence of polycystic

ovary syndrome

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3.Comparison of the Different PCOS

Phenotypes Based on

Clinical Metabolic, and Hormonal Profile, and their Response to

Clomiphene

Garima Sachdeva, Shalini Gainder, Vanita Suri, Naresh Sachdeva1, Seema Choprafrom http://www.ijem.in on Sunday, September 27, 2020, IP: 27.62.199.110]

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Oocyte Quality in PCOS

Dr Neha Mathur

Polycystic ovarian syndrome is a metabolic disorder affecting the women in reproductive age group. It is a syndrome complex involving hyper androgenicity, irregular menstruation and oligo ovulation. Different criteria viz National Institute Health in 1990 and Rotterdam criteria 2003 are available that define the syndrome(1). PCOS is the most common cause of anovulatory infertility in females(2). Prevalence of the syndrome varies according to diagnostic consensus used, with estimates ranging from 9% according to National Institutes of Health consensus, up to 18% with the Rotterdam consensus(3).

The concept of oocyte quality represents the competence of the cell for development i.e its ability to undergo meiotic maturation, fertilisation and proper embryonic development(4) This competence is acquired during follicular development by cross talks between the follicle and the surrounding granulosa cells. As there is disturbed follicular growth i.e antral follicular growth arrest in polycystic ovarian disease patients, there may be altered microenvironment, especially during controlled ovarian stimulation. Although the pathogenesis of polycystic ovaries is unknown, it is a complex and heterogeneous disorder in which the patients shows marked increase in androgen secretion(5). This hyperandrogenic melieu alters the interfollicular microenvironment, mainly by increasing the reactive oxygen species in the cells thereby leading to aberrant folliculogenesis (6). There are few studies available at present assessing the association between reactive oxygen species (ROS), oxidative stress, and female infertility(7). Oxidative stress is defined as a disequilibrium between the production and neutralization of reactive oxygen species (ROS), which may occur as a result of excess ROS production and/or as a result of deficiency of antioxidant mechanisms.(8) Higher quantity of ROS is found in follicular fluid of PCOS and this affects the overall quality of oocyte and thereby affecting the fertilisation rate(9). Oocyte quality can be evaluated based upon morphology, genetics and OMICS(genomics). Morphologically – Metaphase II oocytes can be evaluated for nuclear maturation and morphology of cytoplasm. Along with this other morphological characteristics like perivitelline space, polar body, granular cytoplasm, smooth endoplasmic reticulum incidence, vacuolization and abnormal zona pellucida have also been observed in patients with PCOD(10). Few studies are conducted in invitro fertilisation and ICSI cycles to compare the effect of hormonal stimulation on patient with and without PCOS. No significant differences were noted in the maturity of the oocytes, oocyte dysmorphism, embryo quality, implantation and pregnancy rates between the patients(11,12). Genetically there has not been any increase in the number of aneuploidies in PCOS patients conceived after IVF compared to non PCOS patients(13).

The genomic and proteomics studies have evolved over the course of time and helped a great deal in solving the microenvironment mystery. There has been studies regarding the overall alteration in the global gene expression(14) to overexpression of Hsp27 during oocyte maturation(15). In spite of repeated studies conducted to assess

the difference in oocyte quality of normal patient with PCOS patient, no significant criteria has been developed till now. One important consideration in this is the fact that PCOD is itself a cause of infertility independent of sperm and tubal causes, thus these patients may need controlled ovarian hyperstimulation(COH) more frequently that the others. In cases of artificial reproduction, due to COH, the oocyte quality may be altered due to vascular and inflammatory factors leading to recruitment biases in studies.

Thus to conclude there is little clinical information regarding the poor oocyte quality in PCOD patients. PCOD being a metabolic disorder affects the body at different levels thus creating a hostile environment and hampering the reproductive ability of the patient.

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Wake up Insulin- Be sensitive Life me ENERGY KA TADKA lagao. Here is the acronym for controlling PCOD/PCOS. We all know it's a disease which can be controlled but not cured. This acronym ENERGY is to summarise the Lifestyle modifications and anti inflammatory food.

E- Educate, Exercise - strength training

N- No to junk food/ Laziness

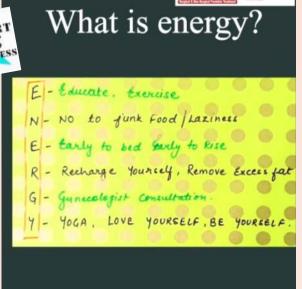
E- Early to bed, early to rise- as per body's natural circadian rhythm, Eat right, add Haldi, Cinnamon, plant based diet

R- RECHARGE yourself, Remove excess fat/

Reduce WC and HC

G- Gynecologist consultation

Y- Yoga, Love Yourself, Believe in yourself.





INDIAN FERTILITY SOCIETY STATEMENT (14 April, 2020)

COVID-19 & FERTILITY RECOMMENDATIONS FOR CLINICS & PATIENTS

For Details Visit www.indianfertilitysociety.org

IFS ACTIVITIES VIBRATE MEETINGS



IFS ACTIVITIES VIBRATE MEETINGS

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Genetics in infertility- How much should a clinician know?	OHSS- Prevention and Troubleshooting	Counselling in ART
Dr. M VENUGOPAL Brown Brow	Difference 12 th Jan, 2021 Times 3:00pm IST.	Dr. Poonam Nayar Consultant Clinical Psychological support Chairperson: Pre-Congress workshop on Chairperson: Pre-Congress workshop on Chairperson: Conference of IFS, December 2017-2020 International Conference of IFS, December 2017-2020 Inters: 20" Jan 2021 Ittmer
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Certificate Per Lecture Supported by an unrestricted educational grant from Zyclus dedicated[ife Naturogest SR' Briogyns ZyhMG HP 00	Supported by an unrestricted educational grant from	Supported by an unrestricted educational grant from The makers of The makers of Naturogest SR' Briogyna ZyhMG HP © ©
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Recurrent Pregnancy Loss: Evidence-based management - Case file	Handling hyper-stimulation in PCOS	Reducing TIME and INVESTIGATIONS in fertility Management
Dr. Anupama Bahadur Additional Professor Dapartment of Obstatrick & Gymecology Rainikaan Ottatrakaana)-249 203 Distor: 03r4 Feb, 2021 Time: 3:00pm IST.	Dr. Sunita Chandra Chairperson and Director - Rajendra Nagar Hospital & IVF Hospital Chairperson and Director - Rajendra Nagar Hospital & IVF Hospital Chairperson and Director - Rajendra Vandra Berchary IIF SUP Chairper Berchary IIFS UP Chairper Berchary II	Dr Aswathy Kumaran Chart Mitter Reproductive Medicine - Aster MIRAKL Aster Mitter Rottakk, Keräf BBS, DGO M.S. DNB, FNB (Reproductive Medicine) Constructive of ESiME COP upideline 3000 1 years experience as Obstetrician ans Gynaecologist Lotter: 12 ^{re} Feb, 2021 3:00pm 15T.
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Troubleshooting in Ovarian Stimulation in IUI <mark>-</mark> Case Files	Endometrioma during IVF Cycle - Lecture	Hysteroscopy and Uterine Factor-Is Removal must for Septum, Polyp & Adhesions?
Dr. Neeru Thakral Director Takvil Hospital And Forlity Center & Director Takvil Hospital And Forlity Center & Director Takvil Hospital And Forlity Center & Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Call Generational WF Center (UBBAC)N. Secretary IFS Haryana chapter Secretary IFS Haryana chapter Secretary IFS Haryana chapter Secretary IFS Haryana chapter	Dr. Nymphea Walecha Bas, MS. Constant Incharge - Fortis Ridge IVF, Shalimar Bag, Delhi. Scusive Member - ISB and FPSI Constant Incharge - Fortis Ridge IVF, Shalimar Bag, Delhi. Scusive Member - ISB and FPSI Constant Incharge - Fortis Ridge IVF, Shalimar Bag, Delhi. Scusive Member - ISB and FPSI Constant Incharge - Fortis Ridge IVF, Shalimar Bag, Delhi. Scusive Member - Staff St Gintroment and Fertility? Vier Weadown - Staff St Gintroment and Fertility?	Dr. Shalini Chawla Khanna B.B.S., D.G.O., D.N.B., M.A.M.S Sciori VF & Laparoscopic Consultant - Max Hospitals, Delhi & NCR Sciori VF & Laparoscopic Consultant - Max Hospitals, Delhi & NCR Executive Wember - Indian Fertility Society 2020-2022. Difficient of the March, 2021
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IFS ACTIVITIES VIBRATE MEETINGS

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IFS ACTIVITIES VIBRATE MEETINGS



IFS ACTIVITIES CHAPTER ACTIVITIES

Western Maharashtra Chapter

Date: 4th October, 2020



Vidarbha Chapter

Date: 30th January, 2021



IFS ACTIVITIES CHAPTER ACTIVITIES

Odisha Chapter

Date: 24th February, 2021



Karnataka Chapter

Date: 21st March, 2021

Vidarbha Chapter

Date: 10th June, 2021

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U.K. Chapter Date: 12th June, 2021



Rajasthan Chapter

Date: 19th June, 2021



Dr. Narender Gupta Dr. Gunjan Jain Dr. Taru Chaya Bansal

IFS ACTIVITIES MISCELLANEOUS



6:05-6:26 PM 6:25-6:30 PM

Rita Singh

IFS ACTIVITIES e-CMES



When it's time, join your Webex meeting here. Meeting number (access code): 187 370 3094

Meeting password: 12345

Join by phone Tap to call in from a mobile device (attendees only) +91-40-6480-2006 India Global call-in numbers | Toll-free calling restrictions

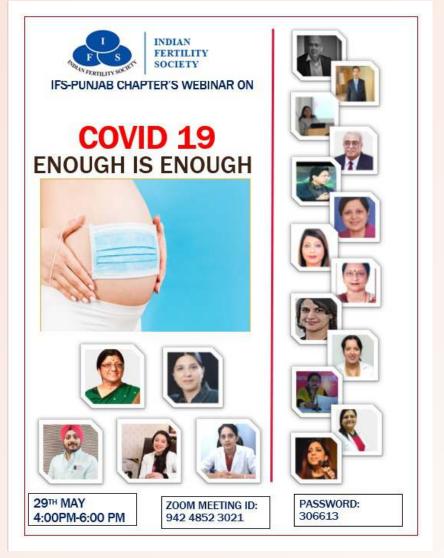
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IFS ACTIVITIES WEBINARS









IFS ACTIVITIES SIG ACTIVITIES

IFS SIG- Embryology Date: 1st October, 2020

Vitr	ification: Knowho the Latest from th Web Thursday, 1st	w, Tips and Tricks the Eminent Experts Dinar October, 2020	and
President, IFS	Secretary General, IFS	- 8:00 PM Chairperson - Convenor SIG	Moderator – Co Convenor SIG
2	2		5
Dr. Sudha Prasad	Dr. Neena Malhotra	Dr. Geeta Goswami Faculty	Dr. Parag Nandi
	Dr. Sujatha Ramakrishr	nan Dr. Vijay Mangoli	
	FIOSIG	annine	
		n to Webinar Dr. Geeta	
06:0	5 PM - 06:10 PMWelcor	ne Address Dr. Parag N n in reproductive "Dr. S	andi ujatha Ramakrishnan,
06:0 06:10 PM - 06:55 PM	5 PM - 06:10 PMWelcor "Vitrification, Application tissues and Current Pro	ne Address Dr. Parag N n in reproductive "Dr. S	andi ujatha Ramakrishnan, ELD
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IFS SIG- ENDOMETRIOSIS Date: 10th October, 2020



IFS SIG- Holistic Medicine Date: 5th October, 2020



https://tinyurl.com/IFS-HM

IFS SIG-POR Date: 17th October, 2020



IFS ACTIVITIES SIG ACTIVITIES IFS SIG-PCOS

Date: 18th October, 2020



IFS SIG- Fertility Preservation Date: 20 October, 2020



IFS SIG-APPLIED GENETICS Date: 31st October, 2020

31st October 2020 (Saturday), 5pm onwards.

Registrations Open



WEBINAR SERIES FU **GENETIC DEFECTS IN IVF BABIES** . SPECIAL INTEREST GROU BLOCK YOU DATE 30.01.2021 DR SA SESSION ECTURES GENETIC RISKS OF IVF/ICSI IN N APT. PREG NCY COMPLICATI BIRTH DEFECTS 5:00 PM TO 5:20 PM 5:25 PM TO 5:45 PM THEME: THE RISK OF BIRTH DEFECTS IN CONCEPTION BY ART MODERATORS SOTH OF JANUARY' 2021 inDN SATURDAY || 5:00 PM ONWARD REGISTRATION OPENS INDNA LIFE SCIENCES

IFS ACTIVITIES SIG ACTIVITIES

IFS SIG-APPLIED GENETICS

Date: 30th January, 2021



IFS SIG-PCOS Date: 20th February, 2021



IFS SIG-Search Methodology ► Date: 1st & 2nd April, 2021



IFS SIG-Applied Genetics Date: 16th April, 2021



IFS SIG-APPLIED GENETICS

Date: 18th June, 2021



INTERNATIONAL PRESENTATION FROM IFS MEMBERS- ASRM 2020





Milind RAMCHANDRA Ubale

Professor and Head Department of Microbiology Rajiv Gandhi Medical College & Chatrapati Shivaji Maharaj Hospital Thane, India

Reduction of Bacterial Colony Forming Units in an Obstetrics Operation Theater using Cold-Plasma based Dielectric Barrier Discharge Air ¹Dr. Milind Ubale M.D. ²Dr. Rajvi H. Mehta Ph.D ¹ RGM College & CSM Hospital, Kalwa, Thane ² Trivector Biomed LLP, Mumbai, INDIA





Satish Manohar Patki

Head of the Institute Patki Hospital, IVF Consultant Kolhapur, India





IMPROVEMENT IN THE BLASTOCYST FORMATION AND SUBSEQUENT CLINICAL PREGNANCY RATES FOLLOWING THE USE OF COLD-PLASMA BASED AIR PURIFICATION SYSTEM IN THE EMBRYO CULTURE LABORATORY Dr. Satish M Patki, MD, Dr. Rajvi Mehta Ph.D.,

3

Retrospective study to compare between hyaluronan enriched medium and blastocyst transfer medium for frozen embryo transfer and its impact on CPR in patients with 2 or more in vitro fertilization/intracytoplasmic sperm injection (IVF-ICSI) cycle failures. R.Sharma, A.Gupta, F. Rahman, N.Kaur, Rohan ORIGYN FERTILITY & IVF CENTER, NEW DELHI, INDIA



4



Role of blastocyst morphology in predicting clinical outcomes in single frozen blastocyst transfers

> Majumdar G, Sehgal S, Gupta S, Tiwari N, Satwik R and Majumdar A Center of IVF and Human Reproduction, Sir Ganga Ram Hospital, New Delhi, India



IMPACT OF ENDOMETRIOSIS ON ANXIETY, DEPRESSION AND QUALITY OF LIFE AND ITS ASSOCIATION WITH PREGNANCY OUTCOMES IN INFERTILE PATIENTS AT A TERTIARY LEVEL INFERTILITY CENTRE IN INDIA.



Kanad Dev Nayar, Poonam Nayar, Shweta Gupta, Minal Singh, Ratnaboli Bhattacharya,Gaurav Kant, Rahul Gahlot, Kapil Dev Nayar HA Akanksha IVF Centre, Delhi, India

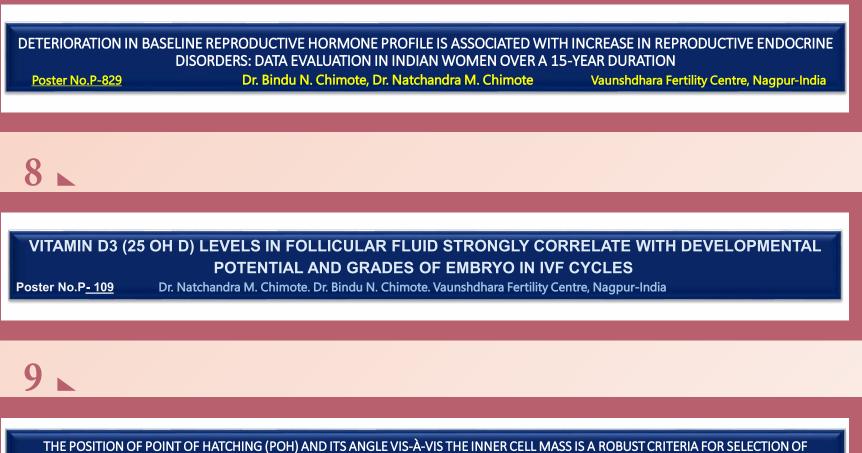


DOES CONTROLLED OVARIAN HYPERSTIMULATION RESPONSE VARIES WITH PCOS PHENOTYPES: A PROSPECTIVE COHORT STUDY AT A TERTIARY INFERTILITY CENTRE IN INDIA.



Kanad Dev Nayar, Shweta Gupta, Minal Singh, Ratnaboli Bhattacharya,Eshna Gupta, Rahul Gahlot, Kapil Dev Nayar Akanksha IVF Centre, Delhi, India 7

INTERNATIONAL PRESENTATION FROM IFS MEMBERS- ASRM 2020



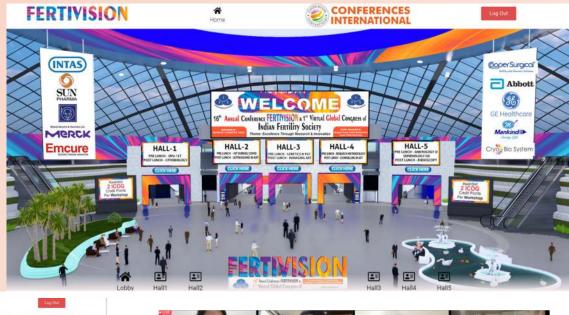
HATCHING BLASTOCYST FOR ENHANCED LIVE BIRTH RATES IN IVF CYCLES

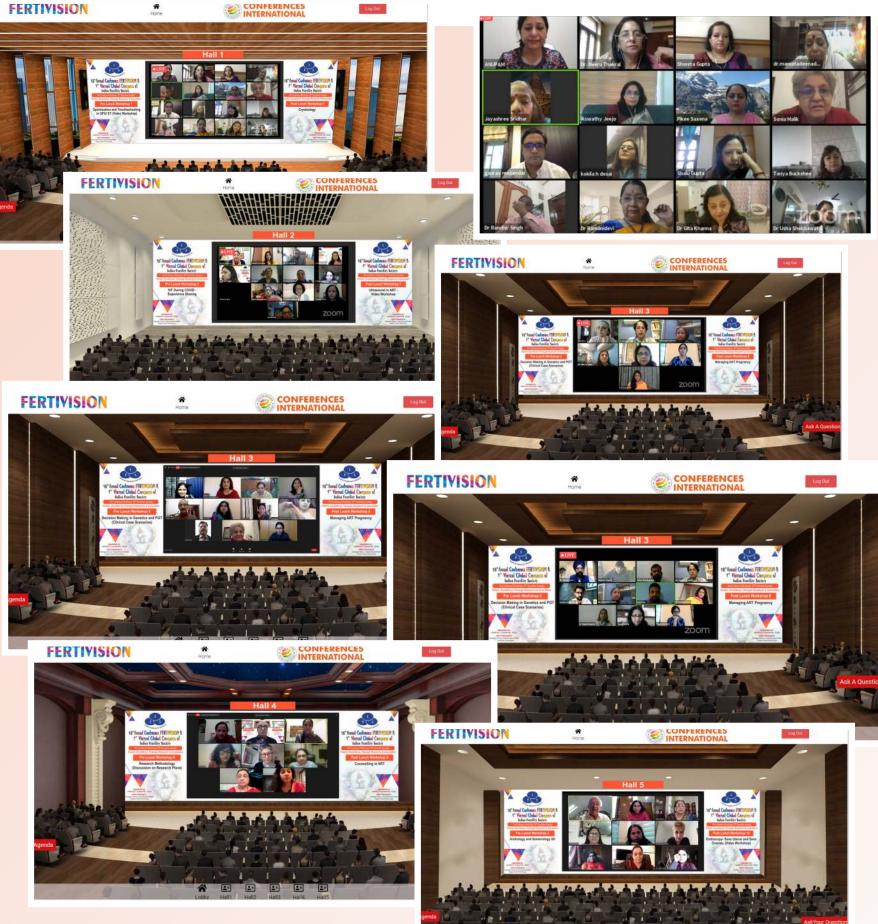
Poster No.P-942 Dr. Bindu N. Chimote, Nishad Chimote, Dr. Amogh Chimote, Dr. Riju Chimote, Dr. Natchandra M. Chimote Vaunshdhara Fertility Centre, Nagpur-India

Corresponding Author: Dr. Bindu N. Chimote email: <u>bindunm10@yahoo.com</u> Mobile No. +919890346199

Workshop Memories

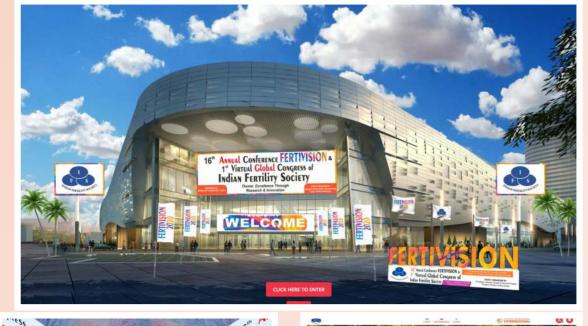
FERTIVISION 2020

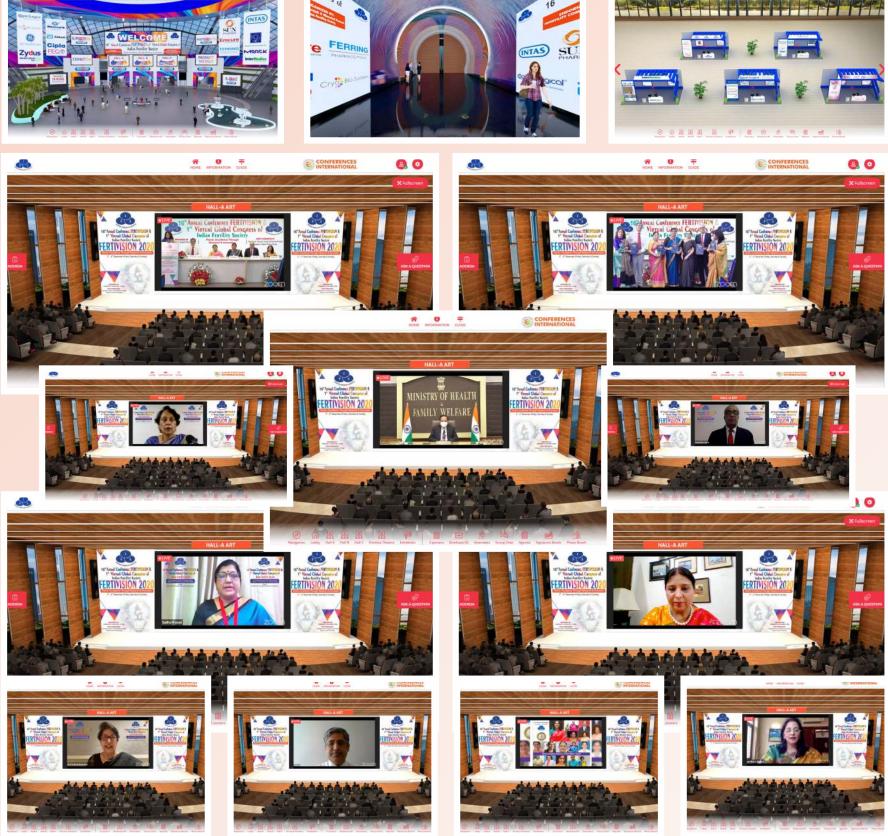


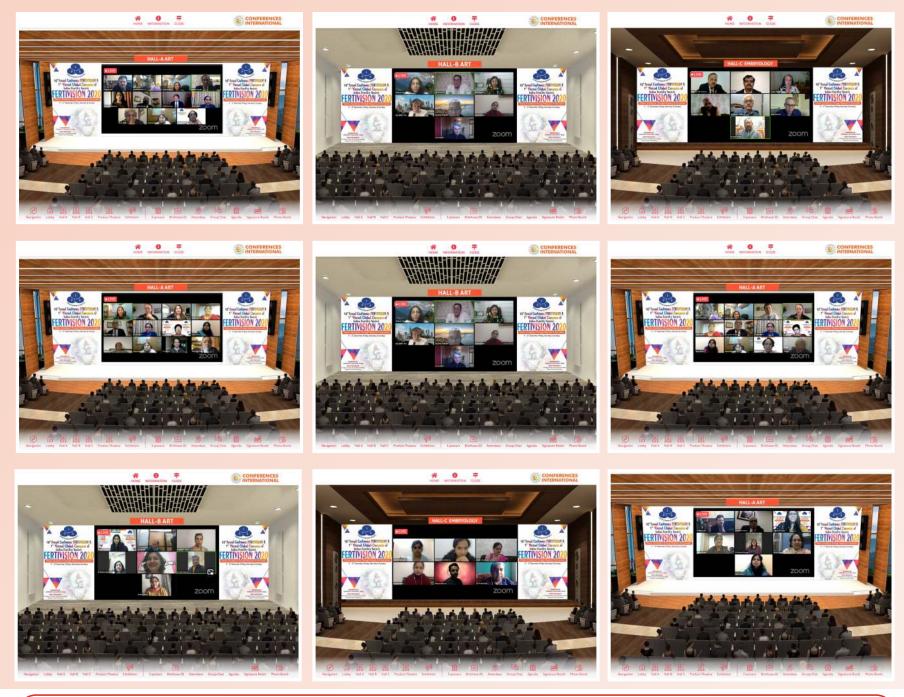




FERTIVISION 2020







16th Annual Conference of Indian Fertility Society which was also 1st Virtual and Global conference **FERTIVISION 2020** and was hosted jointly by the four northern chapters Chandigarh, Punjab, Haryana & Himachal Pradesh.

Participated by

- * 70+ International Faculties
- * 300 National Faculties
- Nearly 2000 Delegates participated in the 10 workshops organized
- More than 3000 logins for the main conference
- * 130 Original Research Papers were Presented

