



Ayurved Darpan - Journal of Indian Medicine

A Peer Reviewed Journal

Review Article

CONTEMPORARY AND AYURVEDIC PERSPECTIVE OF POLYCYSTIC OVARIAN SYNDROME (PCOS): A CRITICAL REVIEW.

Ashwini Bansode¹, Umesh Agawane², T. Vishala³, Kavita C. Mule⁴

P. G. Scholar¹, Associate Professor^{2,4}, Professor and H.O.D.³,
Department of Prasutitantra and Streeroga, Yashwant Ayurvedic College P.G.T. & R.C., Kodoli, Kolhapur.

*Corresponding Author: Dr. Ashwini Bansode, email: ashwinibansode1@gmail.com

Article Received on: 02/11/2016

Accepted on: 22/12/2016

ABSTRACT:

Polycystic ovary syndrome (PCOS) is a highly prevalent heterogeneous syndrome of clinical and biochemical androgen excess, ovulatory dysfunction and polycystic ovaries. It affects about 6–10% of women worldwide and is thought to be one of the leading causes of female sub-fertility. Androgen excess, insulin resistance play important role in pathogenesis of PCOS along with genetic factors. It is now increasingly perceived as disorder of changed life styles. In *Ayurveda*, PCOS is described under the headings of *Yonivyapad* and *Artavadushti*. This paper reviews the contemporary and ayurvedic perspectives of PCOS to develop holistic approach for the prevention and treatment of PCOS.

KEY WORDS: polycystic ovarian syndrome, *Ayurved*, *Yonivyapad*, insulin resistance.

INTRODUCTION:

Polycystic ovary syndrome (PCOS, is a highly prevalent heterogeneous syndrome of clinical and/or biochemical androgen excess, ovulatory dysfunction and polycystic ovaries (PCO).¹ Women with PCOS are at increased risk of reproductive abnormalities. They also have an increased risk of developing type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). Stein and Leventhal were the first to describe the triad of menstrual dysfunction, PCO and androgenic features.² Worldwide, PCOS affects 6–10% of women³⁻⁶. In India, PCOS affects 9- 22% of young girls in their reproductive age^{7,8} which makes it most common endocrinopathy in women of reproductive age group. The National Institute for Health (NIH) Criteria 1990 was revised in 2003 and Rotterdam criteria⁹ has been adopted world over to diagnose PCOS. However, recently in 2006, Androgen Excess

Society (AES) has come up with a consensus statement, defining PCOS as a hyperandrogenic state and emphasizes the presence of either clinical and/or biochemical features of hyperandrogenism along with other features of PCOS for diagnosis.¹⁰

Pathogenesis:

Androgen excess: Androgen excess is considered by some investigators to be the sine qua non of PCOS; however, only 80–85% of women with clinical hyperandrogenism have PCOS.¹¹

Ovulatory dysfunction and polycystic ovaries: In PCOS, ovarian hyperandrogenism, hyperinsulinemia from insulin resistance and altered intraovarian paracrine signaling can disrupt follicle growth. The consequent follicular arrest in PCOS is accompanied by menstrual irregularity, anovulatory subfertility and the accumulation of small antral follicles within

the periphery of the ovary, giving it a polycystic morphology.^{9, 12} Follicular arrest in PCOS develops when granulosa cells in antral follicles normally begin to express aromatase (at a size of 7 mm).¹³ An excess intraovarian 5 α -reduced androgens has shown to inhibit granulosa cell aromatase activity in vitro and impair follicle growth.¹⁴

Insulin resistance and PCOS: Many women with PCOS have insulin resistance beyond that predicted by their BMI, with 50–70% of these women demonstrating insulin resistance by various measures.¹⁵ Most women with PCOS are young and develop compensatory hyperinsulinemia, with impaired glucose tolerance detectable more readily by oral or intravenous glucose testing than by basal glucose measures.¹⁶ Chen YH et al reported that reduced glucose transporter 4 (GLUT 4) expression is one of the reason of insulin resistance in women with PCOS.¹⁷

Gonadotropin abnormalities: LH hypersecretion increases serum immunoactive and bioactive LH levels in about 70% of women with PCOS,¹⁸ and elevated LH pulse amplitude and frequency induces a twofold to threefold elevation in circulating LH versus FSH levels. Increased LH pulse frequency in PCOS, from enhanced hypothalamic gonadotropin releasing-hormone (GnRH) pulsatile release, occurs owing to reduced steroid hormone negative feedback on LH secretion because of androgen excess.¹⁹

Genetics: With 20–40% of first-degree female relatives of women with PCOS affected by the syndrome, PCOS is more prevalent among family members²⁰ than in the general population.⁴ Genes for which association with PCOS or its component traits have been replicated include fibrillin 3 (*FBN3*) and 17 β -hydroxysteroid dehydrogenase type 6 (*HSD17B6*).^{21, 22}

Environmental factors: Lifestyle profoundly affects the phenotypic expression of PCOS. Weight gain worsens metabolic and reproductive abnormalities of PCOS, as evidenced by increased total and abdominal obesity as well as insulin resistance, menstrual irregularity and hyperandrogenism in women with the most severe PCOS phenotype.²³ A sedentary lifestyle alone also contributes to metabolic dysfunction in PCOS because moderate-

intensity exercise without weight loss improves insulin resistance and decreases body adipose tissue.²⁴ Bisphenol A (BPA), a widely used estrogenic industrial plasticizer accumulation in susceptible women might exaggerate the severity of the PCOS phenotype.²⁵

Clinical features: Women with PCOS often seek care for menstrual disturbances, clinical manifestations of hyperandrogenism, and infertility. Menstrual disturbances commonly observed in PCOS include oligomenorrhea, amenorrhea, and prolonged erratic menstrual bleeding. Hirsutism is a common clinical presentation of hyperandrogenism occurring in up to 70% of women with PCOS.²⁶

Diagnosis: Women with possible PCOS and other androgen excess disorders should undergo a relatively straightforward evaluation. Women to be evaluated would present with unwanted or excess body and/or facial hair growth, irregular menstruation, polycystic-appearing ovaries detected incidentally on ultrasonography, alopecia and/or acne. The evaluation should confirm the presence of features of PCOS and also exclude related disorders.

The **Rotterdam criteria**²⁷ of assessment require the presence of two out of the following three criteria:
Oligomenorrhoea and or/ anovulation
Hyperandrogenism (clinical and / or biochemical)
Polycystic ovaries on ultrasound, with the exclusion of other etiologies such as congenital adrenal hyperplasia, androgen secreting tumours, Cushing syndrome, thyroid dysfunction and hyperprolactinaemia.

Management: Current treatment of PCOS can be summarized as follows:

Treatment of infertility: Weight loss is recommended as first-line therapy for the management of infertility in overweight and obese women with PCOS. This is followed by induction of ovulation (OI) with Clomiphene citrate. Subsequently, administration of insulin sensitizer with Clomiphene is advisable. Gonadotropin therapy and FSH hormone are the next option followed by Gonadotropins with insulin sensitizer. A study reported that treatment with metformin, a

antihyperglycemic drug increased live birth rates in obese women.²⁸

Treatment of androgen-related symptoms:

Combined hormonal contraceptives (CHC) are a good treatment option for those patients that do not wish to become pregnant. Anti-androgenic therapy is used to reduce the masculine effects of testosterone like alopecia, hirsutism.²⁶

Ayurvedic Perspective of PCOS²⁹:

Ayurveda classifies PCOS as a kapha disorder. *Ayurveda* describes Polycystic Ovarian Syndrome to have an equal involvement of the *Dosha*, *Dhatu* and *Upadhatu*. In Ayurveda, PCOS is not defined as single disease, rather its symptoms bear a resemblance to the terminologies defined as 'Anartava' (Amenorrhoea), 'Yonivyapad' (anatomical and physiological disorders of the reproductive system) like - *Arajaska* (Oligomenorrhoea due to vitiation of *Vata Dosha*), *Lohitakshaya* (Oligomenorrhoea due to vitiation of *Vata-Pitta Doshas*), *Vandhya* (infertile), *Pushpaghni Revati* (Idiosyncratic anovulatory menstruation), *Abeejata* (anovulation), *Rajodushti* and *Ashtartava Dushti* (menstrual flow disorder due to vitiation of *Doshas*) etc. The terms *Raja* and *Artava* have been used synonymously or otherwise in the classics. Usually *Raja* is considered as the *Upadhatu* of *Raktadhatu* whereas *Artava* as the *Saptam Dhatu* itself. Similarly, their *Srotasa* (channels) are also two entirely different entities. In the present paper, *Raja* has been considered as the menstrual flow while *Artava* is indicative of the ovum.

CORRELATION OF PCOS WITH CERTAIN AYURVEDIC TERMINOLOGIES:

ArajaskaYonivyapad: When *Pitta* situated in *Yoni* and uterus vitiates *Rakta*, the women becomes extremely emaciated and discolored, this condition is known as *Arajaska*³⁰. *Acharya Chakrapani* has described amenorrhoea as a symptom.

LohitakshayaYonivyapad: The *Nidan Sevan* of *Vata-Pitta Pradhana Aahar-Vihar* causes a vitiation of these *Dosha* resulting in *Rajaksheenata* (scanty menstruation), the lady suffers from burning sensation, emaciation and discoloration. This may be presented in either of the previously discussed ways. Again, a similarity to the contemporary symptom of

menstrual irregularity is noted but it fails to clarify oligo/anovulation.

VandhyaYonivyapad: *Sushrutacharya* quotes this type of *Yonivyapad* presenting as *Nashtartava* (loss of menstruation).³¹ *Charakacharya* states this condition to arise due to loss of ovulation. *Harita* elaborates on six types of *Vandhyayoni*, each having specific features, management and prognosis. One of them is *Anapatya Vandhya* (infertility) wherein *Dhatukshaya* is etiological factor of *Nashtartava*. Here, *Artava* is considered as the *Saptadhatu* or ovum and its loss results in infertility. However this type is incurable. The above mentioned *Anapatya Vandhyayoni* can be fairly compared with PCOS due to the similar features of anovulation and absence/irregularity of menstruation thereby resulting in sterility. However, other clinical features tend to vary.

Abeejata (Anovulation): *Sushrutacharya* states the aetiological factors of *Shukradushti* (vitiation of sperm) in males to be similar to those of *Rajodushti* in females leading to *Abeejata*. The same factors are also responsible for the vitiation of *Doshas* in females causing the vitiation of *Raja/ Artava*. Hence, just as '*Shukramabeejata*' (azoospermia) is seen as a result of vitiation of *Shukra*, a condition of '*Artavaabeejata*' (anovulation) is noted in females due to vitiation of *Artava*. *Charakacharya* too quotes frequent or untimely coitus, over-exercise, unbalanced diet that includes *Ruksha* (dry), *Tikta* (bitter), *Kashaya* (astringent), *Atilavana* (excessively salty), *Amla* (sour) and *Ushna* (hot) *Aahar*, as also *Chinta / Shoka* (stress-related tension), *Bhaya* (fear), *Krodha* (anger) and *Aghata* i.e. injuries due to *Shastra* (weapon) or *Kshara* (alkali) as the causative factors of *Shukradushti*.³⁰ These can be correlated with the current lifestyle changes.

Ashtartava Dushti: *Vagbhatacharya* states that like *Shukra*, *Artava* can too be vitiated by the *Doshas* resulting in eight types of *Artavadushti*. Such vitiation leads to *Abeejata*.³²

Rajodushti: This terminology, put forth by *Sushrutacharya* is a result of the vitiation of *Raja* by the *Dosha*, primarily *Vata* and *Pitta* resulting in its *Ksheenata* (Oligomenorrhoea). The other clinical features of PCOS are however not observed.

AYURVEDIC MANAGEMENT OF PCOS:

1) "Nidana Parivarjana"³³ (avoid the causative factors) is said to be the very first step towards the management of PCOS. As Agnimandya, Medovridhi, Apana Vayu and Kapha dushti plays the major role in the pathogenesis of the syndrome, so taking above fact into the consideration, Pathya Ahara-Vihara (dietary regimen & exercise) is to be used.

2) For Agnimandya and Aampachana, use of Trikatu Churna³⁴, Chitrakadi Gutika³⁵, Shadushana Churna³⁶, Haritaki Churna (*Terminalia Chebula*), Hingwashtaka Churna³⁷ is to be done in order to palliate the Srotovarodha and to facilitate the Apana-Vatanulomana.

3) For Medovridhi (*Obesity*), use of Takrarishta, Madhu like lekhanadravyas (scrapping agents) along with Yava, yavaka, kulattha etc. as aahara (diet) is mentioned by Acharya Charaka in chikitsa of Atistula³⁸ (obese). Moreover, lifestyle modification as well as regular exercise is also emphasized.

4) To remove the Sanga (obstruction) of Aartavavah Srotas, Uttar-Basti³⁹ (douche) is given with Dhanvantari Taila.⁴⁰

5) Vamana Karma (emesis) - as it alleviates the Srotovarodha by eliminating vitiated Kapha. As Kapha is soumya in prakriti, decrease in Kapha consequently increases Aartava of Aagneya nature.⁴¹

6) Shatpushpa and Shatavari Churna⁴² (*Asparagus racemosus* Wild⁴³) are to be used in females with deficiency or loss of Aartava, women getting their menstruation but not conceiving. 7) Kanchanara Guggulu⁴⁴, Sukumara Ghrita⁴⁵ for reducing the size of formed ovarian cysts.

8) Pathadi Kwath mentioned by Acharya Sushruta in Vatakaphaja Aartava dushti³⁹ given orally along with the matrabasti of Shatpushpa taila⁴⁶ after the cessation of menstrual cycle for seven days is found efficient due to its properties of Aampachana, agnideepana, Vatanulomana, Srotoshodhana and Vata-Kaphashamana.

12) Regular practice of Yoga i.e. Uttanapadasana, Sarvangasana, Halasana, Mayoorasana, Suryanamaskara, Vakrasana and Sheersasana in amenorrhoea⁴⁷.

The main objective of the chikitsa is-

- To flame the Jathragni and dhatvagni with the use of Deepana- pachana dravyas to correct the Agnimandya.

- To remove the Sanga of Aartava-vaha srotas and Srotoshodhana through various Samshodhana karmas to balance the imbalanced doshas i.e. reduction of Kapha and Anulomana of Apana Vata; to nourish the dushta dhatus and to regulate the irregular menstrual cycle.
- To reduce the weight through Lekhana dravyas, Pathya aahara- vihara and regular exercise to regulate the hormonal imbalance
- To reduce the size of already formed ovarian cysts through Kanchanara Guggulu & Sukumara Ghrita as Kanchanara is very useful in treating extra growths or tumors.
- To enhance the chances of conception with the use of Shatpushpa and Shatavari Churna. As Shatavari (*Asparagus racemosus* Wild) is known for its phytoestrogenic properties.

CONCLUSION:

Integration of *Ayurvedic* treatments into modern medical approaches for PCOS has the potential to improve patient outcomes. Encouraging results can be obtained with Shastrokta formulations, Panchakarma, Pathya aahara and regular exercise due to its holistic approach towards-Samprapti-Vighatana.

REFERENCES:

1. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: etiology, pathogenesis and diagnosis. *Nat Rev Endocrinol.* 2011;7(4):219-31.
2. Stein IF, Leventhal ML. Amenorrhoea associated with bilateral polycystic ovaries. *American Journal of Obstetrics & Gynecology.* 29(2):181-91.
3. Asuncion M, Calvo RM, San Millan JL, Sancho J, Avila S, Escobar-Morreale HF. A prospective study of the prevalence of the polycystic ovary syndrome in unselected Caucasian women from Spain. *The Journal of clinical endocrinology and metabolism.* 2000;85(7):2434-8.
4. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. *The Journal of clinical endocrinology and metabolism.* 2004;89(6):2745-9.
5. Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, Azziz R. Prevalence of the polycystic ovary syndrome in unselected black and white women of the southeastern United

- States: a prospective study. *The Journal of clinical endocrinology and metabolism*. 1998;83(9):3078-82.
6. Michelmore KF, Balen AH, Dunger DB, Vessey MP. Polycystic ovaries and associated clinical and biochemical features in young women. *Clinical endocrinology*. 1999;51(6):779-86.
 7. Nidhi R, Padmalatha V, Nagarathna R, Amritanshu R. Prevalence of polycystic ovarian syndrome in Indian adolescents. *Journal of pediatric and adolescent gynecology*. 2011;24(4):223-7.
 8. Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, Vaidya R. A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. *Indian journal of endocrinology and metabolism*. 2014;18(3):317-24.
 9. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertility and sterility*. 2004;81(1):19-25.
 10. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. *The Journal of clinical endocrinology and metabolism*. 2006;91(11):4237-45.
 11. Azziz R, Sanchez LA, Knochenhauer ES, Moran C, Lazenby J, Stephens KC, et al. Androgen excess in women: experience with over 1000 consecutive patients. *The Journal of clinical endocrinology and metabolism*. 2004;89(2):453-62.
 12. Jonard S, Robert Y, Cortet-Rudelli C, Pigny P, Decanter C, Dewailly D. Ultrasound examination of polycystic ovaries: is it worth counting the follicles? *Human reproduction (Oxford, England)*. 2003;18(3):598-603.
 13. Gougeon A. Regulation of ovarian follicular development in primates: facts and hypotheses. *Endocrine reviews*. 1996;17(2):121-55.
 14. Agarwal SK, Judd HL, Magoffin DA. A mechanism for the suppression of estrogen production in polycystic ovary syndrome. *The Journal of clinical endocrinology and metabolism*. 1996;81(10):3686-91.
 15. DeUgarte CM, Bartolucci AA, Azziz R. Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment. *Fertility and sterility*. 2005;83(5):1454-60.
 16. Legro RS, Kunesman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. *The Journal of clinical endocrinology and metabolism*. 1999;84(1):165-9.
 17. Chen YH, Heneidi S, Lee JM, Layman LC, Stepp DW, Gamboa GM, et al. miRNA-93 inhibits GLUT4 and is overexpressed in adipose tissue of polycystic ovary syndrome patients and women with insulin resistance. *Diabetes*. 2013;62(7):2278-86.
 18. Lobo, R. A. in *Infertility, Contraception, and Reproductive Endocrinology 3rd edn* (eds Mishell, D. R. Jr, Davajan, V. & Lobo, R. A.) 447-487 (Blackwell Scientific Publications, Cambridge, 1991).
 19. Blank SK, McCartney CR, Marshall JC. The origins and sequelae of abnormal neuroendocrine function in polycystic ovary syndrome. *Human reproduction update*. 2006;12(4):351-61.
 20. Kahsar-Miller MD, Nixon C, Boots LR, Go RC, Azziz R. Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS. *Fertility and sterility*. 2001;75(1):53-8.
 21. Urbanek M, Woodroffe A, Ewens KG, Diamanti-Kandarakis E, Legro RS, Strauss JF, 3rd, et al. Candidate gene region for polycystic ovary syndrome on chromosome 19p13.2. *The Journal of clinical endocrinology and metabolism*. 2005;90(12):6623-9.
 22. Jones MR, Mathur R, Cui J, Guo X, Azziz R, Goodarzi MO. Independent confirmation of association between metabolic phenotypes of polycystic ovary syndrome and variation in the type 6 17beta-hydroxysteroid dehydrogenase gene. *The Journal of clinical endocrinology and metabolism*. 2009;94(12):5034-8.
 23. Carmina E, Bucchieri S, Mansueto P, Rini G, Ferin M, Lobo RA. Circulating levels of adipose products and differences in fat distribution in the ovulatory and anovulatory phenotypes of polycystic ovary syndrome. *Fertility and sterility*. 2009;91(4 Suppl):1332-5.
 24. Bruner B, Chad K, Chizen D. Effects of exercise and nutritional counseling in women with polycystic ovary syndrome. *Applied physiology, nutrition, and metabolism = Physiologie*

- appliance, nutrition et metabolisme. 2006;31(4):384-91.
25. Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM, et al. Endocrine-disrupting chemicals: an Endocrine Society scientific statement. *Endocrine reviews*. 2009;30(4):293-342.
26. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. *Clinical epidemiology*. 2013;6:1-13.
27. Boyle J, Teede H. Polycystic ovary syndrome An update. *Australian Family Physician*. 2012;41:752-6.
28. Morin-Papunen L, Rantala AS, Unkila-Kallio L, Tiitinen A, Hippelainen M, Perheentupa A, et al. Metformin improves pregnancy and live-birth rates in women with polycystic ovary syndrome (PCOS): a multicenter, double-blind, placebo-controlled randomized trial. *The Journal of clinical endocrinology and metabolism*. 2012;97(5):1492-500.
29. Dr. Kadam Ruta et al. Contemporary and Traditional Perspectives of Polycystic Ovarian Syndrome (PCOS): A Critical Review. *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)* 2014;.Volume 13,(Issue 9 Ver. VI (Sep.)): PP 89-98.
30. Agnivesha CS, revised by Charaka & Dridabala with Elaborated vidyotini hindi commentary by Pt. Kashinath shastri Dr.Gorakhanath chaturvedi Chikitsasthana 30/17 pg no. 842 Edition 2009 Chaukhambha Bharati Academy, Varanasi.
31. Sushruta Sushruta Samhita, Edited with ayurveda tatva sandipika by Kaviraja Ambikadutt shastri, edition 2011 Uttartantra 38/10 pg no.203.
32. Vagbhata, Astanga Hridayam Edited with the vidyotini hindi commentary by atrideva gupta edited by Vaidya Yadunandana Upadhyaya, edition 2012 chaukhambha prakashana, Varanasi,Sharirasthana 1/10 pg no.231.
33. Dalhana ; Sushruta Samhita, Nibandh Sangraha and Nyaya Chandrika commentary, editor Jadavji T, Uttara tantra 1/25; Chaukhamba Sanskrit Sansthana, Varanasi; ed-2014; p-597.
34. Ibid. Sushruta Samhita Sutrasthana 38/59: p-168.
35. Shastri PK & Chaturvedi GN; Charaka Samhita Vidyotini hindi commentary, Chikitsasthana 15/96; Chaukhamba Sanskrit Sansthana, Varanasi; reprinted ed -2012; p-467.
36. Indradeva T; Chakradatta Vaidayaprabha commentary, editor Prof. Ramanath Dwivedy, Agnimandya chikitsa 6/9; Chaukhamba Sanskrit Sansthana, Varanasi; reprinted ed- 2014; p-69.
37. Ibid. Chakradatta Agnimandya chikitsa 6/2; p-69.
38. Shastri PK & Chaturvedi GN; Charaka Samhita Vidyotini hindi commentary, Sutrasthana 21/21-27; Chaukhamba Sanskrit Sansthana, Varanasi; reprinted ed -2012; p-415.
39. Dalhana; Sushruta Samhita Nibandha Sangraha and Nyaya Chandrika commentary, editor Jadavji T, Sharirasthana 2/14; Chaukhamba Sanskrit Sansthana, Varanasi; ed-2014; p- 345.
40. Kamidi Vijaya Kumari NM. Study of uttarvasthi with Dhanvantari Tailain Female infertility. *Int J Res Ayurveda Pharm*. 2013;4(2):257-61.
41. Ibid. Sushruta Samhita; Sutrasthana 14/7; p-59.
42. Sharma PH; Kashyapa Samhita, commentary; Kalpasthana Shatapushpashatavari-kalpadhyaya shlok no- 13; Chaukhamba Sanskrit Sansthana, Varanasi; ed- 2012; p- 186.
43. The Ayurvedic Pharmacopoeia of India. Part I vol IV. Govt. of India, Ministry of Health and Family Welfare, Dept. of AYUSH, New Delhi, 2004 ; P. no.- 108.
44. Shrivastava Shailja; Sharangadhara Samhita Jeevanprada commentary, Madhyama Khanda 7/95-100; Chaukhamba Sanskrit Sansthana, Varanasi; ed- 2011; p-206.
45. Gupta KA; Ashtanga Hridaya; Chikitsasthana- 13/41-47; Chaukhamba Sanskrit Sansthana Varanasi; edi- 2012; p-515.
46. Patel K.D. Dei L, Donga SB, Anand N. Effect of Satpushpa Tail Matrabasti and Pathadi kwath on polycystic ovarian disease AYU [Serial online] 2012;33:243.
47. Ibid. Yoga ka vaijanika rahasya evam yaugika chikitsa, edition-1999; p- 146.

Cite this article as:

[Ashwini Bansode, Umesh Agawane, T. Vishala, Kavita C. Mule. Contemporary And Ayurvedic Perspective Of Polycystic Ovarian Syndrome \(Pcos\): A Critical Review. Ayurved Darpan - Journal of Indian Medicine, October - December 2016, Vol. 1 Issue 4](#)