

DIAGNOSIS AND SONOGRAPHIC APPEARANCE OF POLYCYSTIC OVARY SYNDROME (PCOS)

METHADODOLOGY:

Peer review articles published in last 5 years were searched to conduct this literature review.

Michener LRC online data base is used to accesses the journals and articles which focused on patient's demographics, epidemiology, pathophysiology, clinical manifestations, ultrasound methods and finding, medical management and prognosis of PCOD.

INTRODUCTION:

Polycystic ovary syndrome (PCOS) is a common endocrinopathy in women of reproductive age, with heterogeneous presentation of hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology. (1) Also associated is insulin resistance and obesity. (2)

Currently, three main diagnostic criteria system are accepted for PCOS including, The Rotterdam (ROT), Androgen excess society (AES) and National Institute of Health (NIH). (2)

They all agree for PCOS diagnosis include, polycystic ovarian morphology(PCOM) with > 12 antral follicles of 2-9mm or ovarian volume of 10 cc in at least one ovary, Oligo/amenorrhea and hyperandrogenism.(2)

Even after these criteria met one should exclude other causes of hyperandrogenism like congenital adrenal hyperplasia and adrenal secreting tumors by checking free testosterone and 17 alpha hydroxy-progesterone.(2)

Luteinizing Hormone (LH) levels are also high in PCOS but LH levels are not assessed routinely. (3)

DEMOGRAPHICS AND EPIDEMIOLOGY:

PCOS can be seen in women of any ethnic background. (3) In one study of 400 women, 4-4.7% of white women and 3-4% of black women had PCOS. (3)

Around 5-10% of women of reproductive age are affected.(3) Condition can be seen any time from menarche until menopause but generally seen around menarche and usually diagnosed in early adulthood.(4)

25 -50% PCOS tends to cluster in families seen in mother and sisters of PCO patients. (4)

Prevalence among first degree relatives, suggests genetic predisposition and strong inheritance. (4).

PATHOPHYSIOLOGY:

The basic pathophysiology is unknown. However various biochemical abnormalities have been described. (3) 50-70% of patients shows impaired action of insulin on glucose transport and anti-lipolysis on adipocytes in the presence of normal insulin binding. (3)

Increased insulin causes increased androgen production by theca cells of ovaries. (3,4) Insulin decreased production of sex hormone binding globulin (SHBG), and increased unbound testosterone which manifest clinically as acne and hirsutism.(3)

At the level of granulosa cells insulin increase response of granulosa cells to LH and this leads to premature arrest of follicular growth and anovulation.(3) Abnormal gonadotrophins dynamics are a key feature.(3,4) Increased Gonadotrophin Releasing Hormone(GNRH) secretion in pituitary result in increased secretion of LH and elevated LH/FSH ratio.(4)

A genetic component is likely, as PCOS tends to occur among family members. (4) A genetic defect in theca cells may explain the increased androgen production (4)

CLINICAL MANIFESTATIONS:

Clinical presentation varies widely. Women seek medical advice mainly due to menstrual irregularities, hirsutism, acne and infertility. (5)

Menstrual irregularities range from amenorrhea to oligo to normal menses. (5) 30% of women with PCOs has normal menses and 85 -90% of women with oligo has PCOs while 30 40 % have amenorrhea with PCOS. (5)

Signs of androgen excess like hirsutism and acne affects 80% of women. (5) Acne is less prevalent and 15-30 %of women present with acne. (5)

Infertility affects 50% of women and it's the most common cause of anovulatory infertility.(5) 80-90% of women presenting to infertility clinic has PCO due to arrest in follicular growth, when they reach a diameter of 4-8mm.(5) Therefore dominant follicle does not develop, ovulation does not ensue.(5)

Other associated metabolic disorders with PCOS obesity, include Type 2 Diabetes, endometrial hyperplasia and cancer, hypertension, non-alcoholic fatty liver disease, dyslipidemia and metabolic syndrome.(4) Metabolic features have long term impact on health.(4) It's unclear whether PCOS predisposes to obesity or obesity exacerbates PCOS but prevalence of obesity is 60%.(4)

ULTRASOUND METHODS:

Currently ultrasound (both transabdominal (TA) and transvaginal (TV) is the most widely used non-invasive means of evaluating ovarian morphology (6).

TV(after verbal or written consent), supersede TA because of greater resolution and patients preference since full bladder is avoided, it saves time and is more convenient for patients.(6) 2D u/s remain standard and current consensus from Rotterdam meeting rest on this technique.(6)

Ultrasound machine equipped with 6-7 MHz high frequency transvaginal transducer is recommended as it has better spatial resolution and examination depth and it allows more accurate assessment of internal and external morphological feature of ovaries.(6)

However, TA approach is used by adolescent girls and virginal women who declined TV. (6)

TA 3-5MHz probe offers whole view of the pelvic cavity and so may be used when TV fails to visualize displaced ovaries (6). Although full bladder is required for ovarian visualization but routinely full bladder can compress the ovaries yielding falsely increase length. Therefore area or volume of the ovary to be assessed for correct diagnosis. (6)

Timing of u/s: According to criteria, base line scan of pelvis to be done in early follicular phase (day 1-3) if the patient does not have regular period than u/s may be done at any time or after progestin withdrawal. (6) Uterine dimensions and endometrial thickness should be recorded during base line scans due to risk of endometrial cancer. (6)

Antral follicle count: The ovaries in PCO are enlarged symmetrically, and the shapes change from ovoid to spherical.(6)Ovarian volume can increase by as much as 6 mL; however, almost

30% of patients with a biochemical and pathologic diagnosis of polycystic ovaries have no increase in ovarian volume(7).

The typical polycystic ovary contains 12 or more follicles estimated in both sagittal and transverse plane at any given time. (6) The follicles are from 2-9 mm and no dominant follicle is present and volume increase to 10 ml, and formula to calculate volume is $L \times W \times H \times 0.52$. (6) Characteristically, the follicles are peripherally located in the cortex. (7) However, they can occur anywhere in the ovarian parenchyma (7). The diagnosis of polycystic ovaries should be reserved for patients with at least 5 of these follicles in each ovary. (7)

Typically, the ovaries are hypoechoic in relation to the surrounding pelvic fat and myometrium. (7) Polycystic ovaries often display increased echogenicity. (7) However, as many as one third may remain isoechoic or hypoechoic relative to the myometrium. (7)

The presence of single PCO is sufficient to meet ovarian morphology criteria for diagnosis of PCO. (6)

3D: Relatively new modality, expensive instrument and need more experience to use the technology limits its use. (6) However it facilitates quantitative assessment of follicle count, total ovarian volume and blood flow better than 2D. (6)

OTHER MODALITIES:

MRI: More sensitive than u/s to detect PCO but this modality is not specific enough to permit the diagnosis of polycystic ovarian disease without corroborating laboratory values and features from the patient's history. (7) On T1-weighted images, the ovaries have homogeneously low signal intensity. (7) T2-weighted images reveal high signal intensity within the fluid-filled follicles of the ovarian cortex. (7) The ovarian stroma remains dark on these images. (7)

More sensitive than u/s to detect PCO but this modality is not specific enough to permit the diagnosis of polycystic ovarian disease without corroborating laboratory values and features from the patient's history.(7) Greater cost and more experience is necessary before sufficient criteria can be determined for the diagnosis of polycystic ovarian disease.(7)

MEDICAL MANAGENT:

Treatment is symptom directed.(3) If the patient is overweight then life style modifications including diet and exercise works best.(3) Even small amount of weight loss improves cycle regularity, increased spontaneous ovulation and pregnancy rates.(3) Following a non-Diabetogenic diet, reduces the risk of type 2 Diabetes by increasing insulin sensitivity. (4)

Life style modifications have low compliance and a high dropout rate. (1, 4) To overcome this problem, a structured exercise training program was evaluated in obese anovulatory PCOS patients. (1)

Oral contraceptive pills: For women who need cycle regularity and not interested in conception. (4)

Antiandrogens: In case of hirsutism and acne antiandrogens are used. (4)

Ovulation induction drugs are first line of therapy in anovulatory patients who wish to conceive. (4)

Insulin sensitizing agents: Increase tissue sensitivity to insulin action, and reduce body mass index by 4% and androgen level by 20 %.(4)

Laparoscopic Ovarian Drilling: to reduce androgen production. (4)

PROGNOSIS:

40% of patients with PCOS have obesity which put them at increased risk of Diabetes mellitus and cardiovascular complications.(8)

The chronic anovulation in PCOS leads to endometrial hyperplasia with estrogen without progesterone, and this increases the risk of endometrial carcinoma. (8)

The Royal College of Obstetricians and Gynaecologists (RCOG) recommends induction of withdrawal bleeding with progestogens a minimum of every 3-4 months.(8)

Therefore it's a physician responsibility to educate the patient about the prevention of long term consequences of PCOS. (3)

CONCLUSION:

In my case study, I scanned 31 year old lady with complain of irregular periods and infertility.

Her TV ultrasound feature demonstrate PCOS with both ovaries showing >12 antral follicles of 2-9 mm in diameter arrange in necklace like pattern in periphery of ovaries.

In correlation with this literature review my patient's ovarian volume was normal and she has no history of hirsutism, acne, obesity or metabolic disturbances.

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