A Comparative Study between Adolescent Acne and Post-adolescent Acne

Sook-Kyoung Kang, M.D., Min-Sun Jee, M.D., Se-Jin Ahn*, M.D., Jee-Ho Choi, M.D., Kyung-Jeh Sung, M.D., Kee-Chan Moon, M.D., and Jai-Kyoung Koh, M.D.

Department of Dermatology, Asan Medical Center, College of Medicine, University of Ulsan, S & U Clinic, Seoul, Korea*

Background **: The prevalence of patients over the age of 25 years with acne has significantly increased over the past 10 years. Several etiologic factors such as genetic factor, hyperandrogenism, stress, and cosmetics have been studied.**

Objective **: The purpose of this study is to evaluate the differences between adolescent acne and post-adolescent acne, based on the clinical features, associated diseases and aggravating factors of 327 acne patients.**

Methods **: 327 consecutive patients, first visited to our hospital with clinical facial acne between March 2000 and February 2001, were enrolled. 220 adolescent patients(AP) were under the age of 25 and post-adolescent patients(PP) were over the age of 25.**

Results **: 1. In the face, cheeks and forehead in adolescents, chin and cheeks in post-adolescents were predilection sites. The predominant type was a comedonal type in adolescents and a papulopustular type in post-adolescents($p<0.05$). Severity of inflammatory type was not different between two groups. Familial factors were important in both groups, but more frequently found in post-adolescents with persistent acne($p<0.05$). 2. Rosacea, chloasma, and hyperandrogenic features such as hirsutism and androgenetic alopecia were more frequently observed in post-adolescents. Seborrheic and atopic dermatitis were more frequent in adolescents. Four patients had all the features of chloasma, acne, rosacea, seborrheic dermatitis and hirsutism. 3. Aggravation by season especially summer, sweating, stress, and cosmetics was more frequent in adolescents. In PP, acne was more frequently aggravated by sunlight, foods and menstrual period.**

Conclusion **: Post-adolescent acne was mainly a papulopustular type predominantly located on the chin and cheeks with mild to moderate severity, and family history and hyperandrogenism were strong etiologic factors in persistent acne. Aggravating factors were not significantly different between two groups, which suggested cosmetics and stress might not be important in post-adolescent acne. (Ann Dermatol 14(3) 131~136, 2002).**

Key Words : Adolescent acne, Post-adolescent acne, Comparative study

Received October 25, 2001.
Accepted for publication March 25, 2002.
Reprint request to : Kyung-Jeh Sung, M.D., Department of Dermatology, Asan Medical Center, College of Medicine, University of Ulsan, Seoul, Korea
Tel. (02)3010-3460, Fax. (02)486-7831
E-mail: derm@www.amc.seoul.kr

Acne is generally considered a disorder of adolescence. After peaking during the teenage years, the prevalence of acne progressively decreases, affecting approximately 8% of adults aged 25 to 34 years and only 3% of adults aged 35 to 44 years[1]. Over the last few years we have seen an increasing number of patients aged 18 years and over with acne. Men are affected more frequently and severely than women in the teenage years. However, women

may continue to experience acne well into their adult life, up to and even beyond age 40 years. Previous studies showed that its clinical features were different from those of adolescent acne in many aspects and numerous causalities including stress, cosmetics, genetic factors and possible underlying endocrine abnormalities have been proposed to explain post-adolescent acne[2,3].

This study was performed to evaluate the differences between adolescent acne and post-adolescent acne, based on the clinical features, associated diseases and aggravating factors of 327 acne patients.

MATERIALS AND METHODS

1. Patients

A prospective study of 327 consecutive patients, first visited to our hospital with clinical facial acne between March 2000 and February 2001, was undertaken and the patients who had been treated for the last 3 months before study entry were excluded. The patients under the age of 25 were classified as adolescent acne and the patients over the age of 25 as post-adolescent acne. Post-adolescent acne was divided into persistent and late-onset acne by its onset.

2. Clinical examination

The type, severity and involved site of acne, and scarring were evaluated by three designated dermatologists. Acne grading was assessed by the use of a pattern-diagnosis system proposed by the consensus conference in 1991[4](Table 1). We examined whether these patients suffered from other diseases such as seborrheic dermatitis, chloasma, rosacea, atopic dermatitis, hirsutism and androgenetic alopecia. In woman, hirsutism was diagnosed if the Ferriman-Gallwey[5] score was above 8 and androgenetic alopecia was assessed using either the Hamilton or Ludwig grades[6]. A self-administered questionnaire was given to the patients and included age, sex, menstrual history, family history and aggravating factors(season, stress, foods, cosmetics, and obstetric and gynecological history). Menstrual disturbance was defined as amenorrhea of greater than 3 months duration, or irregularity of the menstrual cycle of greater than 7 days from a standard 28-day cycle, over three consecutive cycles.

3. Statistical analysis

The Student's t-test and χ^2 test were employed to compare the clinical characteristics of the two populations. A probability level of <0.05 was considered significant. All p-values are two-sided.

RESULTS

1. Demographic distribution

In total, 228 women and 99 men were evaluated. 220 patients(67.4%) had adolescent acne and 107 patients(32.6%) had post-adolescent acne. AP consisted of 135 women(61.4%) and 85 men(38.6%), and PP consisted of 93 women(86.9%) and 14 men(13.1%)(Table 2). Their mean age was 19.7 ± 3.2 years and 32.1 ± 6.7 years respectively. In post-adolescent acne, persistent acne was documented in 62 patients(57.9%) and true late-onset was in 45 patients(42.1%).

2. The clinical features

The face alone was involved in 126(57.3%) of AP and 65(61.3%) of PP. The back(32.7%), chest(24.5%) and neck(12.3%) in AP, and the back(31.8%), neck(22.4%) and chest(20.6%) in PP were also involved in order of frequency. The most severely involved areas in the face are the cheeks(40.4%), forehead(34.5%), the whole face(20.9%), perioral area(4.1%) and jawline(0.5%) in AP, and the perioral area(38.3%), cheeks(37.4%), forehead(25.7%), the whole face(12.1%) and jawline(4.7%) in PP in order of frequency.

The type of acne lesion was also assessed. Predominantly inflammatory lesions such as papulo-

Table 1. Severity grading of inflammatory acne lesions

Severity	Papules/pustules	Nodules
Mild	Few to several	None
Moderate	Several to many	Few to several
Severe	Numerous and/or extensive	Many

Table 2. Demographics of age and sex of acne patients

Age (years)	Male (%)	Female (%)	Total (%)
Adolescents	85(85.9)	135(59.2)	220 (67.4)
≤(11	0(0)	2(0.9)	2(0.6)
12-17	28(28.3)	25(11)	53(16.2)
18-24	57(57.6)	108(47.4)	165(50.5)
Post-adolescents	14(14.1)	93(40.8)	106 (32.6)
25-34	12(12.1)	71(31.1)	83(25.4)
35-44	1(1)	19(8.3)	20(6.1)
≤45	1(1)	3(1.3)	4(1.2)
Total	99 (100)	228 (100)	327 (100)

Table 3. Grade of inflammatory types

	Adolescents(%)	Post-adolescents(%)	Total(%)
Mild	69(31.4)	43(40.2)	112(34.3)
Moderat	132(60)	55(51.4)	187(57.2)
Severe	19(8.6)	9(8.4)	28(8.6)

Table 4. Associated diseases in the female patients

Associated diseases	Adolescents(%)	Post-adolescents		
		Persistent(%)	Late-onset(%)	Total(%)
Menstrual disturbances	53(40.5)	22(44)	11(27.5)	33(35.2)
Androgenetic alopecia	2(1.5)	2(4)	5(11.9)	7(7.5)
Hirsutism	8(5.9)	9(18)	7(16.7)	16(17.4)
Rosace	12(8.9)	6(12)	5(11.9)	11(12)
Chloasma	6(4.4)	11(22)	7(16.7)	18(19.6)
Seborrheic dermatitis	22(16.3)	6(12)	5(11.9)	11(11.8)
Atopic dermatitis	8(5.9)	2(4)	2(4.8)	4(4.3)

pustules and nodulocysts were present in 100 (45.5%) and 5 (2.3%) of AP, and 75 (70.1%) and 2 (1.9%) of PP respectively, while 115 (52.3%) of AP and 30 (28%) of PP had principally non-inflamed lesions. For the inflammatory lesions, most patients had mild or moderate severity in both groups(table 3). Scarring was recorded in 159 (72.3%) of AP and 71 (66.4%) of PP.

The family history revealed that 161 (73.2%) of AP and 58 (54.3%) of PP had at least one first-degree relative with acne.

3. Associated diseases

55(42%) of adolescent women and 42(46.2%) of post-adolescent women had at least one feature of possible hyperandrogenism, as characterized by hirsutism, androgenetic alopecia or menstrual disturbance. Hyperandrogenism was observed in 26(52%) of persistent type and 17(41.5%) of late-onset type in post-adolescent acne. In adult women, menstrual disturbance was 44% of patients with persistent acne and in 27.5 % of patients with late-onset acne. Hirsutism was present in 18% of persistent acne sufferers and in 16.7% of patients with late-onset acne. Androgenetic alopecia was recorded in 4% of patients with persistent acne and in 11.9% of patients with late-onset acne. The prevalences of rosacea(8.9% vs. 12 %) and chloasma(4.4% vs. 19.6%) were higher in PP

than in AP. The prevalences of seborrheic dermatitis(16.3% vs. 11.8%) and atopic dermatitis(5.9% vs. 4.3%) were higher in AP than in PP(Table 4). Characteristically four persistent PP had all the features of hirsutism, rosacea, chloasma, and seborrheic dermatitis as well as acne.

4. Aggravating factors

66(30%) of AP and 21(19.8%) of PP experienced an aggravation of their acne in summer. In AP, acne was aggravated in summer(77.6%), winter(14.1%), spring(4.7%) or autumn(3.6%) in order of frequency, and PP experienced an aggravation in summer(65.9%), spring(21.9%), winter(9.4%), or autumn(3.1%) in order of frequency. Excessive sweating aggravated acne in 105(47.7%) of AP and 36(33.6%) of PP. Exposure to sunlight was an aggravating factor in 49(22.3%) of AP and 28(26.2%) of PP.

141(64%) of AP and 63(59.4%) of PP experienced that their acne flared during periods of increased physical and psychological stress. 72(53.7%) of AP and 45(54.4%) of PP experienced premenstrual acne flare. Pregnancy resulted in no effect in 12.1%, a flare in 21.2% and an improvement in 12.1% of 36 patients who experienced pregnancy. Acne lesions were aggravated by cosmetics in 30(13.6%) of AP and 14(13.2%) of PP. 60(27.3%) of AP and 34(32.1%) of PP experienced an aggravation of acne by smoking, alcohol or several foods such as meat, chocolate and fatty foods.

DISCUSSION

Acne is principally a disease of adolescence. The prevalence of facial acne in 16-18-year-olds ranges from 81 to 95% in boys and 79 to 82% for girls[7]. In most individuals acne will resolve in early adulthood; however, the condition persists into adult life in a minority of the population especially women. Goulden et al[8]. showed that clinical facial acne was present in 16% of women and 6% of men aged 25-34. The prevalence did not significantly decline between 35-44 years but falls steeply after the age of 45 with only 2% of women and 1% of men being affected. In our study, post-adolescent acne was more frequently observed in women(40.8%) than in men(14.1%) and only a few patients were affected over the age of 45.

Acne is generally limited to the parts of the body that have the largest and most abundant sebaceous glands-the face, neck, chest, upper back, and upper arms. Acne begins on the forehead and cheeks in teenagers but lesions of adult acne are located most commonly around the mouth, chin, and jawline[1,2,9]. Especially, post-adolescent acne appears first in two triangles delineated by the angle of the mouth, the crease down to the jaw line and the lateral point of the chin, which are richly supplied by mental artery and are possibly areas of high androgen sensitivity[9]. Soon the condition spreads to involve the whole perioral areas. In our study, predominantly facial acne was present in 57.3% of AP, compared with 61.3% of PP, which is not significantly different to each other. The second frequent location was the back in both groups. In the face, the cheeks(37.4%) and forehead(25.7%) are the most common sites in AP while the perioral area(38.3%) and cheeks(37.4%) are the predominant sites in PP, which is consistent with the previous reports[9]. Our study confirms that the most predominant type of post-adolescent acne is a papulopustular type with mild to moderate severity and scarring is common[10]. The comedonal type was significantly more frequent in adolescence than in adulthood. There was no significant difference in severity of the inflammatory type between two groups.

There has previously been some evidence that acne is an inherited disease. Goulden et al[11]. showed an odds ratio of 3.93 when comparing the occurrence of adult acne in first-degree relatives of patients with acne compared with those of non-affected individuals and demonstrated the possibility of a familial predisposition to persistent post-adolescent acne. In most people, acne-prone follicles undergo evolution to acne-resistant follicles in early adulthood. However, genetic make-up may determine the failure of acne-prone follicles to evolve into acne-resistant follicles in early adult life[11]. Over the half of our PP had a family history of adult acne and significantly more patients with persistent acne(62%) had family history than patients with late-onset acne(38.1%)($p<0.05$), which suggests that familial factors are important in determining individual susceptibility to persistent type of post-adolescent acne as well as adolescent acne.

Numerous causalities have been proposed to explain post-adolescent acne, including hormonal factors, genetic susceptibility, stress, cosmetics and a

difference in *Propionibacterium acnes* among post-adolescents[1,2,10-12]. The sebum excretion rates of post-adolescent women with persistent acne have been shown to be significantly higher than those of normal controls and a local abnormality of androgen metabolism, in particular the conversion of dihydrotestosterone to testosterone by type I alpha-reductase in acne-prone follicles is likely to be important in post-adolescent acne[1,10,11,13]. This may lead to abnormal follicular keratinization as well as increased sebum production. However, enhanced androgen production does not have an effect on the severity of acne[3]. In our study, 46.2% of PP and 42% of AP had at least one symptom of possible hyperandrogenism such as hirsutism, androgenetic alopecia and menstrual disturbance. While hyperandrogenism in AP was due to menstrual disturbance, hirsutism and androgenetic alopecia were significantly more frequent in PP($p<0.05$). Furthermore, it has been suggested that post-adolescent acne without evidence of alopecia, hirsutism, or menstrual disturbance is a sign of hyperandrogenism and PP may represent a sub-group who have underlying ovarian, adrenal or local androgen abnormalities[10,14]. Griffiths[9] described that the combination of small pinhead acne, rosacea and sensitivity, and mild seborrheic scaling was the most commonly encountered among the red-faced women and this condition called an overlap of androgen-dominant symptoms or MARSH syndrome indicating Melasma, Acne, Rosacea, Seborrheic eczema and Hirsutism. In our study, hyperandrogenic features were evident in PP. The relation with post-adolescent acne, rosacea, chloasma and seborrheic dermatitis was neither prominent nor was the prevalence of associated diseases significantly different between persistent acne patients and late-onset acne patients. Because chloasma occurs probably due to estrogen that induces an inhibition of sebum production[15], the low prevalence of chloasma in PP can be explained. Although it is notable that four patients had those features of MARSH syndrome, the further investigation as to whether MARSH syndrome really exists is needed.

Acne vulgaris is generally known to improve in summertime. Sunbathing is thought to promote the resorption of inflammatory lesions by increased blood flow and ultraviolet A irradiation has been suggested to reduce the sebum excretion rate[16]. In contrast to this, our study revealed more than half of patients experienced an aggravation in summer. UVB-radiation was found to enhance the comedogenicity of squalene and thickening of stratum corneum, the UV-callus, which intensifies occlusion of follicular infundibuli[17].

Some authors state 60-70% of females have premenstrual worsening of their acne[15,18]. The pilosebaceous duct orifice is the smallest at the 15th to 20th day of the cycle, and the acne deteriorates 3-4 days thereafter[18]. In our study, one half of females experienced premenstrual flare and there was no significant difference between AP and PP. In two thirds of patients, acne flared by physical and psychological stress. There is circumstantial evidence that persistent stress activates the pituitary-adrenal axis, resulting in increased secretion of androgenic adrenal steroids[2]. Some authors characterized that post-adolescent acne was quite common in hard-working professional women, and stress and cosmetics could perpetuate acne[2,10] while no significant difference between AP and PP was observed in our study. The old-fashioned "acne diet" stressed consumption of fruits, vegetables, and wholegrain cereals and discouraged consumption of sweets, fried and fatty foods, and many dairy products[19]. One third of our patients considered foods, smoking or alcohol as aggravating factors. Smoking may affect some process in the pathogenesis of acne. Cutaneous blood flow is reduced secondary to inhibition of endothelial prostacyclin synthesis giving a vasoconstrictor effect, while smoking could theoretically have an anti-inflammatory effect on acne[20].

In our study we confirmed that post-adolescent acne is mainly a papulopustular type predominantly located on the chin and cheeks with mild to moderate severity, and family history and hyperandrogenism were strong etiologic factors in persistent acne. Although there were no significantly associated diseases with post-adolescent acne, four patients had the features of MARSH syndrome. We should always exclude these five diseases in red-faced women. Aggravating factors were not significantly different between two groups, which suggested cosmetics and stress might not be important in post-adolescent acne.

REFERENCES

1. White GM. Recent findings in the epidemiologic evidence, classification, and subtypes of acne vulgaris. J Am Acad Dermatol 39:S34-37, 1998.
2. Kligman AM. Postadolescent acne in women. Cutis 48:75-77, 1991.
3. Cibula D, Hill M, Vohradnikova O, Kuzel D, Fanta M, Zivny J. The role of androgens in determining acne severity in adult women. Br J Dermatol 143:399-404, 2000.
4. Pochi PE, Shalita AR, Strauss JS, et al. Report of the consensus conference on acne classification. J Am Acad Dermatol 24:495-500, 1991.
5. Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 21:1440-1447, 1961.
6. Venning VA, Dawber R. Patterned androgenetic alopecia. J Am Acad Dermatol 18:1073-1078, 1988.
7. Rademaker M, Garioch JJ, Simpson NB. Acne in school children: no longer a cause for dermatologists. Br Med J 108:287-289, 1989.
8. Goulden V, Stables GI, Cunliff WJ. Post-adolescence of facial acne in adults. Br J Dermatol 137(suppl. 50):40 (Abstr.), 1997.
9. Griffiths WAD. The red face-an overview and delineation of the MARSH syndrome. Clin Exp Dermatol 24:42-47, 1999.
10. Goulden V, Clark SM, Cunliffe WJ. Post-adolescent acne: a review of clinical features. Br J Dermatol 136:66-70, 1997.
11. Goulden V, Mcgeown CH, Cunliffe WJ. The familial risk of adult acne: a comparison between first-degree relatives of affected and unaffected individuals. Br J Dermatol 141:297-300, 1999.
12. Seukeran DC, Cunliffe WJ. Acne vulgaris in the elderly: the response to low-dose isotretinoin. Br J Dermatol 139:99-101, 1998.
13. Thiboutot D, Gilliland K, Light J, Lookingbill D. Androgen metabolism in sebaceous glands from subjects with and without acne. Arch Dermatol 135:1041-1045, 1999.
14. Vexiau P, Husson C, Chivot M, et al. Androgen excess in women with acne alone compared with women with acne and/or hirsutism. J Invest Dermatol 94:279-283, 1990.
15. Fisher DA. Desideratum dermatologicum-cause and control of premenstrual acne flare. Int J Dermatol 39:334-336, 2000.
16. Gfesser M, Worret WI. Seasonal variations in the severity of acne vulgaris. Int J Dermatol 35:116-117, 1996.
17. Mills OH, Porte M, Kligman AM. Enhancemant of comedogenic substances by ultraviolet radiation. Br J Dermatol 98:145-150, 1978.
18. Williams M, Cunliffe WJ. Explanation of premenstrual acne. Lancet 2:1056-1057, 1973.
19. Rosenberg EW. Acne diet reconsidered. Arch Dermatol 117:193-195, 1981.
20. Mills CM, Peters TJ, Finlay AY. Does smoking influence acne? Clin Exp Dermatol 18:100-101, 1993.