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Agnimandya-INDUCED *Artava Dusti* IN POLYCYSTIC OVARIAN SYNDROME: A KRIYA *Sarira*-BASED INTEGRATIVE REVIEW

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ABSTRACT

Background: *Polycystic ovarian syndrome (PCOS) is increasingly recognized as a systemic metabolic-endocrine disorder rather than merely a gynecological condition. It involves complex interactions between metabolic dysfunction, hormonal imbalance, and chronic inflammation. While modern medicine explains PCOS primarily through insulin resistance and hypothalamic–pituitary–ovarian (HPO) axis dysregulation, Ayurveda offers a more foundational understanding rooted in Agni and Dhatu Parinama* [1,2].

Objective: *To explore the role of Agnimandya in the pathogenesis of Artava Dusti in PCOS through a Kriya Śarira-based integrative review.* ***Methods***: *A narrative integrative review was conducted using classical Ayurvedic texts and contemporary biomedical literature. Concepts related to Agni, Dhatu Parinama, and Artava Utpatti were critically analyzed and correlated with modern pathophysiological mechanisms* [14–17].

Results: *Impairment of Jatharagni and Dhatvagni leads to incomplete metabolism, resulting in Ama formation and defective tissue nourishment. Among all tissues, Medo Dhatu becomes predominantly affected, leading to metabolic disturbances analogous to insulin resistance. This ultimately causes obstruction of Artavavaha Srotas and dysfunction of Apana Vāta, manifesting as Artava Dusti* [3,6].

Conclusion: *Thus, PCOS can be interpreted as a systemic disorder originating from Agnimandya, progressing through Medo Dusti, and culminating in reproductive dysfunction.*

KEYWORDS: *PCOS, Agnimandya, Artava Dusti, Kriya Śarira, Insulin resistance, Dhatu Parinama*

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is one of the most prevalent endocrine disorders affecting women of reproductive age, characterized by menstrual irregularities, anovulation, hyperandrogenism, and metabolic disturbances such as obesity. In addition to reproductive dysfunction, PCOS is associated with long-term complications including type 2 diabetes mellitus, cardiovascular disease, and metabolic syndrome [1,9].

Modern biomedical understanding attributes PCOS to interrelated mechanisms such as insulin resistance, hyperandrogenism, and dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis[2,4]. Insulin resistance plays a central role by promoting hyperinsulinemia, thereby enhancing ovarian androgen production and disrupting follicular maturation. However, these explanations remain largely reductionist and fail to provide a unified perspective of the disorder.

Ayurveda, in contrast, offers a holistic framework based on fundamental physiological principles. Although PCOS is not described as a distinct entity, its clinical features resemble *Artava Dusti*, *Nashtartava*, and *Kapha–Vata Pradhana Yonivyapada*. The central concept underlying these conditions is *Agni*, the governing principle of digestion and metabolism [14-17].

In *Kriya Śarira*, *Agni* regulates *Ahara Paka* and *Dhatu Parinama*. Impairment of *Agni* (*Agnimandya*) leads to the formation of *Ama*, which disrupts tissue metabolism and causes *Medo Dusti*, comparable to metabolic dysfunction. This ultimately affects *Artava Utpatti*, resulting in *Artava Dusti* [14-17].

Thus, PCOS can be understood as a systemic metabolic disorder originating from *Agnimandya*, providing a comprehensive integrative perspective for its pathogenesis.

REVIEW OF LITERATURE

Hormonal Imbalance in Polycystic Ovarian Syndrome (PCOS)

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Hormonal imbalance represents a fundamental and defining feature in the pathophysiology of polycystic ovarian syndrome (PCOS), involving complex dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis, insulin signaling pathways, and peripheral endocrine function. This endocrine disruption is responsible for both reproductive and metabolic manifestations of the disorder.

1. Hypothalamic–Pituitary–Ovarian (HPO) Axis Dysfunction

PCOS is characterized by altered pulsatile secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus, which leads to preferential secretion of luteinizing hormone (LH) over follicle-stimulating hormone (FSH) from the anterior pituitary. This results in an increased LH: FSH ratio (commonly >2:1), a hallmark endocrine abnormality in many PCOS patients.

Elevated LH stimulates ovarian theca cells to produce excess androgens (such as testosterone and androstenedione), while relatively low FSH levels impair granulosa cell function and aromatization of androgens to estrogens. Consequently, follicular maturation is arrested, leading to anovulation and the formation of multiple immature follicles, which are visualized as "polycystic ovaries" on ultrasonography.

2. Hyperandrogenism

Hyperandrogenism is a central endocrine abnormality in PCOS and may be of ovarian or, less commonly, adrenal origin. Increased androgen production leads to:

a) Hirsutism
b) Acne
c) Androgenic alopecia
d) Follicular arrest and anovulation

At the ovarian level, excess androgens disrupt normal folliculogenesis by inducing premature follicular atresia. Additionally, androgens alter intra-ovarian signaling pathways and inhibit dominant follicle selection.

3. Role of Insulin Resistance and Hyperinsulinemia

A majority of women with PCOS exhibit insulin resistance, independent of obesity. Compensatory hyperinsulinemia plays a crucial role in amplifying hormonal imbalance through multiple mechanisms:
Stimulation of ovarian theca cells → increased androgen production
Suppression of hepatic sex hormone-binding globulin (SHBG) → increased free testosterone levels
Synergistic action with LH → further enhancement of androgen synthesis
Insulin also directly affects the hypothalamus and pituitary, contributing to abnormal GnRH pulsatility and LH hypersecretion. Thus, insulin acts not only as a metabolic hormone but also as a key modulator of reproductive endocrinology in PCOS.

4. Estrogen Imbalance and Progesterone Deficiency

In PCOS, chronic anovulation leads to persistent low progesterone levels, as corpus luteum formation does not occur. Meanwhile, estrogen levels (particularly estrone) remain relatively elevated due to peripheral aromatization of androgens in adipose tissue.
This results in a state of "unopposed estrogen exposure", which contributes to:

a. Endometrial hyperplasia
b. Irregular menstrual cycles
c. Increased risk of endometrial carcinoma in long-standing cases

5. Role of Anti-Müllerian Hormone (AMH)

Recent studies have highlighted elevated Anti-Müllerian Hormone (AMH) levels in PCOS. AMH is secreted by granulosa cells of small follicles and reflects ovarian reserve. In PCOS:

a) AMH levels are significantly increased due to a higher number of small antral follicles
b) Elevated AMH inhibits follicular sensitivity to FSH
c) This further contributes to follicular arrest and anovulation

Additionally, emerging evidence suggests that AMH may influence GnRH neuron activity, thereby contributing to neuroendocrine dysregulation.

6. Adrenal Contribution

In some patients, adrenal glands contribute to androgen excess through increased secretion of dehydroepiandrosterone sulfate (DHEAS). This suggests that PCOS is not purely an ovarian disorder but involves broader endocrine dysregulation.

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7. Integrative Ayurvedic Correlation

From an Ayurvedic perspective, the hormonal imbalance observed in PCOS can be understood through the interplay of *Doṣas* and Dhatu metabolism:

a) *Pitta Doṣa* governs metabolic and hormonal transformations; its vitiation contributes to abnormal biochemical activity, comparable to hyperandrogenism
b) *Kapha Doṣa*, particularly *Medo Dhatu*, is associated with structural and metabolic accumulation, correlating with insulin resistance and hyperinsulinemia
c) *Vata Doṣa* (*Apana Vata*) is responsible for ovulation and menstruation; its obstruction (Avarana) by *Kapha* and *Meda* leads to anovulation

The root cause remains Agnimandya, leading to impaired *Dhatu Parinama* and formation of *Ama*, which disrupts the normal functioning of *Artava* Dhatu.

Thus, hormonal imbalance in PCOS is not an isolated endocrine disturbance but a manifestation of systemic metabolic derangement involving *Agni*, *Doṣas*, and *Dhatus*.

Hormonal Factor	Change in PCOS	Physiological Effect	Ayurvedic Correlation
LH	Increased	Excess androgen production	*Pitta + Vata Dushti*
FSH	Decreased/Normal	Impaired follicular maturation	*Artava* Kshaya
Androgens	Increased	Hirsutism, anovulation	Pitta Dushti
Insulin	Increased	Hyperandrogenism,metabolic dysfunction	Medo Dushti, Kapha Vriddhi
SHBG	Decreased	Increased free testosterone	Agnimandya
Estrogen (Estrone)	Increased	Endometrial proliferation	Pitta Vriddhi
Progesterone	Decreased	Anovulation, irregular cycles	Apana Vata Dushti
AMH	Increased	Follicular arrest	Artava Dushti

MATERIALS AND METHODS

This study was conducted as a narrative integrative review to examine the role of *Agnimandya* in the pathogenesis of *Artava Duṣṭi* in Polycystic Ovarian Syndrome (PCOS). Classical Ayurvedic texts, including *Charaka Saṃhita*, *Suśruta Saṃhita*, and *Aṣṭaṅga Hṛdaya*, were reviewed to extract relevant concepts about *Agni*, *Dhatu Parinama*, and *Artava Utpatti* [14-17].

A structured literature search was performed using electronic databases such as PubMed, Scopus, and Google Scholar. Keywords including "PCOS," "insulin resistance," "ovulatory dysfunction," "*Agnimandya*," and "*Artava Duṣṭi*" were used to identify relevant studies[1-13].

The extracted data were critically analyzed and thematically synthesized. Classical Ayurvedic concepts were systematically correlated with contemporary biomedical mechanisms to develop an integrative framework. Based on this synthesis, a conceptual *Samprapti* model was formulated to delineate the sequential progression from *Agnimandya* to *Artava Duṣṭi* in PCOS.

RESULTS

The present integrative analysis delineates a coherent pathophysiological framework for polycystic ovarian syndrome (PCOS) through the lens of *Kriya Śārīra*, emphasizing *Agnimandya* as the primary initiating factor[14-17] . The findings demonstrate a sequential and interdependent cascade involving impairment of *Agni*, derangement of *Dhatu Parinama*, vitiation of *Doṣas,* and dysfunction of *Srotas*, ultimately culminating in *Artava Duṣṭi*. This integrative model highlights PCOS as a systemic metabolic disorder with reproductive manifestations.

***Agni* Dysfunction as the Primary Pathological Event**

The analysis of classical *Ayurvedic* principles establishes *Agnimandya* as the fundamental disturbance underlying the disease process [14]. Impairment of *Jatharagni* leads to incomplete digestion (*Apakwa Ahara Paka*), resulting in the formation of *Ama*, a metabolically toxic intermediate[14]. This Ama acts as a systemic pathological entity, interfering with nutrient assimilation and cellular metabolism.

At a deeper level, dysfunction of *Dhatvagni* disrupts the process of *Dhatu Parinama*, leading to both qualitative (*Guṇataḥ*) and quantitative (*Parimaṇataḥ*) abnormalities in tissue formation [14-17]. Among the seven *Dhatus*, Medo *Dhatu* exhibits predominant

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involvement, characterized by abnormal accumulation and altered metabolic activity. This observation aligns with the metabolic phenotype of PCOS, wherein adipose tissue dysfunction contributes to insulin resistance and endocrine imbalance [3,8] .

Derangement of *Dhatu Parinama* and *Artava Utpatti*
Ahara → *Jatharagni* → *Rasa* → *Rakta* → *Artava* [14]
Disruption at any stage results in defective formation of *Artava* (*Artava Vaigunya*), clinically manifesting as menstrual irregularities, anovulation, and infertility [2,4]

Medo *Duṣṭi* and *Srotorodha*
Impaired *Medo Dhatvagni* results in excessive accumulation of *Meda* (*Meda Vriddhi*), leading to *Srotorodha*, particularly affecting *Artavavaha Srotas* [14-17]. This can be correlated with ovarian dysfunction and follicular arrest [11].

Vata Avarana
Obstruction caused by *Kapha* and *Meda* leads to *Avarana* of *Apana Vata*, resulting in ovulatory dysfunction and menstrual irregularities [14-17].

Samprapti Flow
Agnimandya → *Ama* → *Rasa Duṣṭi* → *Medo Duṣṭi* → *Srotorodha* → *Vata Avarana* → *Artava Duṣṭi*

Integrative Correlation

***Ayurvedic* Concept**	**Modern Correlation**
Agnimandya	Metabolic dysfunction
Ama	Chronic inflammation [6]
Medo *Duṣṭi*	Insulin resistance [3,8]
Srotorodha	Ovarian dysfunction [11]
Vata Avarana	HPO axis dysregulation [2,4]

DISCUSSION

The present integrative review attempts to reinterpret the pathogenesis of polycystic ovarian syndrome (PCOS) through the lens of *Kriya Śarira*, with particular emphasis on *Agnimandya* as the initiating pathological event[14-17]. Unlike the compartmentalized understanding in contemporary biomedicine, *Ayurveda* offers a systemic and hierarchical model wherein digestion, metabolism, and reproduction are interlinked through the principle of *Agni* [14-17]. This review highlights that PCOS is not merely a disorder of the ovaries but a systemic metabolic derangement that originates at the level of impaired transformation.

At the outset, *Jatharagni* plays a pivotal role in determining the qualitative nature of *Ahara Rasa*, which serves as the substrate for all subsequent tissue formation [14]. Impairment of *Jatharagni* results in incomplete digestion and the formation of *Ama*, a metabolically toxic intermediate that disrupts physiological homeostasis [14]. In the context of PCOS, this concept can be correlated with altered metabolic processing, particularly postprandial dysmetabolism and impaired glucose handling. The persistence of such metabolic inefficiency contributes to systemic inflammation, a hallmark feature of PCOS pathophysiology [6].

Moving beyond primary digestion, the concept of *Dhatvagni* provides a deeper insight into tissue-level metabolism. Each *Dhatu* undergoes a process of transformation governed by its specific *Agni*, ensuring both structural integrity and functional competence [14-17]. In PCOS, the dysfunction of *Medo Dhatvagni* assumes particular importance. Impaired lipid metabolism leads to excessive accumulation of Meda Dhatu, not merely a quantitative increase but also a qualitative derangement. This abnormal *Meda* can be understood as metabolically active adipose tissue that contributes to insulin resistance, hyperinsulinemia, and subsequent endocrine disturbances [3,8]. Thus, the *Ayurvedic* concept of *Medo Duṣṭi* shows a remarkable parallel with the modern understanding of adipose tissue dysfunction and metabolic syndrome.

One of the most significant contributions of this integrative model lies in explaining the mechanism of *Srotorodha*. The accumulation of *Ama* and vitiated *Meda* leads to obstruction of microchannels, particularly the *Artavavaha Srotas*[14-17]. This obstruction not only affects the structural integrity of ovarian tissue but also interferes with its functional capacity. From a biomedical perspective, this may be correlated with ovarian stromal hypertrophy, follicular arrest, and altered intra-ovarian environment seen in PCOS [11]. The concept of *Srotorodha* thus provides a unifying explanation for both structural and functional abnormalities.

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Another crucial aspect is the role of *Vata Doṣa*, particularly *Apana Vata*, which governs reproductive functions including ovulation and menstruation[14-17] . The phenomenon of *Avarana*, wherein *Kapha* and *Meda* obstruct the normal functioning of *Vata*, offers a mechanistic explanation for ovulatory dysfunction. In modern terms, this can be likened to the disruption of the hypothalamic–pituitary–ovarian (HPO) axis, where altered neuroendocrine signaling leads to irregular gonadotropin secretion and anovulation [2,4]. The Ayurvedic explanation of *Vata Avarana* thus captures both neural and endocrine dimensions of PCOS pathophysiology.

Furthermore, the involvement of *Pitta Doṣa* cannot be overlooked, as it governs metabolic and hormonal transformations. Although not the primary *Doṣa* in PCOS, its imbalance contributes to hyperandrogenism and associated clinical features such as acne and hirsutism [4]. This reflects the interplay of all three *Doṣas*, establishing PCOS as a *Tridoṣaja Vyadhi* with predominance of *Kapha* and *Vata*.

An important strength of the Ayurvedic framework is its ability to integrate seemingly disparate pathological processes into a coherent model. For instance, the concept of *Ama* can be correlated with chronic low-grade inflammation, which is increasingly recognized as a central feature in PCOS [6]. Elevated inflammatory markers, oxidative stress, and cytokine imbalance contribute to insulin resistance and ovarian dysfunction. Thus, *Ama* serves as a functional equivalent of inflammatory mediators, bridging classical concepts with modern biochemical understanding.

Despite these conceptual parallels, certain limitations must be acknowledged. The primary challenge lies in the lack of objective, quantifiable measures for assessing *Agni* and *Ama*. While clinical assessment based on symptoms is well described in Ayurveda, it lacks standardization and reproducibility in research settings [23,24]. This poses a barrier to integrating *Ayurvedic* concepts into evidence-based frameworks. Additionally, the absence of well-designed interdisciplinary studies limits the validation of these correlations.

The present review also identifies a significant research gap in the translation of *Ayurvedic* principles into measurable parameters. There is a need to develop validated assessment tools for *Agni* status, possibly through composite indices incorporating digestive, metabolic, and biochemical markers. Similarly, correlating *Medo Duṣṭi* with parameters such as body mass index, lipid profile, and insulin resistance indices may provide objective support to classical concepts [3,8].

From a clinical perspective, this integrative understanding has important implications. It shifts the focus from symptomatic management of PCOS to addressing the root cause, namely *Agnimandya*. Therapeutic strategies aimed at improving *Agni*, reducing *Ama*, and correcting *Medo Duṣṭi* may offer a more holistic and sustainable approach. This aligns with current trends in integrative medicine, which emphasize lifestyle modification, metabolic correction, and individualized treatment [22].

In conclusion, the discussion underscores that PCOS is best understood as a systemic metabolic disorder rooted in impaired transformation rather than an isolated reproductive pathology. The *Kriya Śarira*-based model provides a comprehensive framework that integrates digestion, metabolism, and reproduction into a unified continuum. By bridging classical *Ayurvedic* concepts with modern biomedical insights, this approach not only enhances conceptual clarity but also opens new avenues for research and clinical application.

CONCLUSION

The present integrative review establishes that polycystic ovarian syndrome (PCOS) can be comprehensively understood as a systemic metabolic disorder rooted in *Agnimandya* [14–17]. From a Kriya *Śarira* perspective, impaired Agni initiates a cascade involving *Ama* formation, defective *Dhatu Parinama*, and *Medo Duṣṭi*, leading to *Srotorodha* and subsequent *Vata Avarana*, ultimately resulting in *Artava Duṣṭi* [14–17].

This stepwise *Samprapti* suggests that reproductive abnormalities in PCOS are secondary to underlying metabolic dysfunction rather than isolated ovarian pathology. The observed parallels between *Ayurvedic* concepts and modern mechanisms—such as insulin resistance, chronic inflammation, and hypothalamic–pituitary–ovarian axis dysregulation—highlight a significant conceptual convergence [2–4,6].

This integrative framework emphasizes the importance of addressing the root cause by restoring *Agni* and correcting metabolic imbalance. Further research is required to develop objective assessment parameters and validate these correlations through interdisciplinary studies [22–24].

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REFERANCE

1. *Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, et al. Polycystic ovary syndrome. Nat Rev Dis Primers. 2016; 2:16057.*
2. *Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. international evidence-based guideline for PCOS. Hum Reprod. 2018;33(9):1602–18.*
3. *Dunaif A. Insulin resistance in women with PCOS. Endocr Rev. 1997;18(6):774–800.*
4. *Escobar-Morreale HF. Polycystic ovary syndrome: definition, aetiology and treatment. Nat Rev Endocrinol. 2018;14(5):270–84.*
5. *Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Hum Reprod. 2004;19(1):41–7.*
6. *González F. Inflammation in PCOS: underlying mechanisms. Fertil Steril. 2012;97(1):38–45.*
7. *Barber TM, Franks S. Genetics of PCOS. Clin Endocrinol. 2013;79(4):466–73.*
8. *Diamanti-Kandarakis E, Dunaif A. Insulin resistance in PCOS. Trends Endocrinol Metab. 2012;23(8):428–35.*
9. *Legro RS. Obesity and PCOS. Endocrinol Metab Clin North Am. 2016;45(2):349–63.*
10. *Moran LJ, Pasquali R, Teede HJ, Hoeger KM, Norman RJ. Treatment of obesity in PCOS. Lancet Diabetes Endocrinol. 2015;3(9):760–71.*
11. *Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. PCOS: etiology and pathogenesis. Endocr Rev. 2011;32(5):779–821.*
12. *Lim SS, Davies MJ, Norman RJ, Moran LJ. Overweight, obesity in PCOS. Hum Reprod Update. 2012;18(6):618–37.*
13. *Palomba S, Santagni S, Falbo A, La Sala GB. Complications of PCOS. Hum Reprod Update. 2015;21(5):575–92.*
14. *Charaka Samhita, Sutrasthana.*
15. *Charaka Samhita, Chikitsasthana.*
16. *Sushruta Samhita, Sharirasthana.*
17. *Ashtanga Hridaya, Sutrasthana.*
18. *Sharma PV. Charaka Samhita (English translation).*
19. *Tripathi B. Ashtanga Hridaya commentary.*
20. *Lad V. Textbook of Ayurveda.*
21. *Dash B, Sharma RK. Charaka Samhita translation.*
22. *Patwardhan B. Bridging Ayurveda with modern science. J Ayurveda Integr Med. 2014.*
23. *Sharma H, Chandola HM. Ayurveda research methodology. AYU. 2011.*
24. *Tiwari S. Ayurveda physiology concepts.*