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CONCEPTUAL STUDY ON POLY CYSTIC OVARIAN SYNDROME WITH AYURVEDA AND MODERN PERSPECTIVE:

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ABSTRACT

Poly Cystic Ovarian Syndrome (PCOS) is one of the most common metabolic disorders among the women of reproductive age. Polycystic ovarian syndrome (PCOS) affects 4% to 12% of women of reproductive age. The most challenging and distressing aspect of PCOS for any patient, varies from time to time. Like Hirsuitism as a teenager to infertility. Women suffering from PCOS present with a of symptoms associated with menstrual dysfunction and androgen excess. These symptoms generally impact on quality of life. The women suffering from PCOS are at increased risk of obesity, insulin type II diabetes mellitus, cardiovascular disease, infertility, cancer, multiple morbidities and psychological disorders. This review summarizes what the literature has so far provided from guidelines to diagnosis of PCOS according to both Ayurveda as well as Modern. Finally, the review will stress on the various aspects of treatment and screening recommendations currently used in the management of this condition. It is hoped that it will help the women not only to be diagnosed and managed properly for their symptoms like hirsutism, irregular menses etc but also to educate and manage for continuing health risk of insulin resistance throughout their lives. In Ayurveda, there is no direct reference about PCOS but when we go through the Ayurvedic literature there are many references which are nearer to sign and symptoms of PCOS. In Ayurveda, this

condition is not explained as a single disease entity, but given under the headings *Yonivyapada* (genital disorders) and *Artava* (menstrual disorders). After description of eight disorders of *Artava*, destruction of *Artav (Nashtartava)* has been described by Acharya Sushruta and *Vagbhata* respectively. In *Nashtartava* the *Dosha* (*Vata* an *Kapha*) obstruct the passage of *Artava*, thus *Artava* is not visualize properly.

KEYWORDS: PCOS, Hirsuitism, *Artava*, *Nashtartava*, Screening.

INTRODUCTION

Polycystic ovarian syndrome (PCOS) was originally described in 1935 by Stein and Leventhal as a syndrome manifested by amenorrhoea, hirsuitism and obesity associated with enlarged polycystic ovaries. This heterogeneous disorder is characterized by excessive androgen production by the ovaries mainly. PCOS is a multifactorial and polygenic condition.[1] Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder, leading to several health complications, including menstrual dysfunction, infertility, hirsutism, acne, obesity, and metabolic syndrome.[2] However, its pathophysiology remains largely unknown but many believe that PCOS appears to be familial, with its various aspects differentially inherited from one generation to the next. Polycystic Ovarian Syndrome (PCOS), also referred to as hyperandrogenic anovulation (HA), or Stein–Leventhal syndrome (Evans and Riley, 1958), is one of the most common endocrine system disorders that affect women in their reproductive age (Azziz et al., 2004). Described since 1935 by Stein and Leventhal (1935), it represents a condition in which an estimate of 10 small cysts of a diameter ranging between 2 and 9 mm develop on one or both ovaries and/or the ovarian volume in at least one ovary exceeds 10 ml (Balen and Rajkowha, 2003). Systematic screening of women according to the National Institutes of Health (NIH) diagnostic criteria estimated that 4–10% of women of reproductive age suffer from PCOS (Azziz et al., 2004). Although it was previously considered as a disorder of adult women, recent evidence suggests that PCOS is a lifelong syndrome, manifesting since prenatal age.

AIMS: Aim of this review is to evaluate and discuss about Poly cystic ovarian syndrome its prevalence, etiology, sign and symptoms and its management in both perspective Modern as well as Ayurveda.

OBJECTIVES: Is to elaborate the concept regarding Poly cystic ovarian syndrome in both perspective Ayurveda as well as Modern.

MATERIAL AND METHODS: From *Brihatrayee* and other Ayurveda literatures, articles related to Poly cystic ovarian syndrome are compiled. From Modern books and research papers and e-sources compilation are done.

Prevalence: The prevalence in India of polycystic ovary syndrome (PCOS) in women of reproductive age ranges between 9.13% to 36%.[3] According to the Rotterdam diagnostic criteria, the prevalence of PCOS in adolescents varies between a minimum of 3% (Hashemipour et al., 2004) and a maximum of 26% (Driscoll, 2003). However, the prevalence of the disease in children is still considered unknown (Kamangar et al., 2015).

PCOS at different stages of life

The progression of PCOS during different life stages is poorly known because of the paucity of cohort studies with long-term follow-up. A study compared clinical and biochemical parameters of PCOS women and healthy controls who visited a medical center at a mean age of 29 years and returned 6 years later on average. In this longitudinal sample, aging was associated with an increase in the number of regular menstrual cycles, a decrease in serum androgen levels, and a decrease in IR 18. The reasons for this attenuation of PCOS features over time are not clear. Other studies have focused on PCOS manifestations in specific age groups, as detailed below.

PCOS in childhood. The interaction between a genetic predisposition and some prenatal and postnatal environmental factors seems to take part in the pathophysiology of PCOS. Intrauterine growth retardation or small for gestational age (or both) and high levels of androgens during the intrauterine period could lead to an increased production of glucocorticoids which may induce epigenetic modifications and increase the risk of PCOS.[4]

PCOS in adolescence. PCOS is often diagnosed in adolescence. Menstrual irregularity, acne, and hirsutism are the major findings in this age group. However, these features of PCOS overlap with those of normal adolescence. Family history of PCOS, overweight or low birth weight, exposure to androgens during gestation, precocious puberty, obesity, and IR are risk factors that are related to the development of the syndrome. The diagnosis of PCOS during adolescence is based on stricter criteria than in adult women. It requires unequivocal hyperandrogenism (for example, moderate to severe hirsutism or persistent elevation of serum testosterone levels or both) and ovulatory dysfunction that persists for more than 2 years after menarche.[5]

PCOS in postmenopausal women. Women with PCOS persist with hyperandrogenism even after menopausal transition and continue to manifest metabolic alterations and MS with increased risk of cardiovascular disease. Therefore, postmenopausal women with a history of PCOS during the reproductive years may still have manifestations of the syndrome.[6]

Diagnostic Tools for Polycystic Ovary Syndrome

NICHD/NIH Criteria (1990)	ESHRE/ASRM Rotterdam Criteria (2003)	Androgen Excess Society (AES) Criteria (2006)
Hyperandrogenism Oligo ovulation/anovulation Exclusion of other related disorders	Hyperandrogenism Oligo ovulation/anovulation Polycystic ovaries	Hyperandrogenism Oligo ovulation/anovulation Polycystic ovaries Exclusion of other related disorders

(Modified from criteria of the National Institute of Child Health and Human Development (NICHD)/National Institutes of Health (NIH)/European Society of Human Reproduction and Embryology (ESHRE)/American Society for Reproductive Medicine (ASRM)

Pathology

Typically, the ovaries are enlarged.

Ovarian volume is increased more than or equal to 10cm3.

Stroma is increased.

Presence of multiple (more than or equal to 12) follicular cysts measuring about 2-9mm in diameter.

Clinical Features

- The patient complains of increasing obesity (abdominal -50%), menstrual abnormalities (70 %) in the form of oligomenorrhea, amenorrhoea or DUB and infertility. Presence of hirsuitism and acne are the important features (70 %). Virilism is rare.
- Acanthosis nigricans is characterized by specific skin changes due to insulin resistance. The skin changes due to insulin resistance. The skin is thickened and pigmented (grey brown). Commonly, affected sites are nape of the neck, inner thighs, groin and axilla.

  HAIR-EN syndrome in patients with PCOS is characterized by hyperandrogenism, insulin resistance and acanthosis nigricans.

Investigations

- Sonography – Transvaginal sonography is specially useful in obese patient.

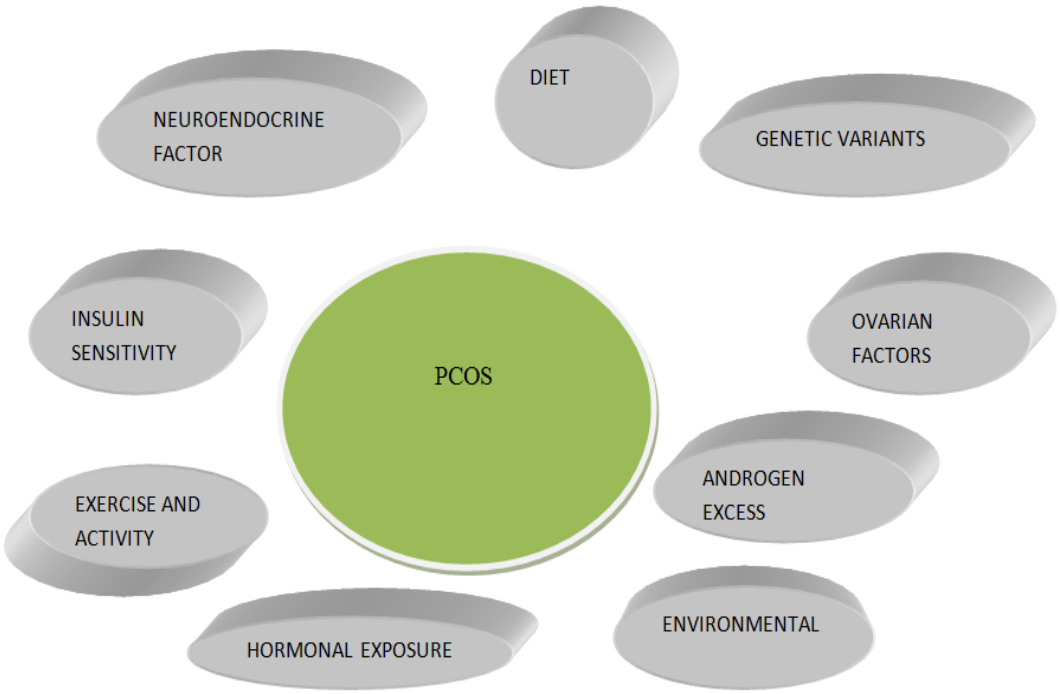
Serum values:

- LH level is elevated and the ratio LH:FSH is > 2:1
- Raised level of estradiol and estrone
- SHBG level is reduced.
- Hyperandrogenism – Andro-stenedione is raised.
- Raised serum testosterone (> 150 ng\dl) and DHEA-S may be marginally elevated.
- Insulin Resistance: Raised fasting insulin levels > 25 microIU\ml and fasting glucose\insulin ratio <4.5 suggests IR (50 %). Levels of serum insulin response >300 microIU\ml at 2 hours postglucose (75gm) load, suggests severe IR.

Laparoscopy – Bilateral polycystic ovaries are characteristic of PCOS.

PATHOPHYSIOLOGY

Previous hypotheses[7]: Many hypotheses emerged trying to explain the pathophysiology of PCOS. Initially, excess intrauterine androgen had been thought to be a main culprit in the development of the disease. Yet recently, human studies showed neither an association between excessive prenatal androgen exposure and the development of PCOS in youth (Hickey et al., 2009) nor an elevation in androgen levels in the cord blood of females born to mothers with PCOS (Anderson et al., 2010). Another hypothesis, the adipose tissue expandability hypothesis, suggested that infants with intra-uterine growth restriction (IUGR) and spontaneous catch-up growth might develop decreased tissue expandability, meaning that they cannot store lipids appropriately in their fat tissues. Consequently, insulin resistance might ensue contributing to PCOS and hyperandrogenemia (de Zegher et al., 2009). However, this does not apply for patients with PCOS who did not have IUGR or had it but without spontaneous catchup growth (Ibáñez et al., 1998, 2009).



(Factors potentially impacting the pathophysiology of PCOS are shown in circles.)

Exact pathophysiology of PCOS is not clearly understood. The following points mentioned below can be considered for the factors for pathophysiology of PCOS.

- Hypothalamic – pituitary compartment abnormality
- Androgen excess
- Anovulation
- Obesity and insulin resistance
- Long term consequences

- **Hypothalamic – pituitary compartment abnormality**
- Increased pulse frequency of GnRH leads to increased pulse frequency of LH. Leptin (a peptide secreted by fat cells and by the ovarian follicle), insulin resistance and hyperandrogenemia are responsible for this.
- GnRH is preferential to LH rather than FSH.
- Increased pulse frequency and amplitude of LH results in tonically elevated level of LH.
- FSH level is not increased. This is mainly due to the negative feedback effect of chronically elevated estrogen and the follicular inhibin.
- Increased free estradiol due to reduced sex hormone binding globulin (SHBG) bears positive feedback relationship to LH.

- The LH: FSH ratio is increased.

ANDROGEN EXCESS

Abnormal regulation of the androgen forming enzyme P450 C17 is thought to be the main cause for excess production of androgens from the ovaries and adrenals. The principal sources of androgens are

Ovary: It produces excess androgens due to- i) stimulation of theca cells by high LH ii) P450 C17 enzyme hyperfunction iii) defective aromatization of androgens to estrogen iv) stimulation of theca cells by IGF-1 (insulin growth factor -1)

Adrenal: Adrenals are stimulated to produce excess androgens by (i) stress (ii) enzyme hyperfunction (iii) associated high prolactin level (20%).

Systemic metabolic alteration (i) Hyperinsulinemia causes: (a) Stimulation of theca cells to produce more androgens. (b) Insulin results in more free IGF-1. By autocrine action, IGF-1 stimulates theca cells to produce androgens. (c) Insulin inhibits hepatic synthesis of SHBG, resulting in more free level of androgens.

Features suggestive of insulin resistance are:

BMI > 25 kg/m², Acanthosis nigricans and waist to hip ratio > 0.85.

(ii) Hyperprolactinemia: In about 20% cases, there may be mild elevation of prolactin level due to increased pulsitivity of GnRH or due to dopamine deficiency or both. The prolactin further stimulates adrenal androgen production.

Anovulation: Because of low FSH level, follicular growth is arrested at different phases of maturation (2-10 mm diameter). The net effect is diminished estradiol and increased inhibin production. Due to elevated LH, there is hypertrophy of theca cells and more androgens are produced either from theca cells or stroma. There is defective FSH induced aromatization of androgens to estrogens. Follicular microenvironment is therefore more androgenic rather than estrogenic. Unless there is estrogenic follicular micro- environment, follicular growth, maturation and ovulation cannot occur. There is huge number of atretic follicles that contribute to increased ovarian stroma (hyperthecosis). LH level is tonically elevated without any surge. LH surge is essential for ovulation to occur.

OBESITY AND INSULIN RESISTANCE

Obesity (central) is recognized as an important contributory factor. Apart from excess production of androgens, obesity is also associated with reduced SHBG. It also induces

insulin resistance and hyper- insulinemia which in turn increases the gonadal androgen production. PCOS is thought to have a dominant mode of inheritance as about 50% of first-degree relatives have PCOS. Etiology of insulin resistance is unknown. Mutations of the insulin receptor gene in the peripheral target tissues and reduced tyrosine autophosphorylation of the insulin receptor, is currently thought to be an important cause. Increased central body fat leads to android obesity

Long-term consequences in a patient suffering from PCOS includes: The excess androgens (mainly androstenedione) either from the ovaries or adrenals are peripherally aromatized to estrone (E,). There is concomitant diminished SHBG. Cumulative excess unbound E, and estrone results in a tonic hyperestrogenic state. There is endometria hyperplasia.

Management: Treatment of PCOS should be proposed not only to alleviate symptoms but also to prevent the occurrence of long-term complications. Combined oral contraceptives and antiandrogens are the standard care to reduce androgen levels and treat symptoms while providing endometrial protection.[8] However, the therapeutic plan should be tailored depending on the desire (or not) of the patient to become pregnant, need for aesthetic approach, and the presence of concomitant metabolic alterations.

The overall goals of therapy of women with PCOS include the mitigation of hyperandrogenic symptoms, management of metabolic abnormalities and reduction of risk factors for type 2 diabetes and cardiovascular disease, prevention of endometrial hyperplasia, planning and obtaining a safe pregnancy if desired, and improving general well-being and quality of life. These goals are ideally achieved by a multidisciplinary team providing patient-centered care.

Management of PCOS needs individualization of the patient. It depends on her presenting symptoms like menstrual disorder, infertility, obesity, hirsutism or combned symptoms. Patient counseling is important.

Treatment is primarily targeted to correct the biochemical abnormalities.

Biochemical abnormalities associated with PCOS
Hyperandrogenism
Hyperinsulinaemia
Hyperlipidaemia
High serum oestrogens
Androgen follicular microenvironment
Hyperprolactinaemia

Insulin resistance Hypersecretion of LH Low serum SHBG Low FSH Low serum progesterone

Weight reduction in obeses patients is the first line of treatment. Body mass index (BMI < 25) improves menstrual disorders, infertility, impaired glucose intolerance (insulin resistance), hyperandrogenemia (hirsuitism, acne) and obesity. Weight reduction (2-5 %) improves the metabolic syndrome and reproductive function.

FERTILITY NOT DESIRED

Management of hyperandrogenism

- Combined oral contraceptive pills are effective. Progestin suppresses LH and estrogen improves SHBG, reducing free testosterone level. Newer progestins (desogestrel) are best suited.
- **Hirsuitism** is due to anovulation, high androgen and insulin levels, decreased hepatic SHBG production and also due to genetic sensitivity of hair follicles to androgens. Correction of metabolic disorder improves it. Antiandrogens (cyproterone acetate, spironolactone, flutamide) may be used.
- **Metabolic disorders**: hyperinsulinaemia (insulin resistance) causes hyperandrogenemia. Insulin resistance is associated with diabetes mellitus, central obesity, dyslipidaemia and hypertension. Metformin increases insulin sensitivity, decreases weight and BMI and reduces LDL cholesterol, blood pressure and the risk of developing diabetes.

Triglycerides level	More than or equal to 150mg\dl
HDL-cholesterol	< 50 mg\dl
Blood pressure	More than or equal to 130\80mm hg
Fasting glucose	More than or equal to 100mg\dl
Abdominal (waste circumference) obesity	>88cm
Presence of three abnormal findings out of the five.	

Hyperinsulinemia: It contributes hyperandrogenemia in women with PCOS. Hyperinsulinemia increases the risk of dyslipidemia, cardiovascular disease and diabetes mellitus. Insulin resistance is the principal abnormality to cause metabolic syndrome.

Endometrial hyperplasia causes abnormal uterine bleeding. Chronic anovulation, hyperestrogenemia, obesity and hyperinsulinemia cause endometrial hyperplasia even

endometrial cancer. Endometrial biopsy may have to be done. Combined oral contraceptive is the treatment of choice to prevent endometrial hyperplasia and abnormal bleeding.

Patient desiring for pregnancy

Anovulation is the common cause of infertility. Improvement of metabolic syndrome is essential.

Ovulation induction is usually achieved by clomiphene citrate following correction of other biochemical abnormalities. In unresponsiveness cases, pure FSH or HMG along with hCG may be administered backed up with monitoring facilities.

Insulin sensitizers: women with PCOS and hyperinsulinemia with BMI> 25, ovulate satisfactorily when clomiphene is combine with metformin. Metformin improves metabolic syndrome by reducing all the parameters: weight, BMI (hyperinsulinaemia), blood pressure and lipid abnormalities. 500mg thrice daily is found to correct the biochemical abnormalities. Pioglita-zone and rosiglitazone are also being used in cases, resistant to metformin.

Surgery: Laparoscopic ovarian drilling (LOD) is done for cases found resistant to medical therapy. It has replaced the conventional wedge resection of the ovaries. Pregnancy rates following ovarian diathermy are higher. Bariatric surgery may be indicated in some PCOS women who are morbidly obese.

Quality of life

PCOS manifests in women at reproductive age when issues such as finding a partner, initiating sex life, and forming a family are often very relevant. Factors that negatively affect physical appearance or femininity or compromise fertility are sources of great anxiety and can lead to imbalances in the psychosexual sphere. The psychological impact of PCOS may even surpass that of chronic diseases such as asthma, diabetes, arthritis, and coronary heart disease 56.

Depression and anxiety are highly prevalent in women with PCOS. Dokras et al. found a fourfold increase in the prevalence of depressive symptoms in patients with the syndrome when compared with controls, even after adjustment for BMI. Daily fatigue and sleep disorders, changes in appetite, and loss of interest in everyday activities were the most common symptoms.[9] Thus, the evaluation of quality of life in women with PCOS is essential for better care and clinical management of these patients.

Ayurveda point of view

PCOS can be considered as a *vata kapha* predominant *vyadhi* at early stages and *tridoshaja vyadhi* at the chronic stage. Different symptomatology in PCOS produced due to the involvement of vitiated *kapha vata* which causes *artavaha srotorodha* and the results in *Artavakshya* or *Anartav* which is expressed as oligomenorrhoea, Amenorrhoea or Hypo-menorrhoea etc. the *rasa* and *rakta dhatu dushti* along with *Agnimandya* observed at this level may lead to the improper formation of *Artava* by the concerned *Dhatwagni.* Here the *pitta* also vitiated due to *agnimandya* and *srotosodha* which leads to improper functioning of *pitta* thus causes improper *pachana* action at *rasa* and *rakta dhatu* resulting in oligomenorrhoea, hypo-menorrhoea etc. Under the description of *Jataharinies*, Kashyap has mentioned *pushpaghni* where the woman menstruates in regular interval but is unable to conceive. The other symptom is corpulent and hairy cheeks. it is incurable *Jataharaini.* Here, the word *pushpa* is used for menstruation which comes at regular interval but is useless means without ovum causing failure to conceive.

PCOS is considered as a metabolic disorder where metabolism in the body is severely affected. The metabolism is the main factor of *Agni* in the body. So, PCOS can be considered as an *Agnidushtijanya vyadhi* also. *Mandagni* and *vishamagni* are the two pathological stage of *agni* seen in PCOS patients.

Nashtartava Doshaiaavrutta Margatvat Artavam Nashyati Striya | (Su. Sa. 2/22) Absence of Artava in female is because of Avarana of Dosha. Commentator *Dalhana* clarifies that *Avarak Doshas* are *Vata* and *Kapha*; as the treatment of *Nashtartava* mentioned here is *Vata Kapha hara*. He also states that increased *pitta* will lead to excessive menstruation, So here *Vata* and *Kapha* should be considered as responsible *Doshas*. Further he says that here *'Nashta'* means which is forming but not seen.

In Ayurveda literature no as such direct correlation of PCOS with any disease is found, though symptomatically menstrual abnormalities amenorrhea or delayed cycle etc. can be correlated with *lakshana* and *Samprapti* of *Nashtartava, Artavakshaya.* According to Acharya Sushruta in *Nashtartava*, *artava* is obstructed by vitiated *Doshas* and in *Artavakshaya* menstruation is delayed, menstrual blood is scanty and associated with pain in Vagina. Mainly *Dushti* of *Rasadi Dhatu* occurs, resulting in improper formation of *Upadhatu* i.e. *Artava.*

In Polycystic ovarian Syndrome mainly ovaries are involved and the description of ovaries in our classics is not found separately, but Acharyas mentioned the term *Beeja-Granthi* which can be correlated with ovary. Acharya Sushruta described the *Artavavaha Srotas* an important *Srotas*/system of female body.[10] Moola of this *Srotas* is *Garbhashya* and *Artva-vaha Dhamanis*. Any injury to *Artava-vaha Srotas* leads to infertility, dyspareunia, and Amenorrhea. Here the *Nashtartava* can be compared to amenorrhea the cardinal feature in PCOS. In a healthy *Beeja granthi* or *Artavavaha Srotasa*, *Beejotsarga* is controlled by *ApanaVata*. *Apana Vayu* along with *Pitta* is responsible for the maturation of follicles by *Pachan karma* of *Pitta* and ovulation/*Beejotsarga*/*Antah Pushpa* &*Artava*/ *Bahir Pushpa Pravartan* karma of *Vata.* Thus due to *Kapha Prakopaka nidana*, Avrana of both the *Vayu* and *Pitta* occurs, thus neither the ovulation takes place nor the maturation of follicles occur leading to the formation of cysts inside ovaries and infertility. Thus ultimately the menstrual abnormalities occur; cycle gets delayed or not occurs monthly due to improper function of H-P-O axis.

***Samprapati Ghatak Dosha*[11]**

Avarita- vata	*Apana* and *vyana*
Avaraka	*Kapha dushya rasa*
Upadhatu	*Artava*
Agni	*Jatharagnimandhya, dhatwagnimandhya*
Srotas	*Rasavaha, artavvaha*
Srotodushti	*Sanga*
Adhisthana	*Garbhasaya, Beejagranthi*

Nidan sevan and vitiated *Doshas* cause dhatu *vaishamyata* vitiated *doshas* have done *kshaya* of *Rasa* and *Rakta dhatu*. *Artava* is an *upadhatu* of *rasa*, less quantity of rasa is responsible for less production of *Artava* Acharya Sushruta said that vitiated *dosha* creates *srotosodhan* and due to *srotorodha*, the quantity of *Artava* ceases or it stops totally.[12]

Symptoms

- ***Yathochit kale adarshanam***: *Yathochita kala* means proper time of appearance of *artava. Adarshanam* means *artava* does not appear at relevant time or it is delayed or disappeared.
- ***Alpata*:** According to *shabdakalpadruma alpa* means *kshudrapramana.* Menstrual blood is reduced in volume.
- ***Yoni vedana*:** *Vitiation* of *vata* causes *yonivedana*.. Due to *artavakshaya* vitiation of *vata* occurs that causes *Yoni vedana*. *Chakrapani* opines that this pain is due to aggravation of

vata dosha caused by loss of *artava* which fills this region, further leads to *khavaigunya* and finally *yoni vedana* occurs. *Yoni vedana* means spastic, radiating and infrequent pain during menstruation.

Some other symptomatology in PCOS which can be corelated with Ayurveda

1. ***Sthaulya* (obesity):** Obesity is considered as main symptom of PCOS and in Ayurveda also *sthaulya* is due to *medodhatu srotosodha* and improper functioning of *Dhatvagni* especially *medo dhatwagni*. Acharya Sushruta says that *sthaulya* and *karshya* arise from *rasadhatu*. Due to *rasa dhatwagnimandya* improper *dhatuparinama* occurs and further formation of *dhatus* got affected and *karsya* occurs. In *sthaulya* improper *dhatwagni* results in *rasadhatu dushti* and due to the *samnaya sidhanta* increased intake of *kapha vardhaka* foods leads to *samanadhatu vruddhi* especially *medodhatu* which further leads to *sthaulya.*
2. ***Atiloma* (hirsuitism):** Ayurveda explained *kesha, loma* and *smashru dosha* as *asthipradoshaja vikaras* and *loma* are considered as the mala of *asthidhatu*. So any deformity at the level of *asthivaha srotas* or *asthi dhatu* itself affects the normal formation of *loma.* In PCOS *asthi dhatu dushti* also occurred due to improper *dhatu parinama* by the impaired *pachaka pitta* or *Dhatwagni.*
3. ***Mukhadooshika* (Acne):** As per Acharya Sushruta, the main *doshas* involved in the pathogenesis are *kapha vata* and *dooshya* is *rakta*. In PCOS acne is seen due to excess androgens in the body due to impaired metabolism of steroid hormones.so here *pitta* vitiation can be seen because *pitta* and *rakta* has *ashraya-ashrayi* relation.
4. ***Nilika* (Acanthuses nigricans):** It is also considered as a *raktapradoshaja vikara* at the level of *twak.* Here *rakta* vitiation causes impaired *bhrajaka pitta* function at the *twak* which leads to discoloration of skin. The *atikarshnyata* is due to the *vata* vitiation along with *pitta.* Acanthuses nigricans in PCOS is due to obesity related Insulin resistance.
5. ***Anapatya* (Infertility):** This can be taken as a symptom or can be taken as complication also. It is due to *artavaha srotodushti* which resulted in improper formation of *Artava* which means ovum. So, anovulation leads to infertility in PCOS.

Name of preparation	Name of the Yoga	Reference
Basti	*Anuvasana basti* *Shatvaryadi uttar basti* *Taila* of *jivaniyadigana dravyas* *shatapushpa taila*	*Ch.Si. 12\18* *Ch. Shi.30\102* *Ka.Kalp.shatapushpa shatavari kalpa*
Varti	*Ikswaku-bija, danti, Chapala, madanaphala, guda, surabija, yavashuka, snuhikshira* in form of *varti*	*Bha. Pra. Chi. 70\22-24* *Yog. Rat. Yo. Vya. Chi.2*
Kwatha	*Tila, karvi, guda* in form of decoction *Krishna tila kwatha* with *guda* *Mishreya methikamulil, Gajara, shatapuspa* etc. in form of decoction	*Bha. Pra. Ch. 70\22-24* *Yog. Ratna. Yonivyapada chikitsa adhyaya* *Harita Samhita*
Churna	*Shatapushpa churna*	*Ka.Shatapuspa shatavarikalpa*
Vati	*Rajah pravartini vati* *Nastapushpantaka rasa*	*Bhai. Ra. 67\58-60* *Bhai. Ra. 67\51-59*
Modaka	*Aswathamuladi modaka* *Agasthi haritaki modaka*	*Bhel. Chi.-4* *H.S. Tru.Sthana 9\63-66*
Taila	*Shatapushpa taila*	*Ka. Shatapuspa shatavari kalpa.*
Ghrita	*Phala grhita* *Maha kalyanaka ghrita* *Kumar kalyana ghrita*	*Bha. Pra. Chi. 70\54-56,58,61* *Yog. Rat.Yo. Vya.Chi. 2*

***Pathyas* for PCOS**

Grains	*Godhooma, kulatha, mudga, tila, yava, methi, ragi, shali rice*
Fruits	*Jambira, dadima, amalaka, jambu, nimbu, pineapple,* watermelon, *kharjura,* orange, papaya.
Vegetables	*Karela,* leafy vegetables, *patola,* carrot, beetroot, *lashuna, ardraka, soorana, sigru,* green banana, papaya, beans.
Taila	Mustard oil, ground nut oil, tila oil
Mamsa	*Rohita matsya, jangala mamsa,* small fishes
Vihara	Exercises, yoga, *pranayama, sooryanamaskar* at early morning.

Apathyas

Food	Maize, *kalaya, chanaka,* potato, *karkati,* excess use of junk, fast and fried food, refined flour like maida, salty foods like chips, Kurkure etc, almond and cashew, tomato, ladies finger
Drinks	Carbonated soft drinks, *madhura dadhi, apyasa, mahisha ksheeram*
Mamsa	Boiler chicken, egg, beef
Taila	Cotton seed oil, *kusumbhataila* (sunflower oil)
Vihara	*Diwaswapna, Ratrijagarana, Atapaseva, Vegadharana, Shoka, Bhaya, Chinta*

CONCLUSION

As from above mentioned paragraph, it can be concluded that PCOS is the emerging issue nowadays. As in the modern treatment the drugs are having more side effects in comparison to Ayurveda medicine. The PCOS is not described in our literature, but can be correlated with *Nashtartava, Artavakshaya, Pushpaghni Jatharini*. There is the need of time to put forward *Chikitsa* of PCOS. *Avaran mukta Prakrita Vayu* and normal functioning *Pitta–Kapha doshas, Rasavaha* and *Artavavaha Srotas* are key factors against *Nashtartava.*

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