



## AN AYURVEDIC ANALYTICAL REVIEW OF GENETIC THEORY AND PATHOGENESIS PERTAINING TO POLYCYSTIC OVARIAN SYNDROME (ARTAVA KSHAYA)

\*<sup>1</sup>Dr. Pooja Ravindra Kadu, <sup>2</sup>Dr. Anjali Vijay Jadhav, <sup>3</sup>Dr. Prathamesh Pradeep Kashikar

\*<sup>1</sup>Assistant Professor, Prasuti Tantra and Stri Roga Department, PDEA's College of Ayurved & Research Centre, Nigdi, Pune.

<sup>2</sup>Professor & HOD, Prasuti Tantra and Stri Roga Department, PDEA's College of Ayurved & Research Centre, Nigdi, Pune.

<sup>3</sup>Assistant Professor, PhD Scholar, Shalyatantra Department, Bhimashankar Ayurved College, Manchar, Pune.



\*Corresponding Author: Dr. Pooja Ravindra Kadu

Assistant Professor, Prasuti Tantra and Stri Roga Department, PDEA's College of Ayurved & Research Centre, Nigdi, Pune.

DOI: <https://doi.org/10.5281/zenodo.20019915>

**How to cite this Article:** \*<sup>1</sup>Dr. Pooja Ravindra Kadu, <sup>2</sup>Dr. Anjali Vijay Jadhav, <sup>3</sup>Dr. Prathamesh Pradeep Kashikar (2026). AN Ayurvedic Analytical Review Of Genetic Theory And Pathogenesis Pertaining To Polycystic Ovarian Syndrome (Artava Kshaya). World Journal of Pharmaceutical and Life Science, 12(5), 303-308.

This work is licensed under Creative Commons Attribution 4.0 International license.



Article Received on 05/04/2026

Article Revised on 25/04/2026

Article Published on 04/05/2026

### ABSTRACT

An estimated six percent to twenty percent of women in their reproductive years have polycystic ovarian syndrome (PCOS), a common heterogeneous endocrine disorder. Early puberty is when clinical symptoms first appear. These include irregular menstrual cycles, anovulation, acne, oligomenorrhea/amenorrhea, hirsutism, and often infertility. The pathophysiology of PCOS is still difficult to understand despite recent technological advancements in science, and the majority of clinical data that has been published thus far has only been related to adult women. After that, adult women and teenage girls are most likely to meet the Rotterdam criteria. Adolescent girls are diagnosed based on traditional criteria such as irregular menstruation, clinical hyperandrogenism, and/or hyperandrogenaemia. On the other hand, adolescent girls have the least significant pelvic ultrasound results compared to adult women. Adolescent girls and women with PCOS are also more likely to suffer from mental health conditions like depression, anxiety, and bipolar disorder. Ayurveda places a high value on women's health, and literature frequently discusses PCOS symptoms and indicators in a similar manner, which is why PCOS and *Artava kshaya* are related. The goal of this review is to present a thorough understanding of *Artava kshaya* and PCOS pathogenesis. The work done here will make it possible to identify girls and adult women who have a high risk of developing PCOS earlier. Prompt adherence to tailored therapeutic interventions will enhance quality of life, prevent related comorbidities, and improve PCOS overall management. The different etiological factors and screening guidelines currently in use to prevent and treat PCOS are highlighted in this review.

**KEYWORDS:** PCOS, Ayurveda, *Artava kshaya*, Genetics, Pathogenesis.

### INTRODUCTION

A common endocrinologic and reproductive condition, polycystic ovarian syndrome (PCOS) affects six to ten percent of women.<sup>[1]</sup> PCOS is also regarded as a multifactorial disease with a range of symptoms that affects adolescent and postmenopausal women in addition to women of childbearing age. Hyperandrogenism, polycystic ovaries, and ovarian dysfunction are the three primary phenotypic traits of

this illness.<sup>[2]</sup> Additionally, this syndrome has been linked to metabolic problems such as obesity, type 2 diabetes mellitus (T2DM), and insulin resistance (found in 60–80% of women with PCOS hyperinsulinemia).<sup>[3]</sup> Hormonal fluctuations, lifestyle choices, and genetics are the main contributing factors to PCOS. In 1935, Stein and Leventhal were the first to provide a more thorough description of PCOS.<sup>[4]</sup> It has generated a great deal of scientific discussion due to its diverse clinical

manifestations, unclear aetiology, complicated pathophysiology, and inadequate diagnosis.<sup>[5,6]</sup> In clinical endocrinology, the diagnosis of PCOS is still debatable. The National Institutes of Health (NIH) criteria were developed in 1990 to provide a comprehensive and descriptive definition for the diagnosis of PCOS.<sup>[7]</sup> After that, in the course of 2003, a Rotterdam workshop formulated the Rotterdam criteria<sup>[8]</sup>, a new diagnostic criterion. Two of the three conditions—oligomenorrhea/anovulation, clinical /biochemical hyperandrogenism, and polycystic ovaries ( $\geq 12$  follicles in each ovary measuring 2–9 mm)—must be present for this criterion to be met.<sup>[9]</sup> PCOS is a complicated condition that affects several organ systems and first appears in the early stages of puberty. Due to innate lifestyle changes followed by excessive stress from a variety of sources, the factors involved in the pathophysiology of PCOS are still growing. According to a recent study conducted on animal models, testosterone plays a significant role in the pathophysiology of PCOS. Ectopic fat storage and adipocyte androgen biosynthesis are important factors that predispose people to PCOS.<sup>[10]</sup> To identify females who are at risk of developing PCOS, it is crucial to pay the focus to the history, physical examination, and laboratory results. Treatment of clinical features and comorbidities is critical to these patients' health and self-esteem, even though delaying diagnostic labelling may be appropriate. Oligomenorrhea, hirsutism, excessive acne, and hair loss are common clinical features. It causes serious mental health issues in adolescence, including depression and anxiety. The most common cause of anovulatory infertility in women is PCOS. Impaired glucose tolerance, Type 2 Diabetes Mellitus, and obesity are among the metabolic effects.<sup>[11]</sup>

### INCIDENCE

PCOS, or polycystic ovarian syndrome, is a common endocrine condition that primarily affects women who are fertile. Globally, it affects 4% to 20% of women who are fertile.<sup>[12]</sup> The incidence of PCOS, a common endocrine disorder affecting women, ranges from 2.2% to 26% in India. The majority of studies have focused on adult women between the ages of 18 and 45.<sup>[13]</sup> PCOS symptoms negatively impact a woman's fertility and reproductive health and affect not only women of childbearing age but also adolescents and postmenopausal women.<sup>[14,15]</sup> It also contributes significantly to metabolic and cardiovascular morbidity when combined with other lifestyle diseases.<sup>[16]</sup> Although several theories have been put forth, ranging from genetic susceptibility to environmental exposure, both in utero and in postnatal life, the precise etiopathogenesis of PCOS is still an area of active research and not fully developed due to the heterogeneity in the representation of clinical and biochemical features.<sup>[17]</sup> Numerous potential genes have been linked to PCOS in the field of genetics. Recent clinical research has consistently shown that metabolic morbidities such as insulin resistance, glucose intolerance, and

cardiovascular risk are linked to hyperandrogenism in Indian PCOS women.

### AYURVEDA PERSPECTIVE

Ancient medical wisdom can be found in Ayurveda. Many references to PCOS signs and symptoms can be found in Ayurvedic literature. In contrast, PCOS is explained under the headings of *Aartavdusti*<sup>[18]</sup>, *Artava kshaya*<sup>[19]</sup>, and *Nashta Artava* rather than as a single disease entity. *Samprapti* (pathophysiology) of *Artava kshaya* was found to be influenced by various *Ahara*, *Vihara*, *Agantuja*, *Mansik Nidana* (aetiology), *Prakruti* (nature), and *Sthana* (site).<sup>[20]</sup> Menstrual and ovulatory aspects of the disease have received adequate attention in *Artava kshaya*. Thus, it can be connected to PCOS symptoms. *Anukta Vyadhi* is another principle that Acharya Charaka describes.<sup>[21]</sup> According to this principle, if a disease's name is not mentioned in an Ayurvedic text, it may be the correlated basis of its signs and symptoms, and treatment may be administered based on its symptomatology. Consequently, there is a correlation between PCOS and *Artava Kshaya*.

### NIDANA OF ARTAVA KSHAYA

Vitiating of the *Vata* and *Kapha doshas* is the cause of *Artava Kshaya*. *Sroto avarodha* will cause the *yathochithakale Artava adarshana* (menses not coming on time), *alpata* (less blood amount), *yoni vedana* (pain), and irregular and sparse menstrual flow. In this condition, *shodhana* of *Artavavaha Srotas* eliminates *Sroto avarodha* and restores the regular menstrual flow, particularly with the aid of *Pitta Vardhaka* (Agnaya) *Dravyas*.<sup>[22]</sup> *Garbhashaya* and *Artavavahi Dhamani* are the *mula* of *Artavavaha Srotas*, where an injury results in amenorrhoea, dyspareunia, and infertility. The tangible expression of menstrual cycle disorders has been explained by various authors. *Acharya Kashyapa* claims that *Atiushana annapana sevana jivaraktaskalana* happens following proper *Snehana* and *Swedan* in a *Mridu Koshta Vyakti Adhika Aushadha sevana*. Consequently, *Pralapa*, *Unmad*, *Hikka*, *Bejoupghata*, and *Pushpopaghata (Artava dushti)*<sup>[23–25]</sup> are caused by *Vata Prakopa*. *Acharya Sushruta* claims that *Vata-Kapha* causes *avarana* to *Artavvahasrotas*.<sup>[26,27]</sup> *Pitta* is excluded because it causes *Artava vriddhi*. Additionally, according to *A. Sushruta*, *Artavavaha srotas* trauma or injury results in *Vandhyatwa* (infertility), *Maithuna asahishnuta* (dyspareunia), and *Artavnasha*.<sup>[28]</sup>

### DISEASE PROGRESSION

All of the body's hormonal, neurological, and metabolic processes fall under the purview of *Kayagni*, *Bhutagni*, and *Dhatwagni*. As a result, Ayurveda emphasizes maintaining appropriate *Agni* as a key component in the treatment of numerous illnesses. *Mandagni* leads to *Artava Kshaya* because of *Kapha Vardhaka* *Ahara* and *Vihara*. *Ama* is formed as a result of this *Mandagni*.<sup>[29]</sup> *Kapha* and *Ama* first vitiate the *Rasa dhatu*. *Artava* is vitiated because it is the *Upadhatu* of *Rasa Dhatu*. *Anartava* (amenorrhoea) or *Alpaartava* (oligomenorrhoea)

result from the vitiated *Kapha* and *Ama* obstructing the *Artava* in the *Artavavaha srotas*.<sup>[30]</sup> As *Kapha*, *Ama*, and *Medas* share similar traits, aggravated *Kapha* and *Ama* with vitiated *Rasa dhatu* gravitate toward *Medo dhatu*. They are therefore drawn to one another. Along with *Rasa dhatu*, *Medo dhatu* is among the first *dhatu*s to represent a *Kapha* annoyance.

#### GENETIC MECHANISM:

The complicated, multifaceted pathophysiology of PCOS is still being studied. Increased luteinizing hormone (LH), decreased FSH, and aberrant gonadotropin-releasing hormone (GnRH) are some of the genetic mechanisms implicated in the pathophysiology of PCOS. The genetic pathophysiology of this disease is significantly influenced by elevated levels of ovarian follicles to FSH, elevated anti-Mullerian hormone (AMH), and elevated secretion of testosterone, oestradiol, and dehydroepiandrosterone (DHEA). The main risk factor for the expression of insulin resistance and metabolic phenotype in PCOS is abdominal fat deposition.<sup>[31]</sup> Thus, the metabolic effects of PCOS are triggered by adipocyte dysfunction and insulin resistance at the post-receptor level. The pathophysiological mechanisms—biochemical and hormonal components—that underlie PCOS have been covered in detail below.

Leptin is a key mediator between women's reproductive health and nutritional status. It has been demonstrated that in PCOS patients, increased leptin levels are linked to indicators of insulin resistance. PCOS women may have higher serum testosterone and leptin levels if their obesity is under control. Another study found no significant correlation between serum testosterone levels and leptin levels, but it did show that high leptin in PCOS women was independent of obesity status.<sup>[32]</sup> Research studies states that the proteomics of follicular fluid from women with PCOS and controls receiving in vitro fertilization treatment revealed altered levels of proteins involved in extracellular matrix remodelling, complement coagulation cascade, vasculature development, angiogenesis, lipid transport, and metabolism.<sup>[33]</sup>

Also Recent studies have confirmed the role of oxidative stress in the pathophysiology of a number of reproductive diseases and conditions, such as PCOS, infertility, and recurrent abortions.<sup>[34]</sup> PCOS women's plasma amino acid levels were found to be significantly abnormal (lower levels of methionine, cysteine, isoleucine, phenylalanine, valine, tyrosine, proline, glycine, lysine, and histidine and higher levels of arginine and alanine) when compared to PCOS control patients. The researchers hypothesized that this was a sign of increased oxidative and metabolic stress in PCOS women. Women with PCOS had reduced arginine bioavailability, which was linked to low nitric oxide levels, elevated oxidative stress, and consequently decreased regulatory T cells (Treg cells).<sup>[35]</sup>

Numerous candidate genes for PCOS have been assessed since genomic science has emerged as a major diagnostic field in recent years. P1A1 (CYP1A1), CYP11A, CYP17A1, CYP19, 17 $\beta$ -hydroxysteroid dehydrogenase (HSD17B6), androgen receptor (AR), sex hormone-binding globulin (SHBG), insulin receptor (INSR), insulin receptor substrate 1 (IRS1), PPAR- $\gamma$ ,<sup>[36]</sup> follicle-stimulating hormone receptor (FSHR), luteinizing hormone receptor type 2 (AMHR2), and interleukin (IL).<sup>[37]</sup>

#### EVALUATION OF PCOS

PCOS aggravation can be prevented with early diagnosis. Based on certain predetermined criteria, PCOS can be diagnosed. The initial diagnostic criteria for PCOS were established in 1990 at a conference organized by the National Institutes of Health (NIH).<sup>[38]</sup> Following that, a workshop in Rotterdam developed the Rotterdam criteria, a new diagnostic standard, in 2003. The Rotterdam criteria are still the suggested method for diagnosing PCOS.<sup>[39]</sup> This criterion requires the presence of two conditions out of the three oligomenorrhoea and/or anovulation, hyperandrogenism (clinical and/or biochemical), PCOS diagnosed by USG.

#### DISCUSSION

There are several factors that contribute to PCOS. Genetic (anuvanshika), *Agni* (food digestion), *Dosha Dhatu* imbalance (vitiation of *Vata* and PCOS can be brought on by *Kapha Dosha* and *dhatu*), lifestyle (*Dincharyajanya*), diet (*Ahara janya*), metabolism, hormones, stress (*Avsaad janya*), and their combinations. PCOS is a multifactorial disorder that is known to be caused by genetic variants. Several candidate genes (CYP11a, CYP21) and their variants have been found to be at risk for PCOS (*Artava Kshaya*) in a variety of populations worldwide. Since there are currently very few published Indian studies on the genetic risk assessment for PCOS in conjunction with Ayurveda, it is imperative to conduct an analysis of the genetic risk factors for PCOS with regard to the aetiologies mentioned in Ayurveda.

Research of this kind would aid in the prevention of PCOS, especially in our nation's rural and tribal areas.<sup>[40]</sup> *Agni* is a crucial component of digestion and metabolism in our bodies, according to Ayurveda. *Agni* is responsible for the digestion, absorption, and assimilation of ingested food, which is necessary for life to continue. The term "*Agni*" refers to the breakdown of food and metabolic products in Ayurveda. *Agni* transforms food into energy, which powers all of our body's essential processes. *Dehagni* is therefore regarded by Ayurveda as the source of life, complexion, strength, health, nourishment, lustre, *oja*, *teja* (energy), and *prana* (life energy).<sup>[41]</sup> Any disruption in the biological process of *Agni* results in the development of disease. In PCOS, the metabolism rate already declines due to decreased *Agni*, which causes affected women to become more obese and exhibit severe PCOS symptoms.<sup>[42]</sup>

*Samanya* (general) and *Vishesha* (specific) are the two categories. *Artava* is regarded as *Upadhatu* in the classics of Ayurveda. *Artava Kshaya* is caused by the same factors that cause *Kshaya* of *Dhatu*s and *Upadhatu*. *Kshaya* of *Dhatu*s can occur in either *Anuloma* (from *Rasa* to *Shukra Kshaya*) or *Pratiloma Kshaya* (from *Shukra* to *Rasa Kshaya*). Since *Artava* is *Upadhatu* of *Rasa*, the *Kshaya* of *Uttara Dhatu* in *Artava Kshaya* results from the *Kshaya* of *Purva Dhatu*, i.e., *Rasa Kshaya* leads to *Uttara Dhatu Kshaya* in addition to *Artava Kshaya*. Thus, one of the causes of *Artava Kshaya* is *Rasa Kshaya*. *Artava kshaya* (PCOS) is primarily caused by vitiation of *Vata* and *Kapha Dosha* and *dhatu*s due to excessive *Vata Kapha vardhaka Ahara Vihara*.<sup>[43]</sup>

These days, it's common to be exposed to a variety of chemicals, either intentionally (cosmetics, household cleaning products, chemotherapeutics, etc.) or unintentionally (pesticides, vehicle exhausts, industrial pollutants, etc.).<sup>[44]</sup> Perfume, sunscreen, deodorant, hair dye, and other personal care products that have become essential grooming ingredients are major contributors to the rising incidence of PCOS. Additionally, oral contraceptives, progestin, antiandrogens, ovulation induction drugs, and surgical procedures are still common treatments in today's medical system. These treatments have a number of adverse effects, including hepatotoxicity, nephrotoxicity, depression, weight gain, and hot flashes. PCOS pathogenesis may be influenced by thyroid dysfunction, hyperprolactinemia, androgen-secreting tumours, Cushing's syndrome (a condition linked to elevated cortisol levels), and congenital adrenal hyperplasia. PCOS has been linked to hormonal imbalance and chemical exposure.<sup>[45]</sup> The pathophysiology of PCOD is also significantly influenced by other causative factors summarized above, such as lifestyle (*Dinacharya janya*), diet (*Ahara janya*), metabolism, and stress (*Avsaad janya*).

As a result, efforts were made to assess these etiological factors as well as genetic factors related to PCOS in order to gain a better understanding of the disease's etiopathology.

In another sense, the main goal of Ayurveda is to provide all-encompassing care by adjusting the *Doshik*, *Dhatu*, and *Agni*. Controlling the three aggravated *Doshas* is made easier with Ayurvedic treatment. It helps to regulate *Artava Dhatu* by giving the reproductive system strength and balance. In order to address the multifactorial and heterogeneous disease, this article would assist in bridging the gap between a widely recognized Ayurvedic approach and contemporary ideas and genetic recognition. Yoga therapy's physical poses (*Asanas*), breathing exercises (*Pranayama*), and Om meditation (*Om Dhyana*) may also aid in lowering fat deposits, improving insulin sensitivity, and rebalancing hormones to alleviate PCOS symptoms. As a type of

holistic mind-body therapy, yoga helps PCOS patients with their anxiety symptoms.

## OUTCOMES

PCOS has significant potential for metabolic and cardiovascular risk, so proper diagnosis and treatment are crucial. Both Ayurveda and contemporary systems lack a complete understanding of the underlying pathophysiology of PCOS. Treatment is therefore frequently concentrated on specific symptoms rather than the syndrome itself. However, PCOS treatment advances along with our understanding of its pathophysiology. Treatment should be tailored to each patient, but it should also address metabolic effects and prevent further complications.

## REFERENCES

1. Norman RJ, Dewailly D, Legro RS, Hickey TE. Polycystic ovary syndrome. *Lancet.*, 2007; 370: 685–697. [PubMed] [Google Scholar]
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod.*, 2004; 19: 41–47. [PubMed] [Google Scholar]
3. Carmina E, Oberfield SE, Lobo RA. The diagnosis of polycystic ovary syndrome in adolescents. *Am J Obstet Gynecol.*, 2010; 203: 201.e1–201.e5. [PubMed] [Google Scholar]
4. Stein IF, Leventhal ML. Amenorrhea associated with bilateral polycystic ovaries. *Am J Obstet Gynecol* 1935; 29: 181–91.
5. Azziz R, Carmina E, Dewailly D. Task force on the phenotype of the polycystic ovary syndrome of the androgen excess and PCOS society. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: The complete task force report. *Fertil Steril* 2009; 91: 456–88.
6. Deswal R, Narwal V, Dang A, Pundir CS. The prevalence of polycystic ovary syndrome: a brief systematic review. *J Hum Reprod Sci.*, 2020; 13: 261–71.
7. Franks S. Controversy in clinical endocrinology: Diagnosis of polycystic ovarian syndrome: In defense of the Rotterdam criteria. *J Clin Endocrinol Metab.*, 2006; 91: 786–9.
8. 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. *J Clin Endocrinol Metab.*, 2006; 91: 4237–45.
9. Witchel, S. F., Oberfield, S. E., & Peña, A. S. Polycystic Ovary Syndrome: Pathophysiology, Presentation, and Treatment with Emphasis on Adolescent Girls. *Journal of the Endocrine Society*, 2019; 3(8): 1545–1573. <https://doi.org/10.1210/js.2019-00078>

10. Rosenfield, R. L., & Ehrmann, D. A. The Pathogenesis of Polycystic Ovary Syndrome (PCOS): The Hypothesis of PCOS as Functional Ovarian Hyperandrogenism Revisited. *Endocrine reviews*, 2016; 37(5): 467–520. <https://doi.org/10.1210/er.2015-1104>
11. El Hayek, S., Bitar, L., Hamdar, L. H., Mirza, F. G., & Daoud, G. Poly Cystic Ovarian Syndrome: An Updated Overview. *Frontiers in physiology*, 2016; 7: 124. <https://doi.org/10.3389/fphys.2016.00124>
12. Ritu Deswal, Vinay Narwal, Amita Dang, and Chandra S. Pundir. The Prevalence of Polycystic Ovary Syndrome, PMID: PMC7879843 PMID: 33627974
13. Nidhi R, Padmalatha V, Nagarathna R, Amritanshu R. Prevalence of polycystic ovarian syndrome in Indian adolescents. *J Pediatr Adolesc Gynecol*, 2011 Aug; 24(4): 223-7. DOI: 10.1016/j.jpag.2011.03.002. Epub 2011 May 19. PMID: 21600812.
14. Azziz R, Dumesic DA, Goodarzi MO. Polycystic ovary syndrome: An ancient disorder? *Fertil Steril.*, 2011; 95: 1544-8.
15. Panda PK, Rane R, Ravichandran R, Singh S, Panchal H. Genetics of PCOS: A systematic bioinformatics approach to unveil the proteins responsible for PCOS. *Genom Data*, 2016; 8: 52–60.
16. Franks S, McCarthy MI, Hardy K. Development of polycystic ovary syndrome: Involvement of genetic and environmental factors. *Int J Androl.*, 2006; 29: 278–85.
17. Ganie MA, Vasudevan V, Wani IA, Baba MS, Arif T, Rashid A. Epidemiology, pathogenesis, genetics & management of polycystic ovary syndrome in India. *Indian J Med Res.*, 2019 Oct; 150(4): 333-344. DOI: 10.4103/ijmr.IJMR\_1937\_17. PMID: 31823915; PMID: PMC6902362.
18. Susrutha, Susrutha Samhita, Nibandha Sangraha commentary of Sri Dalhanacharya edited by Vaidya Yadavaji Trikamaji Acharya, Published by Chaukhambha Krishnadas academy, Varanasi, Edition, Sutra Sthana, chapter-15; 2008. p. 70, 824.
19. Susrutha, Susrutha Samhita, Nibandha Sangraha commentary of Sri Dalhanacharya edited by Vaidya Yadavaji Trikamaji Acharya, Published by Chaukhambha Krishnadas academy, Varanasi, Edition, Sutra Sthana, chapter-15; 2008; 71, 825.
20. Sushruta Samhita Chikitsa- 38/100-101, by Kaviraj Ambikadatta Shastri, Reprint Kashi Sanskrit Granthmala 156, Chaukhambha Sanskrit Sansthan, Varanasi, Reprint edition, 2014.
21. Acharya Chakrapani, Prof. R.H. Singh, Commentary on Charak Samhita, edition, Chaukhambha Surbharati Prakashan Varanasi, Chapter 12. p. 736, Sidhi Sthana, Falamatrasidhi adhyaya, Shlok, 2017; 42.
22. Bhavprakash Nighantu (1500-1600 AD) with Hindi Translation by Chunekar K. C./Pandey, G. S., 7th Ed. Chaukhambha Orientalia, Varanasi, India, 1986.
23. Ashtanga Sangraha Sharir Sthana 1/14, edited by Dr. Shiv Prasad Sharma, Banaras Ayurved Series 19, Chaukhambha Sanskrit Series Office, Varanasi, Reprint edition, 2008.
24. Arezoo Moini Jazani, Kobra Hamdi, Mojgan Tansaz, Hossein Nazemiyeh, Homayoun Sadeghi Bazargani, Seyed Mohammad Bagher Fazljou, Ramin Nasimi Doost Azgomi. Herbal Medicine for Oligomenorrhea and Amenorrhea: A Systematic Review of Ancient and Conventional Medicine, *Bio Med Research International*, vol. 2018: Article ID 3052768; 2018. p. 22. <https://doi.org/10.1155/2018/3052768>
25. Kowsalya R. G et al. A Case Study on Artava Kshaya. *International Ayurvedic Medical Journal {online}* {cited May 2018} Available from: [http://www.iamj.in/posts/images/upload/1143\\_1146.pdf](http://www.iamj.in/posts/images/upload/1143_1146.pdf); 2018.
26. Kashyapa Samhita, Kalpa Sthana. Revati Kalpa Shloka-33, Kashi Sanskrit Granthmala 154, Chaukhambha Sanskrit Sansthan, Varanasi, Reprint edition, 2018; 290.
27. Kashyapa Samhita, Shatapushpa Shatavari Kalpa Adhyaya- 5-6, 14-15, Kashi Sanskrit Granthmala 154, Chaukhambha Sanskrit Sansthan, Varanasi, Reprint edition, 2018.
28. Sushruta Samhita of Maharshi Sushruta with Hindi commentary by Shastri Ambika Dutt, 14th Ed. Chaukhambha Sanskrit Pratishthan, Varanasi, India, 2003.
29. Agrawal, A. K., Yadav, C. R., & Meena, M. S. Physiological aspects of Agni. *Ayu.*, 2010; 31(3): 395–398. <https://doi.org/10.4103/0974-8520.77159>
30. Saini N, Pal PK, Byadgi PS. Critical Analysis of Etiological Factors of Ajirna (Indigestion). *Int J Complement Alt Med*, 2017; 5(1): 00141. DOI: 10.15406/ijcam.2017.05.00141
31. Pellatt L, Hanna L, Brincat M, Galea R, Brain H, Whitehead S, et al. Granulosa cell production of anti-Müllerian hormone is increased in polycystic ovaries. *J Clin Endocrinol Metab*, 2007; 92: 240–5.
32. Pusalkar M, Meherji P, Gokral J, Savardekar L, Chinnaraj S, Maitra A. Obesity, and polycystic ovary syndrome: Association with androgens, leptin, and its genotypes. *Gynecol Endocrinol.*, 2010; 26: 874–82.
33. Ambekar AS, Kelkar DS, Pinto SM, Sharma R, Hinduja I, Zaveri K, et al. Proteomics of follicular fluid from women with polycystic ovary syndrome suggests molecular defects in follicular development. *J Clin Endocrinol Metab.*, 2015; 100: 744–53.
34. Liu, J., & Zhang, D. *Sichuan da xue xue bao. Yi xue ban = Journal of Sichuan University. Medical science edition*, 2012; 43(2): 187–190.
35. Ganie, M. A., Vasudevan, V., Wani, I. A., Baba, M. S., Arif, T., & Rashid, A. Epidemiology, pathogenesis, genetics & management of polycystic ovary syndrome in India. *The Indian Journal of*

- Medical Research, 2019; 150(4): 333–344. [https://doi.org/10.4103/ijmr.IJMR\\_1937\\_17](https://doi.org/10.4103/ijmr.IJMR_1937_17)
36. Hayes MG, Urbanek M, Ehrmann DA, Armstrong LL, Lee JY, Sisk R, et al. Genome-wide association of polycystic ovary syndrome implicates alterations in gonadotropin secretion in European ancestry populations. *Nat Commun*, 2015; 6: 7502
  37. Ganie MA, Vasudevan V, Wani IA, Baba MS, Arif T, Rashid A. Epidemiology, pathogenesis, genetics & management of polycystic ovary syndrome in India. *Indian J Med Res.*, 2019 Oct; 150(4): 333–344. DOI: 10.4103/ijmr.IJMR\_1937\_17. PMID: 31823915; PMCID: PMC6902362.
  38. Franks S. Controversy in clinical endocrinology: Diagnosis of polycystic ovarian syndrome: In defense of the Rotterdam criteria. *J Clin Endocrinol Metab.*, 2006; 91: 786–9.
  39. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, 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.
  40. Khan, M. J., Ullah, A., & Basit, S. Genetic Basis of Polycystic Ovary Syndrome (PCOS): Current Perspectives. *The application of clinical genetics*, 2019; 12: 249–260. <https://doi.org/10.2147/TACG.S200341>
  41. Charaka Samhita, Shastri K, Chaturvedi G. Varanasi: Chaukhambha Bharti Academy. *Charak Chikitsa Sthana*, 2002; chapter 15, verse 3; 459. [Google Scholar]
  42. Agrawal A. K., Yadav C. R., & Meena, M. S. Physiological aspects of Agni. *Ayu*, 2010; 31(3): 395–398. <https://doi.org/10.4103/0974-8520.77159>
  43. Vagbhata, Ashtanga Hridaya with Sarvanga Sundara of Arunadatta and Ayurveda Rasayana of Hemadri; ed. Pt. Hari sadashiva Shastri Paradakara Nidana sthana1/14-16, Chaukhambha Sanskrit Sansthan; Varanasi, 2012; 444.
  44. Nicolopoulou-Stamati, P., Maipas S., Kotampasi C., Stamatis P., & Hens, L. Chemical Pesticides and Human Health: The Urgent Need for a New Concept in Agriculture. *Frontiers in public health*, 2016; 4: 148. <https://doi.org/10.3389/fpubh.2016.00148>  
Dennedy M. C., Smith D., O'Shea, D., & McKenna, T. J. Investigation of patients with atypical or severe hyperandrogenemia including androgen-secreting ovarian teratoma. *European journal of endocrinology* 2010; 162(2): 213–220. <https://doi.org/10.1530/EJE-09-0576>.