AYURVEDIC APPROACH TO UNDERSTAND THE ROLE OF HYPERANDROGENISM IN PCOS ALONG WITH TREATMENT MODALITY: A REVIEW

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ABSTRACT

Hyperandrogenism seen in polycystic ovarian syndrome is an excess of "male" hormones (androgens) such as testosterone and androstenedione. Increased androgens can be detected by elevated levels in the blood, or by clinical manifestations of polycystic ovarian syndrome that include Hirsutism and/or acne. Ayurvedic treatment has a holistic approach towards management of PCOS. Each patient is given a unique therapy predicated on her concrete health needs due to PCOS, rather than a versatile approach. Such personalized approach avails in re-balancing the hormones naturally. Ayurvedic therapy fixes on balancing tissue pabulum, rectifying the imbalance of dosha-bio-energies that govern the body and boosting metabolism.

KEYWORDS: Hyperandrogenism, PCOS, androstenedione, Sukradhaatu, sukraagni, DHEA, ama.

INTRODUCTION

Androgens are natural hormones in the female and form an integral part in the hormonal milieu of women in reproductive period of life. These are important for female in initiation of growth spurt during puberty, maintenance of bone mass before menopause and are
responsible for female libido. In ayurveda human body is composed of seven dhatus. As Sukra is considered seventh dhatu and essential for sustenance of life and nourishment of subsequent dhatu, it may be considered as sex hormones. Complete absence of any specific dhatu will make life impossible. However presence of it in stree sharir responsible for energy and complexion. “tasamapi bala varna shukra pushtim karoti hi” (Bha.pra. 3/88) Acharya chakrapani also accepting presence of sukra in women and clarified that the garbha is formed from essence part of pure sukra not from its by product.[1] “shukrattu nirmaltya prasadaj eva garbho bhavati na tu malajajanyaam kinchit”(cha.chi.15/16 chakrapani tika) It seems to be explanation of sex hormones especially androgens because if conversion of these androgen to oestrogen (Prasad bhaga) responsible for garbha utapati otherwise if mala rupa form then features of hyperandrogenism occur.

CONCEPT OF HYPERANDRODENISM: Androgens are c-19 steroids. At ovarian microenvironmental level the androgens act as substrates for ovarian estrogens. Hyperandrogenism is defined as biochemical or clinical manifestation of androgen excess. It is hallmark of PCOS.

Androgens produced in females from three compartments.
1. Ovary
2. Adrenal gland
3. Periphery and liver

DHEA (Dehydroepiandrostanedione) produced 50% by adrenal & 50% by ovary. Androstenedione 50% from adrenal, 25% from ovary, 25% by peripheral tissue. Testosterone form by peripheral conversion of androstenedione, 25% from adrenal, 25% from ovary. DHT (Dehydrotestesterone) produced by local conversion of testosterone to DHT by action of 5 alfa reductase enzyme at level of skin.

Conversion of androgens to female harmone is by Sukraagni. After the action of sukraagni male hormones convert or destruct in female harmone. This Sukraagni is considered as pitta which is responsible for transformation reflected in the form of hormones & enzymes at different stage of ovarian & menstrual cycle.

When we look towards modern physiology of androgen conversion to female hormone, there is enzyme aromatase (may be considered as Sukraagni) which convert androgen (form by
thecal layer of ovary) to oestrogen in granulose cells of ovary. If, this enzyme activity is reduced androgens are not convert into oestrogen & make androgenic micro environment which result in follicular atresia, arrest of oocyte growth & maturation & end result is anovulation i.e Abeejatvam.

Also, these androgens are destruct in liver or become inactive when bound to SHBG (Sex hormone binding globulin) or albumin. Only 1% remains active. If these androgen production is more or level of SHBG reduced result in hyperandrogenism. Main place of dhatwaagni is liver to work; due to dhatwaagnimandya free androgens are circulating in body.

In PCOS there is hyperinsulenemia along with raised blood glucose levels as insulin is not utilised may be due to ama which is the result of mandagni. Ama spread throughout the body propelled by vitiated vata along the rasavahasrotas result in dhatwaagni mandya and in the end sukraagni mandya. It results in improper conversion of androgen to female hormone & in PCOS this increased level of insulin inhibit formation of SHBG from liver, thus increasing free androgen ultimately creates clinical manifestation of PCOS.

**CLINICAL MANIFESTATION OF HYPERANDROGENISM**

1. **Seborrhagia & acne**

   Acne is seen in 1/3rd of patient with pcos.[2] Sebaceous glands are present in the skin of entire body & highest concentration on face. Acini of gland produce sebaceous material. The secretions reach the surface of the skin via the duct which opens at pore. The glandular cells have testosterone and DHT receptors at the basement membrane. Due to hyperandrogenism secretion of sebum increases and glandular hyperthropy of the acinar cells occur and produce clinical symptom of seborrhea. Propionibacterium acne, a commensal thrives on this sebum and produces a chronic inflammatory reaction which leads to subsequent hyperkeratosis and increased viscosity cause blockage of pores and lead to acne formation.[3]

   According to astangsangrahkar when medomarg (pores of skin) are closed meda (sebum) collected inside the pores & when commensal thrives on this sebum produces chronic inflammatory reaction & blockage of pore lead to acne formation along with puya due to pitta & kapha result in pustule formation[4] which is sometime painfull. Acharya susruta stated that due to vitiation of bhrajak pitta & vyan vayu along with kapha and rakta acne formation occur (su.ni.13/287).[5]
2. Hirsutism
Hirsutism is development of male type of hair distribution in female due to conversion of villous hair to terminal hair due to excess androgen. About 1 in 10 women in reproductive period have hirsutism & PCOS is common cause for it. When pitta aggravation at the level of bhrajaka pitta which is located in skin responsible for hirsutism or when 5 alfa reductase activity increase in skin which convert testosterone to more active metabolite dehydrotestosterone hirsutism occur. Hirsutism is changes in pigmentation, length, diameter and rate of growth of hairs rather than by increase in the number of hairs per unit area. Action of DHT on the dermal papilla of the hair follicle is primarily responsible for initiating the changes, which is reversible to certain extent. The Ferriman and Gallwey scoring system is used to quantify hairgrowth and to monitor response to therapy. It is necessary to differentiate hirsutism from hypertrichosis, which is not dependent on hyperandrogenism. Clinical features of pushphagni jatharani as described by acharya kasaypa give relevance of hyperandrogenism in which women menstruates in time without ovulation and has corpulent & hairy cheeks.[6]

3. Blackening of skin
Bhrajaka pitta is responsible for varna of skin. Pitta ksaya vridhi result in hypo or hyperpigmentation of skin. When vayu & kapha vitiated along with pitta result in shayava aruna varna & sukla varna of skin. Also, pitta vitiated rakta dhatu result in vaikrat varna. In pcos patient skin become thickened, pigmented, velvety, most often found in vulva and may be present on the axilla, on the nape of the neck, below the breast,and on the inner thigh. It is acanthosis nigricans and considered as marker of insulin resistance in hirsute women.[7]

4. Androgenic alopecia
Vitiated vata along with bhrajak pitta causing hairfall of the scalp after this kapha along with rakta causes closure of the papilla of hair follicle so that no new growth occur & lead to khalitya (alopecia).[8] (Su.Ni.13/285). According to modern pathology it is partly genetic and partly androgen dependent. It is interesting that same hormones which cause excess hair growth elsewhere cause alopecia in the scalp.

Female androgenic alopecia starts at the crown and is initiated as widening of the hair parting in the middle, seen in approximately 8% of women with pcos.
LATE SEQUEALE OF HYPERANDROGENISM\[^{9}\]

When hyperandrogenism is in chronic form it creates central obesity, voice change, increased muscle mass, clitoromegaly (virilism). Sedulously assiduous hyperandrogenism even mild having positive correlation with hyperlipidemia, atherosclerosis, non insulin dependent diabetes mellitus, high B.P.

DIFFERENTIAL DIAGNOSIS

1. Congenital adrenal hyperplasia
2. Cushing syndrome
3. Masculinizing tumor of ovary
4. Androgen secreting adrenal tumor
5. Hyperprolectinemia
6. Acromegaly
7. Iatrogenic virilisation

INVESTIGATIONS\[^{10}\]

1. Total testosterone (N-20-80 mg/dl)
   Values are normal or mildly elevated in hirsutism due to PCOS. (<150 mg/dl))
2. DHEAS (N- 100-350 mg/dl)
   It is adrenal androgen level normal or moderately elevated in hirsutism due to pcos & excludes androgen secreting tumor.
3. SHBG (N 18-114nmol/dl)
   Levels decrease in response to high level of androgen (DHEA, DHEAS, Testosterone, androstenedione)
4. 3 alfa Androstenediol G
   3 alfa diol & androsterone G as marker of 5 alfa reductase activity in the skin are increases in hirsutism.
5. LH: FSH ratio > _2:1
6. GTT in obese female for marker of insulin resistance.
   Normal <140
   Impaired glucose tolerance 140-200
   NIDDM > 200
7. Serum prolactin & TFT
   To exclude hyperprolactenemia & hyperthyroidism
8. Serum cortisol
Free urinary cortisol or overnight dexamethasone suppression test for exclusion of cushing syndrome.
9. 17-hydroxyprogesterone level
For exclusion of congenital adrenal hyperplasia
10. USG for identifying pcos
Follicle distribution, stromal echogenicity, volume are important diagnostic criteria.

TREATMENT MODALITIES FOR HYPERANDROGENISM

- NIDANPARIVARJAN (ABSTINENCE OF CAUSATIVE FACTORS)
Firstly to treat the cause. PCOS is considered as a polygenic trait that might result from the interaction of susceptible and protective genomic variants and environmental factors, during either prenatal or postnatal life. These polygenic or prenatal cause considered as Beejadoshaj (Genetic) and postnatal cause can be considered under Apathyanimitaj (Acquired due to improper life style)

Beejadosha (prenatal cause) is prevented by Shodhan therapy, before conception, & after Conception Garbhiniparicharya must be followed by female. Apathyanimitaj (post nattally acquired due to improper life style) is prevented by adaptation of proper Dinacharya, Ritucharya,& Rajaswala paricharya.

- SAMSODHAN CHIKITSHA

Vaman karma
Vamana is a Cleansing procedure intended mainly for the expulsion of vitiated ‘Kapha’. This balances hormonal system. Vaman acts on Thyroid gland. It also stimulates pancreas to secrete insulin in normal level, so P.C.O.D. & hyperandrogenism decreases accordingly.

Virechana karma
Hyperandrogenism is due to vitiated Pitta & virechana is prime therapy of vitiated ‘Pitta’. The process of cleansing is carried out in the small intestine & other Pitta zones. Here drugs that stimulate bowel movement are increased for the expulsion of doshas through rectum. It acts on hormones system like ‘Vaman Karma’.
Basti (Enema)
Basti is the choice of therapy in this context due to its utility in conditions of vitiated Vata. Excess Androgen secretion is from Ovaries and adrenal gland and these could be considered under Pakvashaya, which is seat of Vata Dosha and specially Apana Vata. Especially Anuvasan (enema with medicated oil), Niruha (enema with medicated decoction) and Uttarbasti (enema in the genital tract) which are more beneficial in this condition.

Shirovirechana
Medicines administered through the nose, goes into the Mastishka and expels out the vitiated doshas. Drug though nasal route (i.e. gateway of head) reaches the Shringataka Marma spreads thought the Siras of nose, ear, eye and tongue reaches in Shira (Head, Brain) scratched the morbid doshas promotes the normal physiological function & regulate H-P-O-U axis.

Raktamokshana
According to Ashraya-ashrayi Sidhanta, Pitta is ashrayi of Rakta, so vitiation of Pitta leads to vitiation of Rakta and Rakta is transport medium of nutrition as well as hormone, enzymes etc. Acne, Acanthosisnigricans which are symptoms of Hyperandrogenism is nothing but due to Pitta- Rakta vitiation also menstrual irregularities are also due to these dosha. Thats why Virechana along with Raktamokshana (expulsion of blood) may play principal role for normalisation of hormonal equilibrium

- SAMSHAMAN CHIKITSA
Amapachan & agnideepan chikitsha
As the main cause of disease is agnimandhya at jataragni and dhatwagni level so regularize pitta by using amapachak drugs.
Chitrakadi vati
Panchkola Churanm
Panchasakara Churnam
Trikatu+ Nagkesar
Apivattikar churna

SPECIFIC MANAGEMENT
1. To treat hyperinsulinemia
Vijaysar churna (Pterocarpus marsupium)
Gurmar (Gymnea sylvestris)
Nimba
Triphala
Bilwa churna
Alovera
Prameha prahar churna

2. To normalize level of estrogen
Shatpuspha churna
Shatavari
Black tila (black sesame)
Yastimadhu

3. To treat hyperpigmentation of skin
a. Varnya mahakasaya drugs (cha.su 4)
b. Lodhradi gana drugs (Su.Su.38/14)
c. Eladi gana drugs (Su.Su. 38/25)
Drugs of these gana use in the form of powder, kwatha kalpna or locally.

4. To treat hirsutism
a. Lomshatan yoga formed by mixing shankha, hartzal with kadali swaras. (chakradutta 62/56)
b. Shallaki thorn, hartzal, sudhachurna mix with kadaliswaras useful for removing hair. (Sidha bhashajyamanimala stri roga chikitsha)
c. Mixture of shankha bhasma, hartzal with kanji. (su.chi.1/105)
d. Shankh churna (2part), hartzal(1part), manashila(1/2part), sajjikshara(1part) used locally.\(^12\) (sha. uttarkhanda 11/35)
e. Hartal(2part), shankh churna(6part), palash kshara(2part) with kadali swaras or madarpatra swaras for local application. (sha. uttarkhanda 11/35)
f. Aragwadh taila\(^13\) (chakradutta 62/58)
g. Karpuradi taila (chakradutta 62/58)
h. Kshara taila (chakradutta 62/58)
CONCLUSION
Hyprandrogenism is one of the criteria of PCOS & an important clinical & psychological problem. Approximate 30% patients have acne, 70% hirsutism, 8% androgenic alopecia. It is not directly mentioned in Ayurvedic classics but from the pathogenesis and clinical manifestations of the hyperandrogenism, it is evident that it is Tridoshaja vyadhi. Treatment modalities aim to correct ama dosha (insulin level), regularize the work of dhatwagni & ultimately regulate tridhosha.

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