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Original Research Article

CLINICOPATHOLOGICAL CORRELATION OF ACNE VULGARIS IN ADOLESCENTS AND YOUNG ADULTS: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acne vulgaris is one of the most common chronic inflammatory disorders of the pilosebaceous unit affecting adolescents and young adults. It results from a complex interaction of follicular hyperkeratinization, increased sebum production, Cutibacterium acnes colonization, and inflammation. Clinicopathological correlation provides valuable insights into disease severity, progression, and therapeutic approaches. The objective is to evaluate the clinical profile of acne vulgaris and correlate clinical severity with histopathological findings among adolescents and young adults. **Materials and Methods:** A prospective observational study was conducted in the Department of Dermatology, Faculty of Medical Sciences, KBN University, Kalaburagi, Karnataka, from January 2024 to August 2024. A total of 75 clinically diagnosed cases of acne vulgaris aged 13–30 years were enrolled. Detailed clinical examination, grading of acne severity, and histopathological evaluation of representative lesions were performed. Data were analyzed using appropriate statistical methods, and clinicopathological correlations were assessed. **Result:** Among the 75 study participants, the majority belonged to the age group of 16–20 years. Females constituted a slight predominance. Grade II acne was the most common clinical presentation. Histopathological examination predominantly revealed follicular hyperkeratinization, sebaceous gland hypertrophy, perifollicular inflammation, and inflammatory cell infiltrates. A significant correlation was observed between clinical severity and the degree of inflammatory changes on histopathology ($p < 0.05$). **Conclusion:** Acne vulgaris demonstrates characteristic histopathological alterations that correlate with clinical severity. Understanding these clinicopathological associations can aid in better disease assessment and targeted management strategies.

INTRODUCTION

Acne vulgaris is a chronic inflammatory disorder of the pilosebaceous unit and represents one of the most prevalent dermatological conditions worldwide. It predominantly affects adolescents and young adults, with nearly 85% of individuals experiencing some degree of acne during their lifetime. Although acne is not a life-threatening condition, its visible nature often leads to significant psychological distress, impaired self-esteem, social withdrawal, anxiety, and depression, particularly among young individuals during formative years of life.[1,2]

The pathogenesis of acne vulgaris is multifactorial and involves a complex interplay between increased sebum production, follicular hyperkeratinization, colonization by Cutibacterium acnes (formerly Propionibacterium acnes), and inflammatory mechanisms.[3] The process begins with abnormal desquamation of follicular keratinocytes leading to obstruction of the pilosebaceous duct and formation of microcomedones. Subsequent accumulation of sebum creates an environment favorable for bacterial

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proliferation, triggering innate and adaptive immune responses that contribute to the development of inflammatory lesions.[4]

Recent advances in acne research have highlighted the pivotal role of inflammation throughout all stages of disease development. Studies have demonstrated elevated expression of pro-inflammatory cytokines such as interleukin (IL)-1β, IL-8, IL-17, and tumor necrosis factor-alpha (TNF-α) within acne lesions.[5] These inflammatory mediators promote recruitment of neutrophils, lymphocytes, and macrophages, resulting in the characteristic papules, pustules, and nodules observed clinically. Furthermore, persistent inflammation may culminate in dermal destruction and permanent scar formation.[6]

Several factors influence the occurrence and severity of acne, including hormonal fluctuations, genetic predisposition, dietary habits, stress, cosmetic use, environmental exposures, and lifestyle practices.[7] Hyperandrogenism stimulates sebaceous gland activity and contributes to increased sebum secretion, making adolescence a particularly vulnerable period for acne development. In females, menstrual irregularities and endocrine disorders may further exacerbate disease severity.[8]

Histopathological examination of acne lesions provides valuable insights into the underlying pathological changes occurring within the skin. Early lesions typically demonstrate follicular hyperkeratinization and sebaceous gland enlargement, whereas advanced lesions reveal follicular rupture, intense perifollicular inflammation, granulomatous reactions, and fibrosis.[9] These microscopic findings often parallel clinical severity and may help explain variations in disease presentation and progression.

Despite the high prevalence of acne vulgaris, studies evaluating clinicopathological correlations remain relatively limited, particularly in the Indian population. Understanding the relationship between clinical manifestations and histopathological changes may improve disease assessment and contribute to more targeted therapeutic strategies. Therefore, the present study was undertaken to evaluate the clinical profile and histopathological features of acne vulgaris and to determine their clinicopathological correlation among adolescents and young adults attending the Department of Dermatology, Faculty of Medical Sciences, KBN University, Kalaburagi, Karnataka.

Objectives

1. To assess the demographic and clinical profile of patients with acne vulgaris.
2. To classify acne vulgaris according to clinical severity.
3. To evaluate histopathological changes in acne lesions.
4. To correlate clinical severity with histopathological findings.

MATERIALS AND METHODS

Study Design: Prospective observational study.
Study Setting: Department of Dermatology, Faculty of Medical Sciences, KBN University, Kalaburagi, Karnataka.
Study Duration: January 2024 to August 2024.
Sample Size: 75 cases.

Inclusion Criteria

- Patients aged 13–30 years.
- Clinically diagnosed cases of acne vulgaris.
- Patients willing to provide informed consent.

Exclusion Criteria

- Patients receiving systemic retinoids within the previous six months.
- Patients with drug-induced acneiform eruptions.
- Patients unwilling to participate.

Data Collection: Detailed history regarding age, gender, duration of disease, family history, dietary habits, cosmetic use, and aggravating factors was obtained. Clinical examination included assessment of lesion type, distribution, and acne grading.

Acne Grading

Grade I: Comedones with occasional papules.
Grade II: Papules with comedones and few pustules.
Grade III: Predominantly pustules and nodules.
Grade IV: Severe nodulocystic acne with scarring.

Histopathological Examination: Representative punch biopsy specimens were obtained from inflammatory lesions after informed consent. Tissues were fixed in 10% formalin, processed routinely, embedded in paraffin, sectioned, and stained with haematoxylin and eosin.

Histopathological parameters evaluated included:

- Follicular hyperkeratinisation
- Sebaceous gland hypertrophy
- Follicular rupture
- Perifollicular inflammation
- Neutrophilic infiltrates
- Lymphocytic infiltrates
- Foreign body giant cell reaction
- Fibrosis and scarring

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 25. Quantitative variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Chi-square test was used to assess clinicopathological correlations. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] shows the age-wise distribution of the study participants. The majority of patients belonged to the 16–20 years age group, accounting for 41.3% (n=31) of cases, followed by the 21–25 years age group comprising 29.3% (n=22). Participants aged 13–15 years constituted 16.0% (n=12), while those aged 26–30 years represented the smallest proportion at 13.4% (n=10). The findings indicate that acne vulgaris was

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most prevalent during late adolescence and early adulthood.

Table 1: Age Distribution of Study Participants

Age Group (Years)	Number of Cases	Percentage (%)
13–15	12	16
16–20	31	41.3
21–25	22	29.3
26–30	10	13.4
Total	75	100

[Table 1] shows the age-wise distribution of the study participants. The majority of patients belonged to the 16–20 years age group, accounting for 41.3% (n=31) of cases, followed by the 21–25 years age group comprising 29.3% (n=22). Participants aged 13–15 years constituted 16.0% (n=12), while those aged 26–30 years represented the smallest proportion at 13.4% (n=10). The findings indicate that acne vulgaris was most prevalent during late adolescence and early adulthood.

Table 2: Gender Distribution of Study Participants

Gender	Number of Cases	Percentage (%)
Male	32	42.7
Female	43	57.3
Total	75	100

[Table 2] depicts the gender distribution of the study population. Females constituted 57.3% (n=43) of the participants, whereas males accounted for 42.7% (n=32). The study demonstrated a slight female predominance among patients presenting with acne vulgaris.

Table 3: Duration of Acne Vulgaris

Duration	Number of Cases	Percentage (%)
< 6 months	18	24
6–12 months	29	38.7
1–2 years	20	26.7
> 2 years	8	10.6
Total	75	100

[Table 3] presents the duration of acne among the study participants. The majority of patients (38.7%, n=29) reported a disease duration of 6–12 months. This was followed by 26.7% (n=20) who had acne for 1–2 years and 24.0% (n=18) who had symptoms for less than six months. Only 10.6% (n=8) reported a disease duration exceeding two years. These findings suggest that most patients sought medical attention within the first year of disease onset.

Table 4: Distribution According to Clinical Severity

Acne Grade	Number of Cases	Percentage (%)
Grade I	16	21.3
Grade II	30	40
Grade III	21	28
Grade IV	8	10.7
Total	75	100

[Table 4] illustrates the clinical grading of acne vulgaris. Grade II acne was the most common presentation, observed in 40.0% (n=30) of cases. Grade III acne accounted for 28.0% (n=21), followed by Grade I acne in 21.3% (n=16) of patients. Grade IV acne was the least common, occurring in 10.7% (n=8) of participants. The findings indicate that moderate forms of acne were more prevalent than mild or severe disease.

Table 5: Distribution of Lesion Types (n-75)

Lesion Type	Number of Cases	Percentage (%)
Comedones	71	94.7
Papules	63	84
Pustules	42	56
Nodules	15	20
Scarring	19	25.3

[Table 5] summarizes the various lesion types observed among the participants. Comedones were the most common lesion, present in 94.7% (n=71) of patients, followed by papules in 84.0% (n=63).

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Pustules were noted in 56.0% (n=42), while nodules were present in 20.0% (n=15) of cases. Acne-related scarring was observed in 25.3% (n=19) of participants. These findings highlight the predominance of comedonal and papular lesions in the study population.

Table 6: Site of Involvement (n-75)

Site	Number of Cases	Percentage (%)
Face only	46	61.3
Face and back	17	22.7
Face and Chest	8	10.7
Face, Chest and Back	4	5.3
Total	75	100

[Table 6] demonstrates the anatomical distribution of acne lesions. The face alone was involved in the majority of patients, accounting for 61.3% (n=46) of cases. Combined involvement of the face and back was seen in 22.7% (n=17), while face and chest involvement was observed in 10.7% (n=8). Only 5.3% (n=4) of patients exhibited lesions involving the face, chest, and back simultaneously. These findings emphasize the face as the most frequently affected site.

Table 7: Histopathological Findings in Acne Vulgaris (n-75)

Histopathological Feature	Number of Cases	Percentage (%)
Follicular hyperkeratinization	68	90.7
Sebaceous gland hypertrophy	60	80
Perifollicular inflammation	57	76
Follicular rupture	26	34.7
Neutrophilic infiltrate	38	50.7
Lymphocytic infiltrate	54	72
Foreign body giant cells	11	14.7
Fibrosis/Scarring	18	24

[Table 7] presents the histopathological characteristics observed in biopsy specimens. Follicular hyperkeratinization was the most frequent finding, noted in 90.7% (n=68) of cases, followed by sebaceous gland hypertrophy in 80.0% (n=60) and perifollicular inflammation in 76.0% (n=57). Lymphocytic infiltrates were present in 72.0% (n=54), whereas neutrophilic infiltrates were observed in 50.7% (n=38). Follicular rupture was identified in 34.7% (n=26) of cases, while fibrosis or scarring and foreign body giant cell reactions were noted in 24.0% (n=18) and 14.7% (n=11), respectively. These findings support the role of follicular obstruction and inflammation in acne pathogenesis.

Table 8: Correlation Between Acne Severity and Perifollicular Inflammation

Acne Grade	Mild Inflammation	Moderate Inflammation	Severe Inflammation	Total
Grade I (n=16)	12 (75.0%)	4 (25.0%)	0 (0.0%)	16 (100%)
Grade II (n=30)	14 (46.7%)	13 (43.3%)	3 (10.0%)	30 (100%)
Grade III (n=21)	3 (14.3%)	10 (47.6%)	8 (38.1%)	21 (100%)
Grade IV (n=8)	0 (0.0%)	2 (25.0%)	6 (75.0%)	8 (100%)
Total (n=75)	29 (38.7%)	29 (38.7%)	17 (22.6%)	75 (100%)

Chi-square = 24.68, p = 0.001

[Table 8] demonstrates a significant association between acne severity and the degree of perifollicular inflammation. Mild inflammation was predominantly observed in Grade I acne (75.0%), whereas severe inflammation was most common in Grade IV acne (75.0%). As the clinical severity of acne increased from Grade I to Grade IV, the proportion of patients exhibiting severe inflammatory changes also increased. This association was found to be statistically significant ($\chi^2 = 24.68$, $p = 0.001$), indicating a strong correlation between clinical severity and histopathological inflammatory activity.

Table 9: Correlation Between Acne Grade and Follicular Rupture

Acne Grade	Follicular Rupture Present	Follicular Rupture Absent	Total
Grade I (n=16)	1 (6.3%)	15 (93.7%)	16 (100%)
Grade II (n=30)	7 (23.3%)	23 (76.7%)	30 (100%)
Grade III (n=21)	11 (52.4%)	10 (47.6%)	21 (100%)
Grade IV (n=8)	7 (87.5%)	1 (12.5%)	8 (100%)
Total (n=75)	26 (34.7%)	49 (65.3%)	75 (100%)

Chi-square = 21.53, p- 0.0001

[Table 9] shows the correlation between acne severity and the presence of follicular rupture. Follicular rupture was observed in only 6.3% of Grade I cases, whereas its prevalence increased progressively with disease severity, being present in 23.3% of Grade II, 52.4% of Grade III, and 87.5% of Grade IV cases. Conversely, the proportion of patients without follicular rupture decreased as acne severity

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increased. The association between acne grade and follicular rupture was statistically significant (χ^2 = 21.53, p < 0.001), indicating that follicular rupture is strongly associated with more severe forms of acne vulgaris.

Table 10: Correlation Between Acne Grade and Scarring

Acne Grade	Scarring Present	Scarring Absent	Total
Grade I (n=16)	0 (0.0%)	16 (100.0%)	16 (100%)
Grade II (n=30)	4 (13.3%)	26 (86.7%)	30 (100%)
Grade III (n=21)	8 (38.1%)	13 (61.9%)	21 (100%)
Grade IV (n=8)	7 (87.5%)	1 (12.5%)	8 (100%)
Total (n=75)	19 (25.3%)	56 (74.7%)	75 (100%)

Chi-square = 29.84, p- 0.0001

[Table 10] depicts the association between acne severity and the occurrence of scarring. No scarring was observed among patients with Grade I acne. The prevalence of scarring increased progressively with disease severity, being present in 13.3% of Grade II, 38.1% of Grade III, and 87.5% of Grade IV cases. Conversely, the proportion of patients without scarring decreased as acne severity increased. Statistical analysis revealed a highly significant association between acne grade and scarring (χ^2 = 29.84, p < 0.001), indicating that patients with severe acne are at a substantially greater risk of developing permanent acne scars.

DISCUSSION

In the present study, the majority of patients belonged to the 16–20 years age group (41.3%), followed by the 21–25 years age group (29.3%). This finding is in agreement with the well-established epidemiology of acne vulgaris, which predominantly affects adolescents and young adults due to hormonal changes during puberty and increased sebaceous gland activity. Adityan et al,[10] reported that the majority of acne patients were between 15 and 25 years of age, which is comparable to the findings of the present study. Similarly, Kelhalaya et al,[11] observed that adolescents and young adults constituted the largest proportion of patients seeking treatment for acne vulgaris. Goulden et al,[12] also found that acne was most prevalent during late adolescence and early adulthood, supporting the age distribution observed in the present study. The predominance of younger age groups in the current study may be attributed to androgen-mediated stimulation of sebaceous glands, increased sebum production, and follicular keratinization that characterize puberty and early adulthood.

The present study demonstrated a slight female predominance, with females accounting for 57.3% of cases and males accounting for 42.7%. Similar findings were reported by Kelhalaya et al,[11] who observed a higher proportion of female patients among individuals presenting with acne vulgaris. This trend may be explained by greater cosmetic concern, increased awareness regarding skin health, and a higher tendency among females to seek dermatological consultation. Ghosh et al,[13] also reported female predominance in their study population, which was consistent with the findings of the present study. Likewise, Poli et al,[14] observed that women were more likely to seek medical attention for acne, particularly in the post-adolescent age group. In contrast, Adityan et al,[10] reported a slight male predominance among acne patients, while Layton et al,[155] found that severe inflammatory acne was more common among males due to greater androgenic stimulation of sebaceous glands. Despite these variations, the female predominance observed in the present study is comparable with findings reported in most contemporary hospital-based studies.

In the present study, follicular hyperkeratinization was the most common histopathological finding, observed in 90.7% of cases, followed by sebaceous gland hypertrophy (80.0%), perifollicular inflammation (76.0%), lymphocytic infiltrates (72.0%), neutrophilic infiltrates (50.7%), and follicular rupture (34.7%). Fibrosis or scarring and foreign body giant cell reaction were observed in 24.0% and 14.7% of cases, respectively. [Table 7]

The predominance of follicular hyperkeratinization observed in the present study is consistent with the fundamental pathogenic mechanism of acne vulgaris. Plewig and Kligman et al,[16] described follicular hyperkeratinization as the earliest histopathological event leading to microcomedone formation and subsequent acne lesion development. Similarly, Cunliffe et al,[17] reported follicular plugging and abnormal keratinization as characteristic findings in the majority of acne lesions. Sebaceous gland hypertrophy was observed in 80.0% of cases in the present study. This finding is comparable to the observations of Holland et al,[18] who demonstrated increased sebaceous gland activity and enlargement in acne patients secondary to androgen-mediated stimulation. Increased sebum production is recognized as one of the principal factors contributing to acne pathogenesis. Perifollicular inflammation was noted in 76.0% of cases, highlighting the significant role of inflammation in acne development. Jeremy et al,[19] reported that inflammatory changes are present even in clinically non-inflammatory lesions, suggesting that inflammation is an early event in acne pathogenesis rather than merely a secondary phenomenon. Likewise, Dréno et al,[20] emphasized the central role of inflammatory mediators in initiating and perpetuating acne lesions.

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Lymphocytic infiltrates were present in 72.0% of cases, while neutrophilic infiltrates were identified in 50.7%. Similar findings were reported by Layton et al,[21] who observed a predominance of lymphocytic and neutrophilic inflammatory cells surrounding affected follicles, particularly in papulopustular and nodular lesions. These inflammatory infiltrates contribute to tissue destruction and progression of lesion severity.

Follicular rupture was identified in 34.7% of patients in the present study. This finding is comparable with the observations of Kligman et al,[22] who described follicular wall rupture as a crucial event leading to the release of follicular contents into the dermis and triggering intense inflammatory responses. The frequency of follicular rupture was observed to increase with increasing clinical severity of acne.

Fibrosis and scarring were observed in 24.0% of cases, while foreign body giant cell reaction was noted in 14.7%. Similar observations were reported by Goodman et al,[23] who documented that persistent inflammation and follicular destruction lead to dermal fibrosis and scar formation. Foreign body giant cell reactions have also been described in severe inflammatory acne lesions due to the dermal response to extruded keratin and sebaceous material.

Overall, the histopathological findings observed in the present study are in agreement with previous studies and support the concept that follicular hyperkeratinization, sebaceous gland hypertrophy, inflammation, and follicular rupture represent key pathological events in the development and progression of acne vulgaris.

CONCLUSION

The present study highlights that acne vulgaris predominantly affects adolescents and young adults, with a slight female predominance. Grade II acne was the most common clinical presentation, and the face was the most frequently involved site. Comedones and papules constituted the predominant lesion types observed among the study participants.

Histopathological evaluation revealed follicular hyperkeratinization, sebaceous gland hypertrophy, and perifollicular inflammation as the most common microscopic findings, emphasizing their central role in the pathogenesis of acne vulgaris. Furthermore, the severity of histopathological changes increased with advancing clinical grade of acne.

A statistically significant association was observed between acne severity and perifollicular inflammation, follicular rupture, and scarring. Severe grades of acne were characterized by a higher frequency of follicular rupture, intense inflammatory infiltrates, and permanent scar formation, indicating progressive tissue damage with increasing disease severity.

The findings of this study demonstrate a strong clinicopathological correlation in acne vulgaris and reinforce the importance of early diagnosis and timely therapeutic intervention to prevent disease progression and long-term sequelae such as scarring. Histopathological examination provides valuable insights into the underlying pathological processes and may serve as a useful adjunct in understanding disease severity and progression.

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