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A Cross-Sectional Study in Women With Adult Acne: Clinical Profile, Hormonal Evaluation, and Psychological Impact at a Tertiary Hospital

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Abstract

Background

Acne vulgaris is a key inclusion in the global burden of disease. The pathogenesis often remains elusive, with endocrine disequilibrium being cited as a major influence in recalcitrant acne in females. This study aimed to conduct a clinical evaluation of adult acne according to history, hormonal profiles, and psychological impact.

Methods

This was a cross-sectional descriptive study including patients with adult acne attending MGM Dermatology OPD in Aurangabad, India, from March 2021 to March 2023. One hundred adult patients were enrolled in the study. They were evaluated for hirsutism, hair fall, and menstrual disturbances based on clinical history and examination. Subsequently, serological hormonal investigations and pelvic ultrasound were done for all patients.

Results

Among 100 female patients (mean age 31 years), 48 (48%) had persistent acne and 52 (52%) had late-onset acne, with duration ranging from 1 month to 25 years. Common grades were grade II (n=53; 53%), grade I (n=37; 37%), and grade III (n=9; 9%). A total of 33 (33%) individuals had a history of hirsutism, and 6 (6%) had alopecia. Investigations showed normal dehydroepiandrosterone sulphate (DHEA-S), increased prolactin in 12 (12%), reduced luteinizing hormone (LH) in 51 (51%), follicle-stimulating hormone (FSH) changes (increased in 2 (2%), decreased in 24 (24%)), 6 (6%) with elevated fasting insulin, and 10 (10%) showed raised postprandial insulin. Ultrasound confirmed polycystic ovarian syndrome (PCOS) in 21 (21%) of patients.

Conclusion

In adult females, late-onset acne was slightly more frequent than persistent acne, and most patients had grade II acne. Statistically significant associations were observed between acne severity and FSH, thyroxine (T4), and postprandial insulin, whereas no significant associations were found with other hormonal parameters. Studies of end-organ sensitivity will further aid in understanding the hormonal role in adult acne.

Categories: Internal Medicine, Endocrinology/Diabetes/Metabolism, Dermatology
Keywords: acne vulgaris, adult female acne, adult-onset acne, epidemiology, hormonal acne, lifestyle

Introduction

Acne is one of the most commonly encountered diseases in clinical practice. The presentation of acne depends on multiple factors. It is commonly seen in adolescents. Acne that continues to persist even after the age of 25 years is labelled as persistent adult acne. Acne developing for the first time after 25 years of age is labelled as late-onset or delayed adult acne [1].

Traditionally, acne was considered a disease of adolescence. However, numerous studies have shown that it can persist into adulthood or develop de novo in adults. The prevalence varies amongst various studies, ranging from 12% to 54% in adult women. Hence, adult acne is now considered a distinct clinical subtype, owing to its frequent associations with endocrine and metabolic aberrations. Even though acne has been investigated extensively, large-scale population-based studies elucidating its epidemiology and pathogenesis remain scarce, and most have been conducted on Western populations [2]. The commonly

implicated triggers of acne include cosmetics, stress, hormones, and bacterial colonization and have been corroborated by numerous studies [3]. Khunger and Kumar revealed that hormonal factors have a potential role in the provocation of acne [4]. In contrast, an original study by Slayden et al., which included 30 patients, concluded that acne severity was not associated with BMI, hormonal profile, and age [5]. Goulden et al., in their community-based study of 361 patients, noticed that hyperandrogenism was present in 37% of subjects [6]. Similarly, Aizawa et al. noted significantly higher values of DHT and adrenal androgens when comparing females with acne versus women without acne [7].

Many studies have examined acne-related stress in patients; however, very few look into stress as an etiological factor in causing acne [8]. The complex etiopathogenesis and varied clinical presentation make it necessary to properly investigate the patient and plan management. Numerous dermatological studies have demonstrated that skin cells have a significant role in both producing hormones and expressing various endocrine-related receptors that can modify their function [9]. Corticotrophin-releasing hormone receptor 2, which is found on sebocytes, has a role in coordinating the stress response and promotes sebaceous lipid synthesis. Insulin-like growth factors and growth hormone both stimulate sebocyte differentiation, proliferation, and lipogenesis. The skin behaves like an endocrine organ, expressing androgen receptors, which aid in their role during epidermal proliferation, hair follicle development, and sebogenesis. The skin also possesses certain cell signaling pathway enzymes that have an instrumental role in the local conversion of weaker androgens to their more potent forms [9]. Through the action of enzymes such as 17-beta-hydroxysteroid dehydrogenase and 5-alpha reductase, DHEA and testosterone can be converted to their more active metabolites such as DHT. The process can take place in both keratinocytes and sebocytes. This amplified androgen response, secondary to potent androgen stimulation, is a key mechanistic driver in the pathogenesis of hormonally aggravated dermatoses such as hirsutism, acne, and androgenetic alopecia. These androgens can increase sebaceous gland secretion, especially in the facial sites, usually seen in the chin and jawline, as these sites are sensitive to androgen stimulation.

A complex interplay between hormonal, psychological, and metabolic processes has been instrumental in initiating and aggravating adult acne. However, a paucity of studies on adult females from India introduces a lacuna in the scientific literature. Through this study, we comprehensively investigated the correlation between metabolic profile, hormonal parameters, pelvic ultrasonographic findings, and clinical features. We aim to estimate the prevalence of precipitating factors and assess mental health impacts, addressing the critical need to uncover hormonal drivers, modifiable triggers, and psychological burdens for improved diagnosis and holistic management in affected adults.

Materials And Methods

Ethical approval

Approval was obtained from the scientific and ethical committee (MGM-SAC/05/2023 & MGM-ECHRS/14/225), and informed consent was obtained.

Study design and setting

This is a cross-sectional descriptive study. The study was conducted in a tertiary care center, MGM Medical College and Hospital, MGM Institute of Health Sciences, Aurangabad, India, from March 2021 to March 2023.

Inclusion and exclusion criteria

Female patients aged 25 years and above with acne on the face, chest, back, shoulders, arms, buttocks, and trunk were enrolled in this study. Patients with pre-existing diagnoses of psychiatric illnesses were excluded.

Clinical evaluation

A structured clinical assessment using a comprehensive dermatological history and clinical examination of the patient was performed. Data collected involved factors including key demographic characteristics such as age, sex, age of onset, duration of symptoms, family history, obstetric and menstrual history, concurrent medication use, and dietary history. Participants were also asked about potential aggravating factors (sun, stress, seasonal variations, cosmetics, medications, premenstrual and menstrual aggravations). Acne can be classified into four grades of severity. These include Grade I (comedones, occasional papules); Grade II (papules, comedones, few pustules); Grade III (predominant pustules, nodules, abscesses); Grade IV (mainly cysts, abscesses, widespread scarring) [4].

Investigations

All patients were subjected to comprehensive laboratory testing. The laboratory investigations included follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, triiodothyronine (T3), thyroxine (T4), thyroid-stimulating hormone (TSH), dehydroepiandrosterone sulphate (DHEA-S), total testosterone, fasting insulin, postprandial insulin, fasting blood sugar, and Ultrasonography (USG) of the abdomen and pelvis findings. Postprandial insulin was calculated using a biochemical chemiluminescence immunoassay technology (CLIA test) taken 2 hours after a meal. Insulin resistance (IR) was estimated using the

Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) [10]. Values above 2.5 were suggestive of insulin resistance. Polycystic ovarian syndrome (PCOS) was diagnosed according to the Rotterdam criteria, and hirsutism was evaluated using the Ferriman-Gallwey scale [11]. Clinical photographs for all the patients and anonymized patient records were included to monitor and assess the patients.

Psychological assessment

Patients were also screened for anxiety and depression using the Hamilton Anxiety Rating Scale (HAM-A) [12] and Hamilton Depression Rating Scale (HAM-D) [13], respectively. Stress was measured using the Perceived Stress Scale (PSS) [14]. Additionally, patients were asked about embarrassment, shame, and reduced self-confidence to better evaluate the psychosocial burden of disease.

Statistical analysis

After completing the investigations and history taking, the data were entered into a Microsoft Excel spreadsheet (Microsoft Corp., Redmond, WA), and SPSS software version 25.0 (IBM Corp., Armonk, NY) was used for statistical analysis. Continuous variables were summarized as means ± standard deviations (SD), and categorical variables were expressed as frequencies and percentages, n (%). Associations between categorical variables, such as hormonal or metabolic profiles and acne severity grades, were calculated using Fisher's exact test with Monte Carlo simulation. A p-value < 0.05 was considered statistically significant.

Results

Demographic details of the study participants have been summarized in Table *1*.

Characteristic	Value (N = 100)
Age (years); mean ± SD	31.18 ± 5.8
Age (years); range	25-48
Age distribution, n (%)	
25-30 years	56 (56%)
31-35 years	20 (20%)
36-48 years	24 (24%)
Clinical characteristics	
Age of onset (years), mean ± SD	26.46 ± 6.85
Duration of disease (years), mean	4.72
Duration of disease, range	1 month-25 years
BMI (kg/m²), mean	25.34
Family history of acne, n (%)	31 (31%)
Acne subtype, n (%)	
Persistent acne	48 (48%)
Delayed onset	52 (52%)

TABLE 1: Demographic details of study participants

Lesions most frequently involved the cheeks (n=98; 98%), forehead (n=82; 82%), and mandibular region (n=50; 50%). The temporal region of the face was only involved in 27 (27%) of participants. Truncal, limb, and buttock involvement was uncommon (n=16; 16%). (Table *2*). Patients predominantly presented with Grade II acne (n=53; 53%), followed by Grade I (n=37; 37%), Grade III (n=9; 9%), and Grade IV (n=1; 1%).

Cutaneous examination	Category	No. of Patients	Percentage
Acne grade	I	37	37%
	II	53	53%
	III	9	9%
	IV	1	1%
Sites involved	Cheeks	98	98%
	Forehead	82	82%
	Mandible	50	50%
	Temporal	27	27%
	Arms	9	9%
	Trunk	5	5%
	Buttocks	2	2%

TABLE 2: Distribution according to grades of acne and sites

P-value of <0.05 was considered statistically significant

Key clinical features such as hormonal profile, signs and symptoms, and comorbidities have been mentioned in Table 3.

Characteristic	Value (N = 100)
Symptoms, n (%)	
Redness	80 (80%)
Itching	72 (72%)
Picking	64 (64%)
Pain	28 (28%)
Burning sensation	17 (17%)
Aggravating factors, n (%)	
Stress	53 (53%)
Cosmetics	49 (49%)
Summer aggravation/sun exposure	32 (32%)
Certain medications (oral or topical corticosteroids)	16 (16%)
Winter exacerbation	3 (3%)
History of trauma	0 (0%)
Onset of lesions, n (%)	
Gradual onset	77 (77%)
Sudden onset	23 (23%)
Lifestyle and dietary habits, n (%)	
Mixed diet	85 (85%)
Vegetarian	15 (15%)
Regular cosmetic use	59 (59%)

Pomade oil use	5 (5%)
Excessive face washing (>5 times/day)	18 (18%)
Junk food intake	37 (37%)
Regular consumption of chocolate, nuts, and ice cream	56 (56%)
Excessive milk product intake (>240 ml/day)	18 (18%)
Regular carbonated drinks	16 (16%)
Passive smoking	2 (2%)
Tobacco use	1 (1%)
Alcohol consumption	0 (0%)
Menstrual and obstetric history	
Regular menses, n (%)	69 (69%)
Mild flow	13%
Moderate flow	79%
Severe flow	8%
Irregular menses (oligomenorrhea)	31 (31%)
Dysmenorrhea, n (%)	36 (36%)
Age of menarche (years), mean ± SD	13.89 ± 1.47
Cycle length (days), mean ± SD	31.89 ± 20.39
Duration of menses (days), mean	5.67
Premenstrual acne flares, n (%)	47 (47%)
Offspring, n (%)	66 (66%)
History of infertility, n (%)	10 (10%)
Sought infertility treatment, n (%)	5 (5%)
Clinical findings and comorbidities, n (%)	
Weight gain	39 (39%)
Seborrhoea (historical)	30 (30%)
Decreased scalp hair	14 (14%)
History of hirsutism	33 (33%)
Clinically by Ferriman-Gallwey score ≥ 8 (hirsutism)	9 (9%)
Androgenetic alopecia	6 (6%)
Polycystic ovarian syndrome (PCOS) confirmed on ultrasound	21 (21%)
Couple infertility	9 (9%)
Hypothyroidism	7 (7%)
Diabetes	1 (1%)
Drug consumption history, n (%)	
Oral contraceptive pills (OCP) intake	12 (12%)
Steroid use (oral or topical)	10 (10%)
Androgenic hormone use	6 (6%)
B12 supplement use	4 (4%)

Dermatological examination, n (%)	
Seborrhoea	25 (25%)
Overall skin pigmentation	17 (17%)
Striae	14 (14%)
Acanthosis nigricans	9 (9%)
Orogenital pigmentation	6 (6%)
Palmoplantar pigmentation	3 (3%)
Pigmentary demarcation lines	1 (1%)

TABLE 3: Key clinical features such as hormonal profile, signs and symptoms and comorbidities

Hormonal and metabolic profiles of patients with corresponding reference ranges are mentioned in Table *4*. Statistically significant associations were observed between acne severity and FSH ($p = 0.002$), T4 ($p = 0.048$), and postprandial insulin ($p < 0.001$). No statistically significant associations were observed for LH, prolactin, total testosterone, T3, TSH, fasting blood sugar, fasting insulin, or HOMA-IR ($p > 0.05$). An association with DHEA-S could not be assessed because all participants had normal DHEA-S levels.

Investigation	Reference Range	Unit	Normal, n (%)	Decreased, n (%)	Elevated, n (%)	Statistical Test	p-value	Statistical Significance
FSH	3.4-21.5	mIU/mL	74 (74%)	24 (24%)	2 (2%)	Fisher's exact test (Monte Carlo)	0.002	Significant
LH	8.7-76.3	mIU/mL	49 (49%)	51 (51%)	0 (0%)	Fisher's exact test (Monte Carlo)	0.633	Not significant
Prolactin	2.8-29.2	ng/mL	85 (85%)	3 (3%)	12 (12%)	Fisher's exact test (Monte Carlo)	0.328	Not significant
Total testosterone	0.1-0.75	ng/mL	90 (90%)	1 (1%)	9 (9%)	Fisher's exact test (Monte Carlo)	0.574	Not significant
DHEA-S	35-430	µg/dL	100 (100%)	0 (0%)	0 (0%)	Not applicable	-	Not testable
T3	60-181	ng/dL	89 (89%)	2 (2%)	9 (9%)	Fisher's exact test (Monte Carlo)	0.157	Not significant
T4	4.6-10.9	µg/dL	80 (80%)	2 (2%)	18 (18%)	Fisher's exact test (Monte Carlo)	0.048	Significant
TSH	0.35-5.5	µIU/mL	98 (98%)	-	2 (2%)	Fisher's exact test (Monte Carlo)	1	Not significant
FBS	70-10	mg/dL	84 (84%)	1 (1%)	15 (15%)	Fisher's exact test (Monte Carlo)	0.767	Not significant
Fasting insulin	3.0-25.0	mU/L	93 (93%)	1 (1%)	6 (6%)	Fisher's exact test (Monte Carlo)	0.656	Not significant
Postprandial insulin	26-84	mU/L	79 (79%)	11 (11%)	10 (10%)	Fisher's exact test (Monte Carlo)	<0.001	Significant
HOMA-IR (Insulin Resistance)	< 2.5	Index	52 (52%)	-	48 (48%)	Fisher's exact test (Monte Carlo)	0.977	Not significant

TABLE 4: Association of hormonal and metabolic profiles with acne severity grades in female adults

Hormonal and metabolic profiles of patients with acne, categorized according to laboratory reference ranges and evaluated for associations with acne severity grades. Data are presented as number and percentage of patients, n (%). Associations between acne grades and hormonal or metabolic parameters were assessed using Fisher's exact test with Monte Carlo simulation, wherever applicable. A p-value of less than 0.05 was considered statistically significant.

FSH, follicle-stimulating hormone; LH, luteinizing hormone; DHEA-S, dehydroepiandrosterone sulphate; T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone; FBS, fasting blood sugar; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance.

The hormonal panel revealed elevated serum total testosterone levels in 9 (9%) of patients. A total of 12 (12%) of the patients exhibited hyperprolactinemia (>29.2 ng/ml). Among these, seven had mild elevation (one- to two-fold increase), two patients had moderate increase (two- to three-fold), and three patients had severe elevation (more than three-fold increase). TSH and FSH levels were mildly elevated (< 2 times the upper limit) in 2% of the patients. A total of 51% of patients presented with low LH (<8.7 mIU/ml) and 24% had decreased FSH (<3.4 mIU/ml). LH: FSH ratio >2 was present in 23 (23%) patients. Increased fasting insulin levels were seen in 6 (6%) patients, with elevated postprandial insulin levels in 10 (10%). A total of 15 (15%) of patients had impaired fasting blood glucose levels (susceptible to developing insulin resistance). HOMA-IR surpassed the threshold of 2.5 in 48 (48%) of participants, suggesting insulin resistance, and 15 (15%) patients had coexisting PCOS and insulin resistance (Figure *1*).

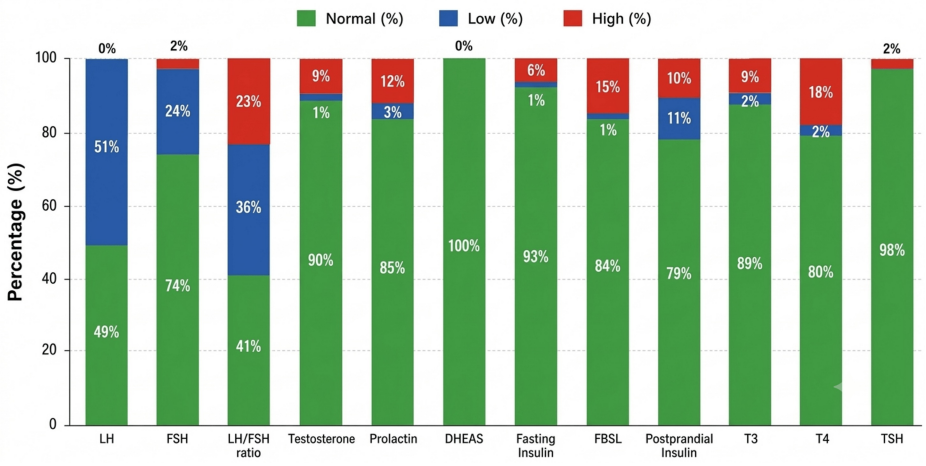


FIGURE 1: Graphical representation of patient profiles with reference to hormonal assays

FSH, follicle-stimulating hormone; LH, luteinizing hormone; DHEA-S, dehydroepiandrosterone sulphate; T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone; FBS, fasting blood sugar; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance

Psychological assessment findings have been summarized in Table 5.

Characteristic	Value (N = 100)
General Psychological Assessment, n (%)	
Anxiety (self-reported on taking medical history)	50 (50%)
Embarrassment	49 (49%)
Shame	31 (31%)
Low confidence	26 (26%)
Hamilton Anxiety Rating Scale (HAM-A), n (%)	
Mild anxiety	22 (22%)
Hamilton Depression Rating Scale (HAM-D), n (%)	
Mild depression	13 (13%)
Perceived Stress Scale (PSS), n (%)	
Mild stress	29 (29%)
Moderate stress	69 (69%)
Severe stress	2 (2%)

TABLE 5: Psychological assessment of the study population

Discussion

Adult acne is a distinct clinical entity with a multifactorial etiopathogenesis, including metabolic, environmental, endocrine, immunological, and psychological factors. However, the exact cause is still poorly understood. Hence, our study incorporated a comprehensive clinical, hormonal, endocrine, and psychological evaluation to better evaluate these factors in the initiation and aggravation of adult acne. Cunliffe et al. found that acne was more prevalent before the age of 16 years in the male population and was prevalent beyond the age of 23 years in the female population [15]. Hence, we enrolled women with acne aged 25 years and older. Among the participants, 36 (36%) had dysmenorrhea, 31 (31%) had irregular menses, and 47 (47%) had a history of premenstrual flares. Similarly, Goulden et al. [6] observed that there

was an aggravation of acne in the premenstrual period in 84.8%. Steinberger et al. found that 41% of the participants complained of irregular menses [16]. Sardana et al. also described irregular menses in their cohort, but only 25.83% were affected [17]. Khunger et al. noted that 73.2% had persistent acne and 26.8% had late-onset adult acne [4]. According to Goulden et al. [6], persistent acne was seen in 82% of his study patients; however, in our study, only 48 (48%) patients had a similar history.

The probable factors that cause acne exacerbations were evaluated. These include cosmetic application, corticosteroid application, stress, oral drug intake, trauma, seasonal variation, and sun exposure. Similar observations were made by George et al. [18], who stated that acne had been aggravated by cosmetic usage in 40%. A total of 2.7% of patients had aggravation of lesions after topical application of steroids. A total of 26.4% of patients had a history of increased lesions with prolonged sunlight exposure. Furthermore, 42.7% said that they had exacerbation of lesions during summer, and 32.7% of patients reported emotional stress as a contributing reason [18]. Khanna et al. observed that 33.1% of patients had acneiform eruptions after procedures like facials [19]. Khunger and Kumar [4] also reported that 22% of patients had increased acne with use of cosmetics. In 33.2% of patients, exacerbation was seen with sun exposure. These findings suggest the role of external factors in adult acne. The factors act via distinct pathways to induce and aggravate acne. Comedogenic cosmetic products may physically occlude sebaceous follicles, leading to microcomedone formation. Corticosteroids play a role in reducing local immunity and promoting hyperkeratosis, and the follicular ostia eventually lead to monomorphic inflammatory eruption without the presence of comedones. Stress stimulates neuroendocrine mediators such as corticotropin-releasing hormone (CRH), which promote sebogenesis [8]. Trauma and friction alter pilosebaceous epithelial integrity, leading to follicular occlusion and subsequent inflammation. Seasonal variation, especially summer temperatures in tropical and subtropical countries, can exacerbate acne via UVA-induced oxidation of squalene to the formation of comedogenic byproducts. UVB induces keratinocyte proliferation, sebum biogenesis, and upregulation of cytokines such as IL-1 and IL-8. Furthermore, pore occlusion may take place due to excessive hydration of the skin in humid conditions [3].

The present study highlights that 31 (31%) had a positive history of acne in the family. These findings are in agreement with those reported by George et al. [18]. They revealed that 48.2% of their patients had first-degree family members who experienced acne in the past. Descriptively, a substantial proportion of individuals in our cohort reported regular consumption of junk food 37 (37%), chocolate, nuts and ice cream 56 (56%), excessive milk product intake 18 (18%), and regular carbonated drinks 16 (16%). Similarly, Di Landro et al. [20] found that lactose-containing substances such as dairy products correlated with aggravation of acne. Goulden et al. [6] also highlighted the role of diet in the pathogenesis of acne.

The history of trichological and endocrinological factors such as hirsutism, decrease of scalp hair, male pattern alopecia, polycystic ovary syndrome (PCOS), a history of thyroid disorder, and diabetes are also seen with adult acne. 33 (33%) patients gave a history of hirsutism, and 14 (14%) patients reported decreased scalp hair. Androgenic alopecia was reported by 6 (6%) patients. Pelvic ultrasound confirmed PCOS in 21(21%) of patients. A total of 7 (7%) of patients in the study gave a history of thyroid disorder; however, only 2 (2%) showed increased TSH, and only 1 (1%) of patients gave a history of diabetes. Gayen et al. [21] found that 28.2% of the study population had hirsutism, which is consistent with our study. Among the various factors, the average BMI was 25.34 kg/m2.

The medication history in this study revealed that 6 (6%) utilized androgenic hormones, 10 (10%) had consumed corticosteroids, 12 (12%) were using oral contraceptive pills, and 4 (4%) were taking additional B12 supplements. Topical and systemic corticosteroid usage 10 (10%) and androgenic hormone intake 6 (6%) were notable medication-related aggravating factors reported by our patients with adult acne. A study by Gayen et al. [21] revealed that acanthosis nigricans and acne were seen in 31.5% of subjects, but these diseases coexisted in only 9 (9%) of our patients.

A vast majority of our participants had Grade II acne, predominantly distributed over the cheek and forehead. This suggests that adult acne commonly presents as mild to moderate inflammatory papules. This is consistent with the findings of Khunger and Kumar [4], who noticed that 55% of their patients had Grade II acne, 6 % had Grade I, 28% had Grade III, and 12% had Grade IV. A study by Anni Telkkälä [22] observed that almost a fifth of the cases had acne only on the face, mostly on the chin and cheeks.

In our study, only 9 (9%) of patients had an increase in total testosterone levels, and DHEA-S levels were normal in all patients. These findings suggest that end-organ sensitivity can play a larger role than androgen levels in adult acne. FSH was normal in most patients, while LH was slightly decreased in 51 (51%) of patients; however, the ratio of LH: FSH was elevated (>2) in 23 (23%) and it was not elevated in the remaining 77 (77%). In a study by Ding et al., normal levels of LH and FSH were seen; however, the ratio of LH/FSH was raised [23].

Most patients had a normal thyroid profile, with derangements only detected in a few. Surprisingly, 93 (93%) of patients had normal fasting insulin, and 79 (79%) had normal postprandial insulin levels. Fasting sugar levels were normal in 84 (84%) patients. By calculating the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), insulin resistance was seen in 48 (48%) with HOMA-IR greater than 2.5; 15 (15%)

were susceptible to developing IR, and another 15 (15%) had PCOS and IR. Kaymak et al. [24] had similar findings, stating that cases and controls of acne had no significant difference in blood glucose, fasting insulin, and HOMA-IR. Çetinözman et al. [25] noted no correlation between hyperandrogenemia and IR in patients having severe acne. In contradiction to the above studies, Del Prete et al. stated that fasting blood sugar, insulin levels, and HOMA-IR were elevated in men having adult acne compared to controls [26]. Aizawa et al. found that raised levels of IGF-1 and mild insulin resistance (IR) were seen in females having acne, without PCOS [27]. IR causes the pituitary gland to secrete LH, further increasing androgen synthesis and inhibiting SHBG production. This causes high circulatory levels of free testosterone, which disrupts ovulatory functions [23].

The nomenclature of PCOS has been recently modified to polyendocrine metabolic ovarian syndrome (PMOS). PMOS, till recently, was considered a primary gonadal axis disorder because of disturbances in the menstrual cycle. Recent evidence suggests that it has a multifaceted etiopathology showing disturbances in metabolic and endocrine metabolism, insulin, and androgens apart from ovarian hormones, indicating a multi-endocrine involvement that extends beyond ovarian pathology. Patients with hyperandrogenism, ovarian endocrine dysfunction, increased gonadotropin levels, and insulin resistance can present with obesity, hyperlipidemia, diabetes, hypertension, liver disease, cardiovascular disease, sleep apnea, psychological disturbances, poor quality of life, dermatological conditions, and reproductive and ovarian abnormalities. These multisystem disturbances have a significant impact on affected individuals and impose an economic burden [28]. Our study shows similar observations and is supported by the observations and findings of global consensus.

There was substantial psychological burden observed among the participants. Mallon et al. suggested that quality of life impairments in acne are comparable to other chronic systemic diseases like Diabetes, Asthma, epilepsy, and Arthritis [29]. Chiu et al. demonstrated experimental evidence of worsening acne with exam stress [30]. Anxiety and other psychological disturbances in patients with acne, with or without scars, can differ from individual to individual and are independent of the depth of the disease. Our study highlighted the presence of mild anxiety in 22 (22%) of subjects. Jovic et al. describe that this aggravation can be linked to neuroendocrine pathways which activate sebaceous gland secretion and promote cutaneous inflammation [8].

All patients had experienced some level of stress due to acne. An astounding 69 (69%) of patients had moderate stress. Sardana et al. [17] also found that stress co-exists with acne, with moderate to severe stress in 65.83% of patients and reduced sleep in 35%. He demonstrated that working women had higher levels of stress in comparison to homemakers and students. Goulden et al. [6] revealed that a stressful environment induces acne flare-ups in 71% of patients. Chiu et al. [30] proved that reduced sleep was one factor of stress in 97.61% of patients. Chronic stress can lead to increased secretion of androgens by adrenal glands, leading to hyperplasia of sebaceous glands. This eventually correlates with the severity of acne [8,30]. Mild depression was also observed in 13 (13%) patients in our study.

Limitations

The shortcomings of this study are a relatively small sample size, lack of a control arm, and a single-center design. Long-term follow-up of patients was not feasible. Genetic analysis and hormones such as sex hormone-binding globulin (SHBG) and anti-Müllerian hormone (AMH) were not included in the study due to limited resources.

Conclusions

This descriptive study demonstrates that patients with adult female acne frequently present with potential precipitating factors, including endocrinological disturbances, dietary triggers, and psychological stress. Statistically significant associations were observed between acne severity and FSH, T4, and postprandial insulin, whereas no significant associations were found with other hormonal parameters. Studies of end-organ sensitivity will further aid in understanding the hormonal role in adult acne. Insulin resistance (IR), evident through increased HOMA-IR values in many acne patients, suggests that all patients can be screened for metabolic syndrome and insulin resistance, if these investigations are easily available and affordable.

This study was conducted in a tertiary health care facility in a developing nation with limited resources. In similar settings, we recommend that testing be done judiciously only on those patients with clinical features of concomitant endocrinological disorders. Future prospective studies may also explore the role of glycemic load, diet, synthesis of androgens, and local and gut bacterial flora. The three main axes in the pathogenesis of acne, which can be explored according to the latest research, include the gut-immune-brain axis, neurogastroenterology and the enteric nervous system, and the psychoneuroimmunoendocrine axis.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the

work.

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Acquisition, analysis, or interpretation of data: Mohammed Tariq Iqbal, Anirudha D. Gulanikar, Shilpa Haibatti (Pathrikar), Gunreddy Aruna, Japsimran Nagpal

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Ethics Committee for Research on Human Subjects issued approval MGM-ECHRS/14/225. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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