Review Article

Role of Ayurveda on PCOS (Polycystic Ovary Syndrome): A Critical Review.

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ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a common chronic anovulatory disorder with androgen excess in women of reproductive age. It is a multifactorial, multisystem disorder but, obesity & insulin resistance are often associated. The patient with symptoms of menstrual irregularities, excess androgen (hirsutism, acne alopecia), and USG shreds of evidence of >2-9 mm size arranged in the cortex of the ovary. **Aims:** To explore and understand ayurvedic theories and practices on PCOS. **Material and methods:** The *Charaka Samhita*, a classical Ayurvedic book, was thoroughly examined to compile a list of relevant references together with Sanskrit commentary. Internet searched to locate study papers and related material. **Conclusion:** According to the Ayurveda PCOS is studied, and its pathogenesis tried to be established. *Kapha Vata* predominant *tridoshaja vikara* & *agnimandhya* is the main reason for pathogenesis. So, it can be effectively treated by *tridosha* pacifying, *agnivardhak chikitsa* with *uttarvasti.*

Keywords: PCOS, Ayurveda, Doshas, Agnimandhya

Polycystic ovary syndrome (PCOS) is the most common cause of infertility in women of reproductive age. It is a multifactorial disorder that shows features as hyperandrogenism, hyperinsulinemia, obesity, insulin resistance, anovulation, and cystic follicles in the ovary [1]. PCOS not corrected may lead to other serious consequences such as type II diabetes mellitus (DM), ovarian cancer, or cardiovascular disorders [2]. Therefore, the exact etiology behind this disorder needs to understand. The mechanism by which obesity causes PCOS-like changes in the ovary has not yet been fully understood. Obesity in PCOS is associated with hyperinsulinemia and insulin resistance [3]. Recent studies also showed that obesity is associated with low-grade inflammation [4]. Compensatory hyperinsulinemia and insulin resistance (IR) exert stimulatory effects on low-grade inflammation in PCOS.

Low-grade inflammation is associated with increased plasma levels of high sensitive C-reactive protein (hsCRP) [4–6]. Earlier it has been shown that obese persons with a high level of hsCRP showed a greater risk of developing type II DM diabetes mellitus [7]. These findings suggest that markers of inflammation may be responsible for the long-term consequences of PCOS. However, the mechanism by which low-grade inflammation induces the pathogenesis of PCOS needs further investigation. Oxidative stress (OS), which increases during inflammation, has also been reported as a potential cause of PCOS [8]. An increase in OS has been shown to correlate with IR. In earlier studies, IR and hyperglycemia have been shown to increase OS, although in one study increased OS have also been demonstrated in non-obese PCOS without IR [9].

Some earlier studies have shown increased lipid peroxidation (LPO) in hyperglycemia [10]. Obesity elevates OS, which in turn contributes to IR. An increase in reactive oxygen species (ROS) generation resulting from hyperglycemia in women with PCOS. [11]. ROS and antioxidants as biomarkers for PCOS patients, demonstrated studies done so far [12]. However, the pathogenesis of PCOS is not yet fully substantiated in the involvement of OS. Various laboratory and domestic animal models were developed to induce PCOS like features. Cystic follicles, hyperandrogenism, and anovulation were mainly observed after treating adult females with various hormones such as dehydroepiandrosterone, estradiol valerate, insulin, anti-progesterone, human chorionic gonadotropin (hCG), and the neonates with testosterone [13]. However, the precise mechanism of insulin resistance, oxidative stress, and low-grade inflammation developed in PCOS has not yet been

investigated. Recently, a rat model for PCOS was developed using a non-steroidal aromatase inhibitor, letrozole [14].

In the present study, we examined temporal changes in morphology, oxidant-antioxidant status, serum hormone levels, and serum levels of low-grade inflammatory markers in letrozole-treated rats to explore the mechanism of PCOS pathogenesis. PCOS is a lifestyle disorder with very complex pathogenesis it affects 2 –26% of females of the reproductive age group & the most common form of chronic anovulation with androgen excess, excluding other causes of hyperandrogenism like non-classical congenital adrenal hyperplasia, androgen-secreting adrenal tumors, hyperprolactinemia, etc. Previously, it is known as Stein Leventhal Syndrome as described by Stein and Leventhal in 1935 which manifests as bilateral polycystic ovaries, symptoms of menstrual abnormalities like amenorrhea, oligomenorrhoea, signs of androgen excess like- hirsutism and obesity [11]. The primary ovarian defect was inferred as the cause but subsequent clinical, morphologic, hormonal & metabolic studies uncovered multiple underlying factors & the term polycystic ovarian syndrome was introduced to reflect the heterogenicity of this disorder. The development of PCOS is attributed to the combination of *environmental factors*- lifestyle modifications from sedentary life, overeating without the proper gap between meals, workload, mental stress, etc. *Genetical factors* – sometimes a family history. Obesity & IR are recognized as one of the major root causes of PCOS leading to infertility. *Insulin resistance* is present in around 65-80% of women with PCOS, independent of obesity & is further exacerbated by excess weight.

Clinical Features: This condition manifests itself from modest monthly abnormalities to severe reproductive and metabolic failure. Oligomenorrhoea (85%-90%) and amenorrhea (85%-90%) are two typical menstrual abnormalities associated with PCOS (30-40 percent). Hirsutism is a prevalent clinical manifestation of hyperandrogenism that affects up to 70% of PCOS patients. Acne can be a sign of hyperandrogenism, with 15-30% of women having this symptom [15].

Diagnosis of PCOS based upon the presence of 2 of the following 3 criteria (*Asrm/Eshre*) –

- Oligo ovulation or Anovulation
- Androgen excess
- USG shreds of evidence of PCOS

By symptoms and signs of PCOS, we can consider it as *Kapha Vata* Predominant *Tridhoshaj Artava Dushti*. As Vata is responsible for the division of cells (granulosa, theca cells), rupture of the follicle, etc. *Pitta* has *paaka karma* associated with the conversion of androgen to estrogen & maturity of follicles. Subsequently, due to its *Shrava Karma*, it results in ovulation with the help of Vata. *Kapha* has a nutritive function along with *Vata* helps in the proliferation of ovarian follicles [16]. Although in Ayurveda description of PCOS is as such not present but to some extent few diseases simulated as described by some Scholars-

1. *Pushpaghani jataharni-* "*vrutha pushpam*" & "*sthula Loma gandasha*": Menses occurs regularly at the usual time. And the women suffering from this type of infertility have obese cheeks covered with hairs. Description of obese cheeks may indicate central obesity which is found in PCOS as steroid hormones, mainly androgen have increased. Also, there is an indication of hirsutism, which is again due to androgen excess [17]. The name *Phuspaghani* here indicates, infertility due to anovulation or due to the formation of bad quality of ovum or defective endometrium.

2. *Vikuta jataharni*: Menses are irregular in terms of duration, color, and amount. In the anovulatory and oligo-ovulatory cycle menses are either scanty or excessive in terms of amount either there is oligomenorrhoea or amenorrhea. Menses may ~~vary~~ in duration of bleeding and color, according to the interval at which menstrual bleeding starts. It seems to be due to oligo ovulation or anovulation found in PCOS [18].

3. *Artavakshaya-* "*yatho uchita kalam aadarshanam alptaa va yoni vedana cha*": In *artava kshya* there are features of oligomenorrhea, scanty menses, and painful menses explains PCOS only partially. Since acharyas mainly focused on the description of *bahaya atarva Lakshana* (menstrual blood) not the other, signs of androgen excess and the presence of cystic ovaries have not been explained [15].

4. Granthibhut artava dushti: As *artava* word used for an ovum, hormone, and menstrual blood. We see in *beeja roopi artava*/ovum is anovulation cystic ovaries if we consider that the *antaha artava*/hormones get vitiated by excess *Kapha* and *Vata.* And the *bahaya artava*/ menstrual blood will also form clots, either with the scanty or heavy flow depending on dosha predominance. Or there may be hyperplasia of the endometrium from polyps. In the *nidana* of *Nasta artava*, Acharya Vagbhata has explained that obstruction of channels *artava* is not evident, the patient remains amenorrheic [18]. All these facts indicate that *Vata Kapha Dosha* predominance in this disorder and symptoms appear according to predominance between two

doshas. But if we closely look at symptoms & signs of PCOS. *Pushpaghani jataharini* described by Acharya Kashyapa seems to be closer to symptoms & signs of PCOS.

✓ *Vratha pushpam*- The ovum or prepared endometrium is not useful for conception.

✓ *Stulalomagandasha*- The patient having chubby or fatty cheeks with excessive hair growth.

AIMS AND OBJECTIVE

- To understand the concept of the menstrual cycle according to the Ayurved.
- To understand the role of tridosha in the development of follicles in the ovaries.
- To understand the etiology & pathogenesis of PCOS, as per Ayurveda theories.

MATERIAL AND METHODS

The data are collected from the *Ayurvedic Samhitas*, journals & articles on google & from previous studies.

OBSERVATION AND RESULTS

In ayurvedic literature, mainly 4 factors are stated essentially for the conception & maintenance of their quality is emphasized for good pregnancy outcomes.

- *Ritu*- appropriate time (ovulating time)
- *Kstera* - field (whole reproductive tract)
- *Ambu* -proper nourishment
- *Beeja* – ovum, menstrual blood, hormones, Artav

Out of all the four factors, *artava* has great importance, as normal physiology of the other three factors depends on the normal functioning of *artava*. "*streenam garbhoupyogi shyaat artavam Sarva sammtam*". In ayurvedic literature, *the artava* word is used for *Antaha Pushpa* – hormones &ovum and *Bahaya Pushpa* – menstrual blood. Features of *bahya Pushpa* and *rituchakra* can help to predict the physiology of *antaha Pushpa*. When the *antaha Pushpa* (hormones) is functional physiologically than the *bahya Pushpa* (menstruation) shows features of *shudha artava* if there is no anatomical deformity in the genital tract.

Features of Normal Menstrual Bleeding: Regular and cyclical menses of one-month cyclic length. As in the ayurvedic or Hindu calendar, two pakshas of 15-15 days equal to one month. Bleeding is considered normal if it occurs at 30 days interval. It should not be greasy or soapy, not associated with burning, and should without any pain. The duration of flow should be for five nights. Flow should not be heavy or scanty.

Rituchakra- Cyclical physiological changes in reproductive organs, in the uterus and ovary due to cyclical changes in hormonal status. As stated in Ayurvedic works of literature that the whole month *beeja rakta* (hormones) brings the following change *Garbhashaye tarpyati pooryati*- forms the endometrial bed and *Manshaad beejaye kalpte*- folliculogenesis

Rajaswala Paricharya- Bramcharyani – During menstruation exertions of all kinds should avoid, whether related to celibacy or another physical as well as mental stress. According to Ayurveda, these things will lead to *vata vrudhi* because due to menstrual losses, the body is emaciating [11]. If we try to understand we found that intercourse should avoid during menses because the genital defense mechanism is hampered due to which female is more prone to infections. And physical stress may lead to the release of more CRH turn more ACTH and cortisol. CRH via direct impact on the hypothalamus inhibit GnRH secretion.

Adha Shayanni/ Darbha Sanstar Shayanni- She should sleep overground/floor has many advantages. The spine is more prone to curving on a soft surface, so sleeping on a firmer may help to align & straighten the neck, and the spine may relieve back or pelvic discomforts during menses.

Yavakam paysha shidham alpam karshanardham ashmniyat- A woman is advised to take light food which gets easily digested to protect her digestive fire [16].

Teekshana ushna amla lavanani varjayet- Foods containing excessive hot, sour, salty properties can cause excessive blood loss by vitiating pitta dosha. So should better be avoided [13]. After studying *rajaswala paricharya*, it is observed that rajah Kala can be considered natural *shodhana Kala* leading to *agnimandhya,* it is necessary to follow *pathya & apathya* prescribed during *shodhana* therapy which is the same as *rajaswala paricharya*. The aim of this is to avoid dosha vitiation & prevent *aama* formation & restore energy prevent menstrual disturbances due to lifestyle changes.

DISCUSSION

In Ayurveda, *the tridosha* is considered a physiological unit of the body. Whereas *dhatu & malas* are considered structural units. So, ovarian follicular recruitment, growth, maturation, ovulation, and ovarian steroidogenesis controlled *tridosha* [18]. We know below umbilicus *vata*

dosha is predominant mainly *vata* is responsible for proper reproductive function. Sun, Moon & air in the outer world, *pitta*, *Kapha & vata* in the inner world or body are responsible for the sustenance of proper functioning of the body. *Kapha* has visarga karma – it is nourishing in function and provides strength**.** Pitta has *aadan karma* has a metabolic function. *Vata* has *vikshepa* karma – it has to disperse function [19,26]**.** If doshas are present in the body within their normal physiological limits & show normal properties then growth, development, maturation, and ovulation of follicles occur timely whereas, imbalanced results in disturbances of this process. So there is a disturbance of the balance of *tridosha* resulting in symptoms and signs of PCOS [24].

Study of etiology, pathophysiology, observing the sign, symptoms of PCOS, and correlating these with the features of *vata, pitta, and Kapha* in their nature as well as in vitiated states, the possible *doshaja* vitiation seems to be *Kapha vata* predominant *tridoshaja vikara* [20, 25]. PCOS is a complicated endocrine illness that is linked to obesity, hirsutism, and chronic anovulation. It is a primary cause of infertility and menstrual irregularities. In Ayurveda, PCOS cannot be linked to a single entity however, it does *resemble pushpaghni jatiharini* [21]. Other names are *shandi yoni vyapad*, *Charak's bandhya, Sushruta's bandhya yoni vyapad*, and Kashyap's *vikuta jatiharini*.[22,23]**.** Obesity is the primary cause and symptom, which can be avoided by practicing *dincharya* and *ritucharya*, and reduced by practicing *pathya aahar*, *vihar*, *aushadh*, and *apathya aahar vihar* limitation.

CONCLUSION

This article gives an understanding of PCOS in the context of Ayurveda. Diseases are treated more effectively if doctors have a better understanding of them. Although PCOS is not mentioned in the Samhita, Acharya makes a point about therapy. Unnamed diseases should get classified according to their Dosha and Dushya, and therapies should get arranged accordingly. *Bandhyayonivyapada* has the most concordance with PCOS of all *Yonivyapada*. Expanded definitions of Aartava, such as menstrual blood, ovum, and hormones, aid in elaborating PCOS symptomatology in an Ayurvedic context and planning treatment options.

REFERENCES

1. Azziz R, Carmina E, Dewailly D, *et al.* Position statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an androgen excess society guideline. J Clin Endocrinol Metab. 2006; 91:4237–45.
2. Norman RJ, Dewailly D, Legro RS, *et al*. Polycystic ovary syndrome. Lancet (London England) 2007; 370: 685–97.
3. Puder JJ, Varga S, Kraenzlin M, *et al.* Central fat excess in polycystic ovary syndrome: relation to low-grade inflammation and insulin resistance. J Clin Endocrinol Metab. 2005; 90:6014–21.
4. Repaci A, Gambineri A, Pasquali R. The role of low-grade inflammation in the polycystic ovary syndrome. Mol Cell Endocrinol. 2011; 335:30–41.
5. Ford ES. The metabolic syndrome and C-reactive protein, fibrinogen, and leukocyte count: findings from the Third National Health and Nutrition Examination Survey. Atherosclerosis. 2003; 168:351–8.
6. Das UN. Obesity, metabolic syndrome X, and inflammation. Nutrition 2002; 18:430–2.
7. Barzilay JI, Abraham L, Heckbert SR, *et al.* The relation of markers of inflammation to the development of glucose disorders in the elderly: the Cardiovascular Health Study. Diabetes. 2001; 50:2384–9.
8. Rezvanfar M, Rezvanfar M, Ahmadi A, *et al.* Mechanistic links between oxidative/nitrosative stress and tumor necrosis factor alpha in letrozole-induced murine polycystic ovary: biochemical and pathological evidences for beneficial effect of pioglitazone. Hum Exp Toxicol. 2012; 31:887–97.
9. Verit FF, Erel O. Oxidative stress in nonobese women with polycystic ovary syndrome: correlations with endocrine and screening parameters. Gynecol Obstet Investi. 2008; 65:233–9.
10. Uzel N, Sivas A, Uysal M, *et al.* Erythrocyte lipid peroxidation and glutathione peroxidase activities in patients with diabetes mellitus. Horm Metab Res. 1987; 19:89–90.
11. Qatanani M, Lazar MA. Mechanisms of obesity-associated insulin resistance: many choices on the menu. Genes Dev. 2007; 21:1443–55.
12. Yeon Lee J, Baw C-K, Gupta S, *et al.* Role of oxidative stress in polycystic ovary syndrome. Curr Womens Health Rep. 2010; 6:96–107.
13. Srivastava RK, Krishna A. Pathophysiology of polycystic ovary syndrome: lesson from animal studies. Proc Ind Nat Sci Acad U S A. 2006; 71(B):189–97.
14. Kafali H, Iriadam M, Ozardali I, *et al.* Letrozole-induced polycystic ovaries in the rat: a new model for cystic ovarian disease. Arch Med Res. 2004; 35:103–8
15. Sharma H Kashayp. Samhita vidhyottini teeka, kalpa sthan. Chaumkhambha Orientalis Varanasi. 2006; 6(33):52-55.
16. Pandey N, Tripathi YB. Antioxidant activity of tuberosin isolated from Pueraria tuberose Linn. J Inflamm (Lond). 2010; 7:47.
17. Sharma H Kashayp. Samhita vidhyottini teeka, kalpa sthan. Chaumkhambha Orientalis Varanasi. 2006; 6(34):113-119.

18. Shastri A.D. Sushrut Samhita Ayurveda tatvasandipika teeka sutra sthana. Chaumkhambha Orientalis Varanasi. 2009; 15(12):89
19. Gupta A, Astang hiradya, Vidhyottini teeka. Chaukambha Prakashan Bhandar. 2003; 3(10):88-95.
20. Veshya S, Bhavprakash, Vedyasanjeevini teeka purva khand. Chaukambha Prakashan Bhandar. 2006; 6:118.
21. Shastri K, Charak Samhita. Vidhyottini teeka chikitsha sthana. Chaukambha Prakashan Bhandar. 2003; 30:223.
22. Gupta A, Astang hiradya. Vidhyottini teeka delhi. Chaukambha Prakashan Bhandar. 2003; 16:118.
23. Tiwari P.V. Ayurvediye Prasuti Tantra evam stri roga part 1 delhi. Chaukambha Prakashan Bhandar. 2010; 1(3):80-88.
24. Shastri K, Charak Samhita. Vidhyottini teeka, chikitsha sthana delhi. Chaukambha Prakashan Bhandar. 2003; 30:223.
25. Shastri K. Charak Samhita, vidhyottini teeka, chikitsha sthana delhi. Chaukambha Prakashan Bhandar. 2003; 30:224-28.
26. Shastri A.D. Sushrut Samhita Ayurveda tatvasandipika teeka sutra sthana. Chaumkhambha Orientalis Varanasi. 2009; 15(12):89.

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