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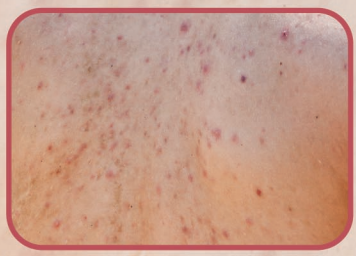




Handbook on
ACNE

Swetalina Pradhan

Foreword
Sandipan Dhar

JAYPEE

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CHAPTER 7

Hormonal Acne

Bhabani STP Singh, Sonal Jain

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INTRODUCTION

Acne affects about 80% of individuals between the age 11 and 30 years. Majority of them remit by 40 years of age, while rest follow an unsure course. However, cases of adult acne are on the rise and women are mostly affected.[1] Adult acne is defined as acne that develops (late-onset acne) or continues (persistent acne) after 25 years of age affecting about 33% of individuals between the age 15 and 44 years. There are two clinical types: (1) Persistent acne and (2) late-onset acne. Both are more common in women. Acne is now regarded as a chronic phenomenon than a temporary adolescent disease.[2]

The principal hallmarks of acne include follicular hyperproliferation and plugging, extensive formation of sebum, activity of *Propionibacterium acnes* (*P. acnes)*, and inflammation. The pathogenesis of adult female acne (AFA) is related to genetic and hormonal factor associated with chronic course, requiring long-term therapy, in few patients for years. Stress, increased use of cosmetics, and hot and humid climate play significant role in the increasing cases of AFA.

There is sufficient evidence on sex hormones and androgen excess playing major role in the pathogenesis of acne. Sebaceous gland possesses all enzymes needed for formation and metabolism of androgen from cholesterol to dihydrotestosterone (DHT). Androgen receptor (AR), estrogen receptor (ER) especially ERα, and progesterone receptors (PR) especially progesterone receptor B (PR-B) are also present in the sebaceous gland.

PATHOPHYSIOLOGY[3-5]

The pathogenesis of acne involves an interaction between the following factors such as:

- Genetic predisposition
- Androgenic hormones excess causing elevated secretion from sebaceous glands
- Periglandular dermal inflammation

The causes of AFA are yet to be fully understood. Several other factors have been indicated as inducing factors, such as UV radiation, stress, obesity, food, smoking, sleep disorders, cosmetics, medications, excessive skin washing, possible resistant *P. acnes,* and endocrine deficiency diseases. Epidermal barrier function defect has also been identified as a cause. Barrier dysfunction and increased transepidermal water loss may cause inception of inflammatory process that is the major change in the commencement of acne.

Genetics

Genetic predisposition is an important susceptible factor, which affects number, size, and activity of sebaceous glands, as well as control the process of follicular hyperkeratinization and innate immunity. Many adult acne women have first-degree relatives with acne during adulthood.

Hormones

Testosterone, dehydroepiandrosterone sulfate (DHEAS) and DHT stimulate sebaceous gland growth and production of sebum. Estrogens on the other hand inhibit the secretion of androgens, cause gene inhibition that decreases growth of sebaceous gland. Hence, sebaceous gland activity depends on the estrogen/androgen ratio. AFA and hormones have following co-relation:

- Sebaceous gland hypersensitivity to androgens
- *Increased peripheral hormone conversion:* Sebocytes and keratinocytes have enzymes for local production of testosterone and DHT. The hyperactive enzyme activity such as 5-alpha reductase, 3-beta-hydroxysteroid dehydrogenase and 17-hydroxysteroid dehydrogenase, associated with elevated pre-hormone peripheral conversion (SDHEA, androstenedione, and testosterone) into more potent androgens (testosterone and DHT). The potency of DHT is 5–10 times more than testosterone.
- An increase in androgens relative to estradiol during premenstrual period, premenopausal scenario, pregnancy, and during intake of progestin-only contraceptives leads to flare of acne.
- *Other hormones which regulate the sebum production:* During stressful conditions, neuropeptides and hormones such as melanocortins and corticotropin-releasing hormone (CRH) stimulate sebaceous glands. CRH increases 3β-hydroxy-steroid dehydrogenase mRNA, which converts dehydroepiandrosterone (DHEA) to testosterone. Also, neuropeptides, histamine, retinoids, vitamin D, and insulin-like growth factor 1 (IGF-1) can also regulate sebum production.
- Melanocortin is a byproduct of proopiomelanocortin. MC-1R and MC-5R are the two receptors expressed on sebocytes involved in regulation of the sebocyte differentiation and lipogenesis.

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- Insulin increases sebaceous gland growth and maturity. Insulin upregulates the GH receptors on sebocytes to perform this action. Insulin also inhibits hepatic production of SHBG (sex hormone binding globulin) and a positive feedback effect on adrenal and ovarian androgen production. High glycemic index foods result in insulin release and in turn cause excess androgen and sebum production.
- Adrenocorticotropic hormone (ACTH) stimulates sebum production. GH (growth hormone) stimulates sebocyte differentiation and conversion of testosterone to DHT. LH stimulates ovarian production of androgens. Hyperprolactinemia causes secretion of adrenal androgens leading to acne.
- Deficient vitamin D is associated with insulin resistance and the metabolic syndrome in cases of polycystic ovary syndrome (PCOS).

Immuno-inflammatory Processes

Propionibacterium acnes, TLR2, and TLR4 recognition cause initiation of an inflammatory reaction via nuclear path nuclear factor-kappa B (NF-κB), leading to the production of cytokines or interleukins and tumor necrosis factor alpha (TNF-α) that maintain the inflammatory cycle by recruiting neutrophils and macrophages. There is also release of antimicrobial peptides such as beta-defensins 1 and 2, cathelicidins and glanulysins. AP1 pathway releases metalloproteinases 1, 3, and 9 that cause degradation of extracellular dermal matrix and lead to scar formation.

Diet

High glycemic index and dairy foods lead to elevated insulin and insulin growth factors 1 levels. Gonads and sebaceous glands both carry receptors for these hormones leading to production of androgens, such as testosterone, and cause inhibition of testosterone conversion to estradiol. These food products stimulate mTORC1, leading to elevated protein and lipid formation, cell differentiation, sebaceous gland hyperplasia, elevated sebaceous lipogenesis, insulin resistance and high body mass index (BMI). Supplements for muscle mass gain contain branched-chain amino acids and peptides, derivatives of whey also cause acne flare.

Drugs

Certain medications such as benzodiazepines, lithium, cyclosporine, ramipril, isoniazid, iodides, bromides, vitamin B-type complexes, serotonin uptake inhibitors, epidermal growth receptor inhibitors, and progestin contraceptives can lead to acne worsening. First-generation progestins such as norgestrel and levonorgestrel carry androgenic properties similar to testosterone. Steroids lead to hyperkeratinization due to elevated expression of TLR2.

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Stress

Stress can cause elevated cortisol secondary to the formation of pro-inflammatory cytokines and CRH. Deprived sleep affects the hypothalamic–pituitary–adrenal (HPA) axis and elevated stress-related hormones, leading to aggravation of acne.

Acne has been associated with many endocrinopathies presenting with myriad of clinical features—hirsutism, alopecia, menstrual problems, ovulatory dysfunction, infertility, early puberty, metabolic syndrome, and virilization.

Various syndromes linked with hormonal problems and acne are listed below:

- Polycystic ovary syndrome
- Cushing's syndrome
- Congenital adrenal hyperplasia (CAH)
- Hyperandrogenism, insulin resistance, and acanthosis nigricans (HAIRAN) syndrome
- Seborrhea, acne, hirsutism and/or androgenetic alopecia (SAHA) syndrome
- Acromegaly Synovitis acne pustulosis hyperostosis osteitis (SAPHO) syndrome
- Pyogenic arthritis, pyoderma gangrenosum, acne conglobata syndrome (PAPA)
- Apert syndrome
- Acrocephalosyndactyly type 1

Polycystic ovary syndrome causes ovarian production of androgen to increase, due to 17α-hydroxylase dysregulation. The androgen excess causes ovarian dysfunction as well as acne, hirsutism, and alopecia. Excess androgens are converted to estrogens (androstenedione to estrone and testosterone to estradiol) in the peripheral tissues via aromatase enzyme, which leads to luteinizing hormone (LH) secretion by pituitary gland. Hyperinsulinemia which causes ovarian formation of androgens followed by LH releases the pituitary. This causes abnormal gonadotropins secretion leading to elevated LH: FSH ratio of ≥2. Elevated LH is pathognomonic but does not confirm the diagnosis of PCOS.

The HAIR-AN syndrome is more severe subtype of PCOS. HAIRAN is of two types—type A or type B. Type A shows mutation of insulin receptor and features of insulin resistance. Type B is autoimmune in nature associated with formation of antibodies to the insulin receptor. HAIRAN syndrome is characterized by increased insulin, testosterone, and androstenedione.

Clinical features of hyperandrogenism in adult females are:

- Acne/seborrhea/oily skin
- Hirsutism
- Menstrual irregularity
- Acanthosis nigricans
- Androgenetic alopecia

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- Cushingoid features
- Clitoromegaly, with elevated libido, deepened voice
- Association with diabetes mellitus
- Obesity

Hormonal acne presents with jawline and chin acne, adult acne, premenstrual and cystic acne with/without menstrual irregularities and hirsutism **(Figs. 1 and 2)**. Androgens fluctuations can cause cyclic acne flare, including premenstrual aggravation. Excess androgens are indicated by cysts, nodules, sudden and widely dispersed lesions.



FIG. 1: Hormonal acne—multiple erythematous papules and nodules over cheeks and mandible along with increased facial hair.



FIG. 2: Hormonal acne—multiples comedones and nodules on cheek, mandible, and chin in a middle-aged female.

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LABORATORY INVESTIGATIONS

Hormonal evaluation is not indicated in case of short bouts of acne or those who respond to standard therapy. Laboratory evaluation is, however, needed in more resistant cases and in case of failure of conventional therapies.[6]

Termination of hormonal therapy such as oral contraceptives at least 1 month prior is a prerequisite for endocrinal evaluation.

- *Testosterone (free and total):* Levels of <200 ng/dL suggest a benign ovarian or adrenal cause, while higher levels point toward ovarian or adrenal neoplasia.[7]
- *Androstenedione:* The secretion is equal from ovaries and adrenals following a circadian rhythm, hence early morning samples are evaluated.
- *Dehydroepiandrosterone:* DHEA >8,000 ng/dL and DHEAS, adrenal tumors must be ruled out, and levels of (4,000–8,000 ng/dL) indicate benign adrenal causes such as hyperplasia.[7]
- *Sex hormone binding globulin:* Low SHBG levels cause free testosterone to rise.
- *Prolactin:* High prolactin should lead to hypothalamic or pituitary evaluation.[6]
- *17-Hydroxyprogesterone:* Elevation (>200 ng/dL) is seen in CAH or non-classic CAH as a result of deficient or absent 21α-hydroxylase.[7]
- LH:FSH ratio of >2 is suggestive of PCOS.
- *Fasting and postprandial insulin:* It should be evaluated in obese or overweight individuals.
- Elevated cortisol level is suggestive of adrenal neoplasia.
- In suspected PCOS, transvaginal ultrasonography is indicated (presence of 12 or more follicles with 2 and 9 mm of diameter or increase in ovarian volume >10 cm^3).
- *Adrenocorticotropic hormone stimulation or dexamethasone suppression test:* Ovarian androgens will not respond to these tests while adrenal androgens will elevate following the ACTH stimulation and reduce with dexamethasone suppression test.[7]

The samples to perform these tests should be collected during the follicular phase, preferably between the first and fifth days of the menstruation with collection of early morning sample, between 8 and 10 AM.

MANAGEMENT

Hormonal therapy is the management of choice for hormonal acne. The various indications of hormonal therapy are:

- Severe premenstrual flare
- Combined oral contraceptives (COCs) are indicated.
- Acne resistant to regular therapy
- Polycystic ovary syndrome
- Acne tarda
- Ovarian or adrenal hyperandrogenism

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BOX 1 Drugs used for the management of hormonal acne.

- *Androgen receptor blockers*:
  - Spironolactone
  - Cyproterone acetate
  - Drospirenone
  - Flutamide
- *Drugs suppressing adrenal androgen production*:
  - Low-dose corticosteroids
- *Drugs suppressing ovarian androgen production*:
  - Gonadotropin-release hormone (GnRH) agonists
  - Combined oral contraceptives
  - Progestins
- *Prolactin suppressor (dopamine agonists)*:
  - Bromocriptine
  - Cabergoline
  - Quinazoline
- *Insulin-sensitizing agents*:
  - Metformin
- *Recent drugs*:
  - Isotretinoin
  - Vitamin D

However, hormonal therapy is not implemented as the only therapy. The aim of hormonal therapy in acne are (1) ovarian and adrenals androgen suppression and (2) sebaceous glands AR inhibition. **Box 1** enumerates various medications with anti-androgenic potential.

Androgen Receptor Blockers

Spironolactone[8]

It is an aldosterone antagonist (potassium-sparing diuretic), antiandrogen, and weak progestin. Its mechanisms of action are given as following:

- Androgen receptors are part of steroid/nuclear receptor superfamily. It competitively causes nuclear blockage of ARs leading to suppression of binding and action of testosterone and DHT.
- Inhibition of type 2 17β-hydroxysteroid dehydrogenase (17β-HSD) enzyme causing suppression of testosterone
- 5α-reductase inhibitor
- Elevation of SHBG leading to decreased circulating levels of free testosterone
- Increases hepatic clearance of testosterone by elevation of hydroxylase and reduction of 5α-reductase activity.

Its dose varies from 50 to 200 mg/day. Male use of spironolactone is not advised due to decreased libido, impotence, and gynecomastia. Pregnancy is an absolute contraindication because it may cause feminization of male fetus and increased chances of hypospadias. Therefore, it is generally prescribed in conjunction with oral contraceptives. Other adverse effects include breast

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tenderness, menstrual irregularities, and hyperkalemia, which should be checked periodically and spironolactone should be stopped if hyperkalemia occurs. Dietary restriction of excess bananas and soda is advised.

Cyproterone Acetate[9]

It is an antiandrogen and progestin. It can be prescribed as monotherapy or can be combined with OCPs [ethinyl estradiol 35 μg/CPA (cyproterone acetate) 2 mg]. Administration is from day 1 to 10 of the menstruation when prescribed singly. It decreases sebum production by preventing androstenedione formation from DHEA. It is used in the management of recalcitrant acne, androgenetic alopecia, and hirsutism. Hepatic toxicity, feminization of male fetus, breast tenderness, and gastric symptoms such as nausea and vomiting are the major side effects.

Topical CPA is a novel preparation (solid lipid nano particles) with increased follicular penetration and is used in both males and females.

Drospirenone

It is a progestin and derivative of 17α-spironolactone. It possesses anti-androgen and anti-mineralocorticoid activity. It is combined with estrogen in lower doses (drospirenone 3 mg/ethinyl estradiol 20, 30 μg), and used for acne vulgaris and hirsutism. Adverse effects are thromboembolic phenomenon, and high potassium levels. Hepatorenal dysfunction and adrenal insufficiency are the major contraindications. The coadministrations with following medicines are contraindicated—angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor agonists, potassium sparing diuretics, potassium supplementation, heparin, aldosterone antagonists, and nonsteroidal anti-inflammatory drugs (NSAIDs).

Flutamide

It is useful in the management of prostate carcinoma. In dermatology, it is used for the management of acne and hirsutism at 250–300 mg/day. Hepatotoxicity is the major side effect limiting its use. Pseudohermaphrodite condition and feminization of male fetus as well as fetal hepatitis are other side effects.

Antisense Ribonucleic Acid Oligonucleotides[10]

Coadministration with penetration enhancers is a new therapy which acts by inhibition of AR expression.

DRUGS SUPPRESSING ADRENAL ANDROGEN PRODUCTION

Low-dose Corticosteroids

Prednisolone 2.5–5 mg/day at night or low dose dexamethasone is used in late onset congenital adrenal hyperplasia (21 hydroxylase or 11 hydroxylase deficiency). Accumulation and shunting of corticosteroid precursors into

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the pathway for androgen biosynthesis form the mechanism of action. Monitoring of serum DHEAS is done for evaluation of effects of steroids. ACTH stimulation test is used to look for adrenal suppression every 2 months.

Ovarian Androgen Blockers

Combined Oral Contraceptives[11]

They are combinations of estrogens (ethinyl estradiol and rarely mestranol) and progestin (CPA, chlormadinone, drospirenone, and derivatives of 19 nortestosterone). Drospirenone is the only Food and Drug Administration (FDA)-approved progestin, which causes AR blockage and has antiandrogenic property. Estrogens possess sebo-suppressive properties. Progestins reduce the incidence of endometrial cancer which is elevated by estrogens. It is started from day 1 to 21 days on/7 days off fashion in the menstrual cycle and is continued at least for 5–6 cycles, when pregnancy test is negative.

Combined oral contraceptives have the following mechanism of action:

- They decrease gonadotropins (LH and FSH) secretion which leads to anovulation and thus decreases androgen production by ovaries.
- Androgen receptor blockage
- Elevate hepatic formation of SHBG, leading to low free testosterone levels
- Progestins lower 5α-reductase activity and prevent formation of potent androgens

Pregnancy, history of thromboembolic episode, hepatic disorder, and smokers ≥35 years, are absolute contraindications to use COCs while relative contraindications include lactation, high blood pressure, migraine, and existing cancers. COCs can worsen few conditions such as insulin resistance and are not useful in diabetes mellitus and clotting disorders.

Gonadotropin-release Hormone Agonists

Gonadotropin-release hormone (GnRH) agonists (buserelin, nafarelin, leuprolide, and triptorelin) are analogs of GnRH stimulate LH and FSH, blocks their cyclic release from pituitary leading to anovulation and causing both ovarian androgen and estrogen suppression. Available formulations are nasal sprays, injections, and subcutaneous implant. They are used in acne and hirsutism. Breastfeeding, vaginal bleeding, and pregnancy are the major contraindications.

Insulin-sensitizing Agents

Insulin resistance reduces insulin use by cells causing hyperinsulinemia which increases ovarian and adrenal androgen secretion and by reducing formation of SHBG. Metformin is useful for acne with PCOS, HAIR-AN syndrome, obesity, and in cases of biochemical hyperinsulinemia. The doses are 500–2,000 mg/day. Most side effects are dose dependent and include nausea and vomiting, its use should be limited to postprandial intake and with low dose of 250 mg.

Table 1 shows the mechanism of action, adverse effects and contraindications of various drugs used in the management of hormonal acne.

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TABLE 1: Various drugs—their mechanism of action and adverse effects.

	Spironolactone	Flutamide	Cyproterone acetate	Combined oral contraceptives (COCs)
Mechanism of action	*Decreases*: • 5α-reductase activity • Free testosterone • Sebaceous gland size and activity	Blocks nuclear binding of androgens	• Blocks testosterone receptors • Decreases release of gonadotropins	*Decreases*: • 5α-reductase activity • Free testosterone • Sebaceous gland size and activity
Dose	50–200 mg daily	62.5–500 mg/day	• 50–100 mg/day from days 1 to 10 of the menstrual cycle • 2 mg with 35 µg of ethinyl estradiol in COCs	• Estrogens—30 to 50 µg • Progesterone • Cyproterone acetate—2 mg Drospirenone—3 mg
Adverse effects	• Hypercalcemia • Breast tenderness • Menstrual irregularities	• Hematological disorders • Muscle cramps • Gynecomastia • Acute liver failure	• Headache • Breast tenderness • Menstrual irregularities • Fluid retention • Liver dysfunction • Blood clotting disorders	• GI disorders • Spotting • Headache • Breast tenderness • Fluid retention • Depression
Contraindications	• Hypotension • Reduced libido • Hyperkalemia • Estrogen-dependent malignancy	Liver disease	• History of meningioma • Liver disease • Hematological disorders • Diabetes • Sickle cell anemia • Severe depression	• Smoking in age >35 years • Cardiovascular system (CVS) diseases • Hematological disorders • Body mass index (BMI) ≥35 • Migraine • Estrogen-dependent malignancy

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RECENT ADVANCES

Vitamin D deficiency can lead to irregular menses, hirsutism, hyperandrogenism associated with PCOS and insulin resistance. Vitamin D supplementation may have advantages.[12]

Sebo-suppressive effect of isotretinoin has been shown to reduce DHT formation and androstenedione as it causes competitive inhibition of 3α-hydroxysteroid oxidation by retinol dehydrogenase.[13]

Topical 1% clascoterone cream (cortexolone 17α-propionate) is the first topical AR inhibitor approved by the US FDA for acne in patients ≥12 years.[14]

CONCLUSION

Pathogenesis, clinical presentation and treatment of hormonal acne is different from classical acne vulgaris. Because of associated systemic abnormalities, the cases of hormonal acne should be given special emphasis and warrant different management approach to correct the endocrine abnormalities. The current chapter has covered all the aspects of hormonal acne which will be definitely a guiding source for the readers to deal with the cases of hormonal acne.

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Handbook on
ACNE

Salient Features

- This book covers acne on a wider perspective by focusing not only on pathophysiology, clinical features, differentials, and treatment modalities but also on psychosocial aspects and sequel of acne in detail.
- Very good-quality images have been used to show minute details and several variants of acne, including the mimickers.
- Clinical approach to a case of acne, role of hormones in acne, and approach to hormonal acne have been discussed in simple language.
- Several treatment modalities: Topical, systemic, and laser-based—have been discussed.
- This book also covers the interesting aspect of the role of diet in acne.
- The language is very simple and lucid to understand which will help the students and treating physicians to understand the basics of acne and help them to manage acne cases.



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