A REVIEW ON THE MORPHOLOGY OF UTERUS IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME

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ABSTRACT
Polycystic ovary syndrome (PCOS) is one of the most common endocrinological pathologies in women during their reproductive years exhibiting a wide spectrum of clinical manifestations. PCOS is associated with chronic oligoanovulation and high circulating sex hormone levels. It is a heterogeneous disorder, with multiple reproductive, cosmetic and metabolic complexities which is characterised by dysfunction in ovulation and clinical or biochemical hyperandrogenism and the presence of polycystic ovarian morphology. It is the most common endocrine cause of infertility and increased the risk of adverse pregnancy outcome, metabolic syndrome, type 2 diabetes mellitus, and some carcinoma. There is a strong link of PCOS with endometrial thickness in the younger age group. The incidence and danger of endometrial cancer development is more in such individuals. Physiological changes that occur in such cases of PCOS are because when this condition occurs, the process of ovulation is affected and stopped, which in turn leads to the endometrial lining not being shed, resulting in the thickening of the endometrium and, hence, bigger threat of endometrial cancer. During ultra-sonography, the image of the endometrium showing thickened and heterogeneous areas was a predictive factor for endometrial malignancy.

Key words: Polycystic Ovaries, Infertile, Uterine Area, Endometrial Thickness, Anovulation, Menstrual cycle, Uterus.

INTRODUCTION
PCOS is a problem with hormones that affects women during their childbearing years (ages 15 to 44). Between 2.2 and 26.7 percent of women in this age group have PCOS. [Fahimeh R.T and Samira B.G]. At the first time, PCOS was described by Stein and Leventhal in 1935 [Balen A, Michelmore K, 2002] as the presence of bilaterally enlarged ovaries with multiple cysts in seven women with infertility, menstrual irregularity and hyperandrogenism [Atiomo WU et al., 2000]. Up to 70 percent of women with PCOS have insulin resistance, meaning that their cells cannot use insulin properly [John C. Mand Andrea D, 2012].

The common age of onset for PCOS is adolescence while the common time of diagnosis is during a woman’s third or fourth decade of life because the majority of symptoms do not become evident until a woman reaches her twenties or thirties, even though some symptoms may appear starting at menarch [Dunaif, 1997]. The exact cause of PCOS is not known. Most experts think that several factors, including genetics, play a role. There are many theories with much evidence pointing towards a genetic link because of the appearance of PCOS in clusters of female relatives and the presence of PCOS in certain individuals before their first menstruation [Franks et al., 2006].

PCOS is caused by an imbalance in the hormones (chemical messengers) in the brain and ovaries. PCOS usually happens when a hormone called luteinizing hormone (LH) (from the pituitary gland) or levels of insulin (from the pancreas) are too high, which then causes the ovaries to make extra amounts of testosterone. The menstrual cycle starts when the brain sends LH and FSH (Follicle stimulating Hormone) to the ovaries. A big surge of LH is the signal that causes the ovaries to ovulate, or release an egg. The egg travels down the fallopian tube and into the uterus. Progesterone from the ovary causes the lining of the uterus to thicken. If the egg is not fertilised, the lining of the uterus is shed. This is a menstrual period. After the menstrual period, the cycle begins all over again. With PCOS, LH levels are often high when the menstrual cycle starts. The levels of LH are also higher than FSH levels. Because the LH levels are already quite high, there is no LH surge. Without this LH surge, ovulation does not occur, and periods are irregular. Girls with PCOS may ovulate occasionally or not at all, so periods may be too close together, or more commonly too far apart. Some girls may not get a period at all [Polycystic Ovary Syndrome, 2014].
Some of the symptoms of PCOS include: (1) Irregular menstrual cycle, (2) too much hair on the face, chin, or parts of the body where men usually have hair. This is called “hirsutism.” Hirsutism affects up to 70 percent of women with PCOS, (3) acne on the face, chest, and upper back, (4) thinning hair or hair loss on the scalp; male pattern baldness, (5) weight gain or difficulty losing weight because of the metabolic abnormalities seen with the disease, (6) darkening of skin, particularly along neck creases, in the groin, and underneath breasts, (7) skin tags, which are small flaps of excess skin in the armpits or neck area [Polycystic ovary syndrome, 2016].

The uterus is a hollow, pear-shaped organ with thick muscular walls. In the young nulliparous adult, it measures 3 in. (8 cm) long, 2 in. (5 cm) wide, and 1 in. (2.5 cm) thick. It is divided into the fundus, body, and cervix. The fundus is the part of the uterus that lies above the entrance of the uterine tubes. The body is the part of the uterus that lies below the entrance of the uterine tubes. The cervix is the narrow part of the uterus. It pierces the anterior wall of the vagina and is divided into the supravaginal and vaginal parts of the cervix. The cavity of the uterine body is triangular in coronal section, but it is merely a cleft in the sagittal plane. The cavity of the cervix, the cervical canal, communicates with the cavity of the body through the internal os and with that of the vagina through the external os [Richard S. Snell, 2012].

The uterus has a very thick wall made up mainly of muscles. The lumen is lined by mucous membrane. The part of the uterus is covered on the outside by peritoneum. The endometrium has a lining of columnar epithelium that rests on a stroma of connective tissue. Numerous tubular uterine glands dip into the stroma. The appearance of endometrium varies considerably depending upon the phase of the menstrual cycle [Inderbir Singh, 2004]. The menstrual cycle is divided into the menstrual, proliferative (follicular) and secretory (luteal) phases. Normally, the average menstrual cycle is a 28-day cycle. Although the successive events constituting the cycle occur continuously, they can be described in three phases: menstrual phase, proliferative (follicular) phase, and secretory (luteal) phase. Menstruation, which begins on the day that bleeding from the uterus starts, occurs when fertilisation does not take place. In this case, the corpus luteum becomes non-functional about 14 days after ovulation, thus reducing the levels of progesterone and oestrogen.

The majority of PCOS women have irregular periods with menstruation being very erratic, infrequent, or painful, with an increase in flow and cramping. Chronic anovulation may present as oligomenorrhea, amenorrhea, dysfunctional uterine bleeding, and/ or infertility [Sheehan, 2004]. Oligomenorrhea refers to infrequent or very light menstruation while amenorrhea refers to the absence of menstrual periods for three or more months in a row [“Medical Dictionary,” 2013].
DISCUSSION

The normal appearance of the endometrium is smooth and regular versus a disrupted appearance. The appearance and thickness of the endometrium vary with different stages of the menstrual cycle. Endometrial thickness (ET) is 1-4mm during menstruation, 5-7mm in the proliferative phase, up to 11mm in the periovulatory phase and 7-14mm in the secretory phase [Goldberg BB and McGahan JP, 2006]. The uterine area and endometrial thickness were considerably increased in the older age group [Usmani, A, Rehman, R and Qamar, A, 2014]. The studies of AmbreenUsmani et al, showed that the endometrial thickness is more in the older group of infertile women which is in contradiction with some studies showing that in normal women with advancing age the endometrium becomes thin. However other studies show no such correlations. Due to the different phases of the menstrual cycle the endometrium of the uterus shows variations in thickness. These variations in thickness range from 3mm, which is usually seen after menses, to 15mm during the luteal phase. However, this thickness normally reduces after menopause [Park, JC et al., 2011]. Abnormal endometrial thickness has been associated with obesity, PCOS and diabetes mellitus [Goldstein, SR, 2010] [Bu, Z, 2012].

Shah, B et al, (2010) reported that 31.4% of adolescent girls suffering from PCOS had endometrial thickness of >7mm. Therefore, there is a strong link of PCO with ET in the younger age group. The incidence and danger of endometrial cancer development is more in such individuals. Thus, this connection must be acknowledged as early as possible to avoid cancerous changes, for these women have a greater chance of developing endometrial cancer, particularly if accompanied with irregular menstrual cycles. Physiological changes that occur in such cases of PCOS are because when this condition occurs, the process of ovulation is affected and stopped, [Nazir, F et al., 2011] which in turn leads to the endometrial lining not being shed, since it is exposed greatly to oestrogen, resulting in the thickening of the endometrium and, hence, bigger threat of endometrial cancer [Kenneth, MN, John, SP and Eran, BL, 2001].

Abnormal thickness can be caused by polyps, fibroids, hyperplasia and cancer [Goldberg BB and McGahan JP, 2006]. Since 1940s, there is emerging evidence of increased risk of gynaecological cancer including endometrial, breast and ovary cancer among women with PCOS [Legro RS, 2007]. Endometrial carcinoma (EC) is the second most frequent gynaecological malignancy among women [Hardiman P et al., 2003]. Major EC-related symptoms include dysfunctional uterine bleeding, hyper-menorrhrea, irregular menstruation, and sterility. The two main types of ECare are oestrogen-dependent type-I (the most prevalent type) and oestrogen-independent type-II carcinomas. Among numerous risk factors, PCOS is commonly considered to be a significant and causative risk factor for the development and progression of type I EC [Chittenden BG, 2009].

The mechanisms underlying EC and PCOS are also unclear, but it is widely assumed that chronic anovulation, which results in continuous oestrogen stimulation of the endometrium unopposed by progesterone, is a major factor [Fahimeh R T and Samira B G]. Obesity, hyperinsulinemia,and hyperandrogenism state in PCOS, result in increased bioavailability of unopposed oestrogens by progesterone due to the increased peripheral conversion of endogenous androgens such as testosterone and androstenedione into oestrogen. Also, Insulin up-regulates aromatase activity in endometrial glands and stroma; endogenous oestrogen production is enhanced in women with high circulating insulin. Oestrogens act as proliferative factors in the endometrial tissue. Continuous exposure of the endometrium to oestrogens with persistent progesterone deficiency, lead to endometrial overgrowth and hyperplasia or cancer [Horn L-C,2007]. The underlying physiological changes in case of PCOs is that due to this condition when ovulation does not take place the endometrial lining is not shed which in turn is exposed excessively to oestrogen resulting in endometrial thickening and hence more risk of developing endometrial cancer Gao JS et al., 2004].

Known factors that affected the endometrium were obesity, reproductive characteristics, certain medical conditions and cigarette smoking which may be explained partly by their effect on oestrogen and progesterone [Hale et al 2002]. Prior to 1982, diagnostic evaluation was done routinely by diagnostic dilatation and curettage. Nowadays, transvaginal sonography is becoming an increasingly popular tool for endometrial assessment with similar false positive rates as endometrial sampling in cancer detection. During ultra-sonography, the image of the endometrium
showing thickened and heterogeneous areas was a predictive factor for endometrial malignancy [Gull et al., 2003]. Tamura et al., (2006) studied the pathophysiology of unexpectedly thin endometrium in women with infertility focussing on uterine blood flow, growth of glandular epithelium, vascular development and angiogenic factors. The density of small blood vessels, the total area of glandular epithelium and the level of vascular endothelial growth factor (VEGF) expression were significantly lower in the thin endometrium compared with the group with normal endometrium. This could suggest that low blood flow is involved in the impairment of the growth of the glandular epithelium and stroma and in VEGF production leading to further poor angiogenesis and further reduction in uterine blood flow.

Nagamani, P and Levine, D (2007) conveyed that endometrial thickness of <6mm will rarely be able to conceive naturally and it is important to note that hormonal replacement therapy (HRT) is one of the most common factors in infertile women which is known to be connected with increased UA and ET.

CONCLUSION

Polycystic ovary syndrome (PCOS) is a health problem that affects one in 10 women of childbearing age. Women with PCOS have a hormonal imbalance and metabolism problems that may affect their overall health and appearance. PCOS is also a common and treatable cause of infertility. Researchers continue to search for new ways to treat PCOS. There are many different imaging appearances of the normal and abnormal endometrium. The ultrasound scan serves as an important tool in preventing primary infertility. Whether using US, MR imaging, sonohysterography or hysterosalpingography, radiologists take into account the patient’s age, stage in the menstrual cycle, and pregnancy status and whether she has undergone hormonal replacement therapy or tamoxifen therapy to make an accurate diagnosis.

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