Correspondence

‘Smoker’s acne’: a new clinical entity?

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Sir, Although postpubertal acne is commonly described as inflammatory, mild-to-moderate and predominantly located on the mandibular region,[1,2] in our clinical practice we observed a form characterized predominantly by micro- and macrocomedones, with few inflammatory lesions, which seemed to affect particularly adult female smokers, that we have conventionally named noninflammatory acne (NIA).

In order to verify the frequency of NIA among women and to examine possible correlations with cigarette smoking, 1000 women aged 25–50 years were randomly enrolled from mothers or other women accompanying children attending our Paediatric Dermatology outpatient clinic (January–May 2006). These women were questioned about their smoking habit, number of cigarettes smoked, juvenile acne (JA) and possible hormonal imbalances. Participants were evaluated for acne and clinical signs of hyperandrogenism.

Subjects with acne who had hormonal imbalances were excluded. Acne was classified as inflammatory (IA; predominantly inflammatory lesions mainly located on jaws, mandible and neck) or NIA (prominent whiteheads, blackheads and microcysts on the malar region and the forehead, inflammatory lesions scarce or absent; Fig. 1).

Among 1000 subjects, 27·7% were smokers and 72·3% nonsmokers. Mean age was 34·6 years (median 36) and did not differ significantly between smokers and nonsmokers, subjects with acne and healthy individuals, IA and NIA. The prevalence of acne was 18·5%, slightly higher than previously reported (12–14%).[1,2]

As shown in Table 1, acne was strikingly more frequent among smokers (115 of 277; 41·5%) than nonsmokers (70 of 723; 9·7%) ($P < 0·0001$). NIA affected 138 (74·6%) of 185 subjects with acne; 105 of 138 (76%) were smokers and 11 of 138 (8%) had a severe form. Of these, nine were smokers (> 15 cigarettes daily).

NIA was the most frequent form of acne among smokers (105 of 115; 91·3%); IA prevailed among nonsmokers (37 of 70, 52·8%) and was always mild-to-moderate in severity ($P = 0·0003$). Predisposing environmental factors were identified in 16 nonsmokers with NIA (two with a severe form).

We found no statistical differences in mean cumulative smoking dose (CSD) (number of cigarettes smoked daily × 365 × number of years) between smokers with (70 876; range 4380–233 600; median 63 802) and without (65 364; range 3285–219 000; median 59 422) acne ($P > 0·05$).

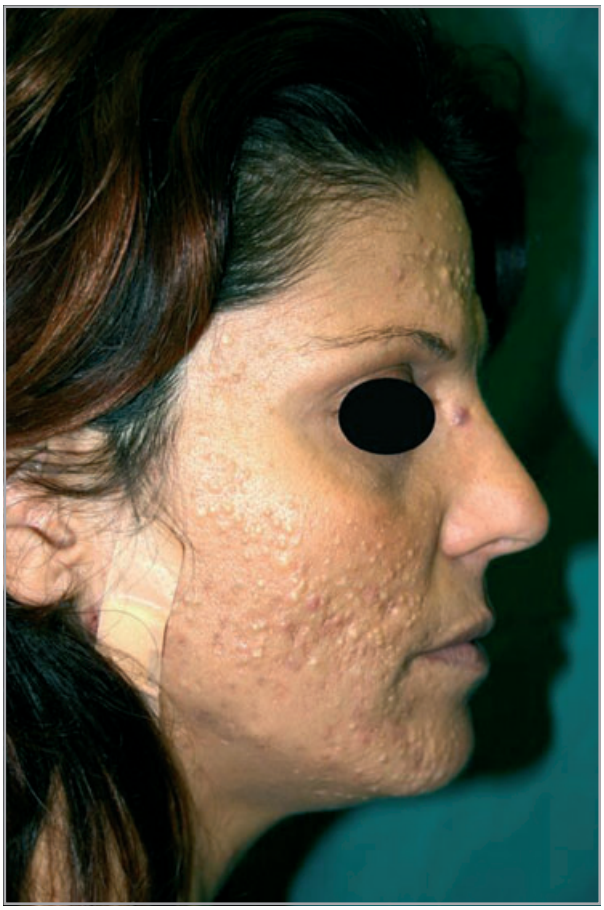

Fig 1. Clinical appearance of noninflammatory acne.

Table 1 Characteristics of the sample

	Smokers	Nonsmokers	Total
No. subjects	277	723	1000
Acne[a]	115	70	185
Noninflammatory acne	105	33	138
Mild-to-moderate	96	31	127
Severe	9	2	11
Inflammatory acne	10	37	47
Healthy	162	653	815

[a]Odds ratio 6·622; 95% confidence interval 4·7–9·3.

Only 35 of 115 of smokers with acne reported the appearance of acne 1–2 years after starting smoking, a finding considered unreliable because of recall bias. Among subjects who had JA (42%), 47% of the smokers had adult acne, as opposed to 18% of the nonsmokers. Smokers with JA had a probability to be affected by current acne that was four times higher than in nonsmokers ($P = 0{\cdot}0042$; odds ratio 4·05; 95% confidence interval 2·6–6·3) which suggests that smoking could be a major contributing factor for adult acne in predisposed subjects.

Epidemiological studies on the correlation between acne and smoking are still controversial.[3–5] Several pieces of experimental evidence support a pathogenetic role of smoking on NIA. Keratinocytes have nicotine acetylcholine receptors, which induce cutaneous hyperkeratinization at high concentration.[6] Nicotine and other components of cigarette smoke induce vasoconstriction and hypoxaemia and have an anti-inflammatory effect on neutrophil and lymphocyte chemotaxis.[7]

Moreover, smoking seems to cause an increase in oxidative stress and reduces the levels of plasma α-tocopherol. We evaluated the levels of squalene, squalene monohydroperoxide and α-tocopherol in sebum [collected from the forehead using Sebutape® (Cuderm, Dallas, TX, U.S.A.)] of 20 smokers with acne (AS), 20 healthy nonsmokers (CTR) and 20 smokers without acne (CTR-S). AS and CTR-S had smoked eight to 14 cigarettes daily for more than 5 years.

Among smokers, sebum vitamin E levels were halved compared with nonsmokers and were associated with an increase in lipid peroxidation (50% decrease of squalene with a parallel increase in squalene peroxide).

Previous studies demonstrated that patients with acne had a higher grade of lipid peroxidation.[8] Squalene peroxides are comedogenic and have a hyperproliferative effect on keratinocytes.[9] Thus, cigarette smoke, through the production of reactive oxygen species, produces an alteration in sebum composition similar to that found in acne. Sebum excretion in AS was three times higher than in CTR and CTR-S; this, together with the absence of differences in mean age and CSD between smokers with and without acne, suggests that clinical expression of acne could be related to individual susceptibility.

Several factors (hormonal alterations, stress, atmospheric pollution, occupational and environmental factors, photoexposure) can contribute to the pathogenesis of NIA.[10] In particular, photoexposure is a predisposing condition for retentional lesions (micro- and macrocomedones), as in Favre–Racouchot syndrome, and is a widespread habit in our region. This could have influenced our statistics, but, according to some biopsies we performed, young women presented severe acne without elastosis, suggesting a marginal influence of photoexposure on NIA.

Our data demonstrate that NIA affects a high percentage of women, among whom the number of smokers is high, which might partially explain the noticeable increase in this pathology.

Recognizing this form is fundamental for providing correct information about the effects of tobacco on the skin, which could contribute to antismoking information programmes.

In some subjects the severity of acne, the clinical peculiarities, the strong correlation with smoking, and the biochemical data could lead to NIA being considered as a new entity among smoking-related cutaneous diseases ('smoker's acne face').

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References

1 Williams C, Layton AM. Persistent acne in women: implications for the patient and for therapy. *Am J Clin Dermatol* 2006; **7**:281–90.
2 Goulden V, Clark SM, Cunliffe WJ. Post-adolescent acne: a review of clinical features. *Br J Dermatol* 1997; **136**:66–70.
3 Schäfer T, Nienhaus A, Vieluf D *et al.* Epidemiology of acne in the general population: the risk of smoking. *Br J Dermatol* 2001; **145**:100–4.
4 Chuh AA, Zawar V, Wong WC *et al.* The association of smoking and acne in men in Hong Kong and in India: a retrospective case–control study in primary care settings. *Clin Exp Dermatol* 2004; **29**:597–9.
5 Mills CM, Peters TJ, Finlay AY. Does smoking influence acne? *Clin Exp Dermatol* 1993; **18**:100–1.
6 Theilig C, Bernd A, Ramirez-Bosca A *et al.* Reactions of human keratinocytes *in vitro* after application of nicotine. *Skin Pharmacol* 1994; **7**:307–15.
7 Misery L. Nicotine effects on the skin: are they positive or negative? *Exp Dermatol* 2004; **13**:665–70.
8 Zouboulis CC, Nestoris S, Adler YD *et al.* A new concept for acne therapy: a pilot study with zileuton, an oral 5-lipoxygenase inhibitor. *Arch Dermatol* 2003; **139**:668–70.
9 Ottaviani M, Alestas T, Mastrofrancesco A *et al.* Peroxidated squalene induces the production of inflammatory mediators in HaCaT keratinocytes. A possible role in acne vulgaris. *J Invest Dermatol* 2006; **126**:2430–7.
10 Cunliffe WJ, Holland DB, Jeremy A. Comedone formation: etiology, clinical presentation, and treatment. *Clin Dermatol* 2004; **22**:367–74.

Conflicts of interest: none declared.

