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SEVERE LATE-ONSET ACNE IN ADULTS: CLINICAL EVOLUTION UNDER SYSTEMIC THERAPY AND THE ROLE OF A MULTIDISCIPLINARY APPROACH

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ABSTRACT

Acne, one of the most common dermatological conditions, has a multifactorial pathogenesis and variable manifestations, requiring a complex therapeutic approach to prevent scarring and reduce psychosocial impact. This paper presents the case of a 28-year-old female patient with severe inflammatory acne of late onset, resistant to conventional therapies. An individualized systemic protocol, combined with dermato-cosmetic care and photoprotection, led to clinical improvement and a two-month absence of comedones. The patient is undergoing fractional laser therapy for residual scarring. This case underlines the importance of a personalized, multidisciplinary approach integrating medical treatment, psychological support, and aesthetic interventions to improve quality of life.

INTRODUCTION

Acne is the most common dermatological condition, with a major impact on quality of life. It may cause scarring, persistent hyperpigmentation, and psychological sequelae. Pathogenically, it is a chronic inflammatory disease of the pilosebaceous unit, characterized by the formation of microcomedones, precursors of clinical lesions. Beyond its cosmetic aspect, the high visibility of acne increases healthcare-seeking behavior, largely due to its psychological consequences. (Baumann 2002)

Acne vulgaris is typical of puberty, reflecting physiological transformations, but may persist into adulthood (Cristodor 2020). It is a multifactorial condition, with an average course of 8 years, sometimes extending to 12–15 years or even lifelong (Baumann 2002; Cristodor 2020). About 70–80 % of cases occur between ages 11 and 25, while in adults it is more frequent in women, often with hormonal involvement and stronger emotional impact (Baumann 2002). Lesions usually affect the face, shoulders, back, and chest; although both sexes are affected, severe forms are more common in men. In all cases, early and individualized treatment is crucial for optimal cosmetic outcomes (Cristodor 2020).

Acnegenesis and comedogenesis are distinct but interdependent processes: follicular epithelial inflammation leads to pustules and papules, while hyperkeratosis produces comedones (Baumann 2002).

The first key factor is sebaceous gland hyperactivity, strongly stimulated by androgens at puberty; testosterone regulates secretion in men, whereas luteinizing hormone enhances post-ovulatory activity in women, explaining premenstrual flares. Hyperandrogenism in polycystic ovary syndrome further exacerbates acne. Sebum induces inflammation, with patients showing increased secretion and hypertrophic glands (Harris et al. 1983). Antiandrogens, estrogens, and oral retinoids are effective. An inverse relationship between sebum and linoleic acid (Strauss & Thiboutot 1999) leads to essential fatty acid deficiency, impairing epithelial barrier and promoting hyperkeratosis (Downing et al. 1986).

The second factor is abnormal follicular keratinization, where adhesive keratinocytes obstruct the follicle, forming open or closed comedones and triggering inflammation (Baumann 2002).

The third factor is bacterial involvement: although Propionibacterium acnes is common in both affected adolescents and healthy adults, excess sebum and hyperkeratosis favor its proliferation, with inflammation driven by free fatty acids from triglyceride breakdown, further amplified by proteases and hyaluronidases (Strauss & Thiboutot 1999).

Acne manifests through symmetrical eruptions composed of non-inflammatory lesions (closed and open comedones) and inflammatory lesions (papules, pustules, nodules, cysts). Healing is usually complete, although atrophic, hypertrophic, or keloid scars may persist.

Non-inflammatory lesions result from androgen-stimulated sebum secretion and follicular obstruction, leading to whitish-yellow closed comedones or black open comedones caused by melanin oxidation. The term “comedone” originates from Greek, referring to the worm-like appearance of the expelled material.

Inflammatory lesions are driven by the proliferation of *Propionibacterium acnes* on the background of excess sebum, triggering immune responses. Papules develop from closed comedones through vasodilation and local edema; pustules result from leukocyte accumulation; nodules and cysts arise from deeper inflammation, often leaving atrophic, hypertrophic, or keloid scars. In some patients, especially women, self-induced lesions such as excoriated acne may appear, along with eczematization, secondary infections, crusts, or scars (Cristodor 2020).

The treatment of acne aims to control contributing factors. While genetic predisposition and the menstrual cycle cannot be influenced, hyperandrogenism, *P. acnes* colonization, local friction, comedogenic soaps, certain medications, oily cosmetics, occupational exposures, radiation, stress, and high-glycemic diets exacerbate the disease. Non-pharmacological measures include avoiding comedogenic products, androgen-based hormonal therapies, and foods with negative impact, alongside smoking cessation. Cryotherapy and ultraviolet exposure may have adjuvant roles. Medical therapy is tailored to severity: topical agents in mild cases, topical or systemic antibiotics in moderate forms, and oral retinoids in severe acne. In selected cases, surgical treatment includes comedone extraction, cyst drainage, or excision of sinus tracts (Cristodor 2020).

Therapeutic success in acne is based on several major directions. Normalization of keratinization and exfoliation is achieved through retinoids, which prevent keratinocyte adhesion and reduce follicular hyperkeratosis. Tretinoin is effective in eliminating comedones and facilitating antibiotic penetration, being recommended as a first-line therapy (Berson & Shalita 1995). Ultrastructural studies have demonstrated that it weakens follicular impactions and reduces microcomedone cohesion (Lauker et

al. 1992). In nodulocystic forms, oral isotretinoin normalizes keratinization and decreases sebum secretion (Baumann 2002). Its administration requires monitoring of hepatic function and lipid profile, but its efficacy in severe forms is exceptional, despite possible adverse effects and teratogenic risk (Solovan et al. 2011).

Eliminating or reducing *Propionibacterium acnes* represents another essential direction. Erythromycin and clindamycin, with comparable efficacy, are the most commonly used topical antibiotics (Thomas et al. 1982). They also exert anti-inflammatory effects by reducing free fatty acids generated from bacterial lipid digestion (Esterly et al. 1978). A current issue is bacterial resistance, with up to 60% of patients presenting resistant strains. Benzoyl peroxide acts by generating reactive oxygen species (Nacht et al. 1981); however, concomitant application with topical tretinoin may reduce its effectiveness (Martin et al. 1998).

Removal of material blocking the pores is achieved with keratolytic agents such as salicylic acid (BHA) and α-hydroxy acids (AHA), with BHA being more effective in reducing comedones. Additionally, comedone extractions or minor surgical procedures may be performed. Commonly used products include retinoids, salicylic acid, AHAs, and azelaic acid. Control of the inflammatory response is achieved with salicylic acid, NSAIDs, or BHA peels. Administration of ibuprofen combined with antibiotics has been shown to significantly reduce inflammatory lesions (Wong et al. 1984). Steroid injections and hydrocortisone creams can also reduce inflammation, but they carry the risk of steroid-induced acne and cutaneous atrophy.

Reducing sebaceous secretion represents another key therapeutic approach. Oral and topical retinoids inhibit sebaceous gland activity, while in women, oral contraceptives are an effective method of hormonal stabilization and sebum reduction.

An effective acne prevention regimen should address all stages of its development. Morning care includes cleansing with a 2 % salicylic acid cleanser, application of a topical antibiotic or azelaic acid, and the use of an SPF 45 sunscreen adapted to skin type. Evening care involves repeating cleansing and applying a topical retinoid. In resistant cases, salicylic acid peels, antibiotics, oral retinoids, or oral contraceptives may be added. Some foundations contain salicylic acid, providing an adjuvant role (Baumann 2002).

The psychological impact of acne is considerable, particularly in adolescents and young adults, where it can lead to anxiety, depression, and even suicidal ideation. In females, excoriated acne is frequently observed, due to compulsive manipulation of lesions, which worsens scarring and pigmentation disorders. For this reason, treatment should be initiated early and, when necessary, combined with psychological or psychodermatological support (Cristodor 2020).

MATERIAL AND METHODS

The present study reports the case of a 28-year-old female patient diagnosed with severe inflammatory acne with late onset. The patient had no dermatological or endocrine history and had not undergone hormonal treatments. The disease had a sudden onset, with the appearance of open and closed comedones, papules, pustules, and deep nodules, predominantly located in the mandibular and malar regions.

The patient had previously undergone topical therapies with retinoids and antibiotics, as well as cosmetic procedures, without satisfactory clinical response. At the time of evaluation, persistent inflammatory lesions and post-inflammatory atrophic scars were observed.

Medical history revealed significant psychological stress in recent months, correlated with exacerbation of eruptions. This suggests a psychoneuroendocrine influence, confirmed in the literature through activation of the hypothalamic–pituitary–adrenal axis and increased cortisol secretion, with pro-inflammatory and seboregulatory effects.

A systemic therapeutic protocol was initiated, likely including isotretinoin, combined with non-comedogenic dermocosmetic products for cleansing and hydration, as well as daily photoprotection.

RESULTS AND DISCUSSIONS

Under the new therapeutic regimen, the patient showed a progressive improvement of the cutaneous appearance, as documented in Figures 1–9. At baseline, the clinical picture was dominated by severe inflammatory acne lesions, characterized by comedones, papules, pustules, and, at times, nodules (Figures 1–3).

A few weeks after the initiation of the dermato-cosmetic protocol, the patient reported a gradual improvement of the skin appearance (Figure 4). However, periodic recurrences persisted, particularly in stressful contexts or during the luteal phase of the menstrual cycle (Figure 5), suggesting the possible involvement of a subtle hormonal substrate. Consequently, a complete endocrinological evaluation (testosterone, free androgens, DHEA-S) was recommended to exclude polycystic ovary syndrome.

Following the introduction of a new therapeutic protocol, tailored to the severe clinical form and including oral isotretinoin in combination with systemic anti-inflammatory and sebum-regulating agents, the patient's evolution became visibly favorable. At dermatological reassessment, moderate inflammatory acne lesions with comedones, scarring, and pigmentary changes were observed (Figure 6).

After one month of treatment, the skin appearance had improved considerably, with a reduction of inflammation and persistence of only moderate lesions and post-inflammatory sequelae (Figure 7). At two months, the patient presented only mild lesions, accompanied by scarring and pigmentary changes (Figure 8). The favorable outcome was confirmed at five months, when no active inflammatory lesions or comedones were observed, with only residual scars and pigmentary alterations remaining (Figure 9).

Throughout the treatment, the psychological impact of the disease was an important aspect to consider. The patient reported decreased self-esteem, social difficulties, and frustration—issues that are well documented in the literature—which justified the integration of psychological support in the case management.

For the maintenance phase, scar remodeling procedures were proposed, such as chemical peels, microneedling, and fractional laser therapy, aiming to correct residual lesions and improve the patient's quality of life. At the end of systemic therapy, the absence of comedones for two consecutive months confirmed the effectiveness of the adopted protocol. Currently, the patient is about to start a fractional laser therapy program for post-acne scarring.

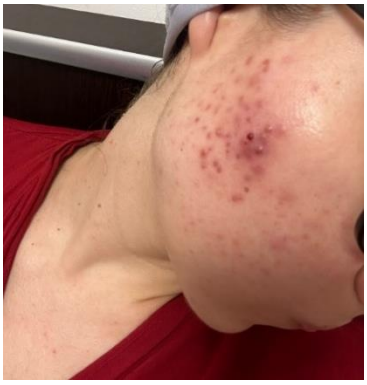

Figure 1. Severe inflammatory acne at onset: comedones, papules, pustules, and nodules

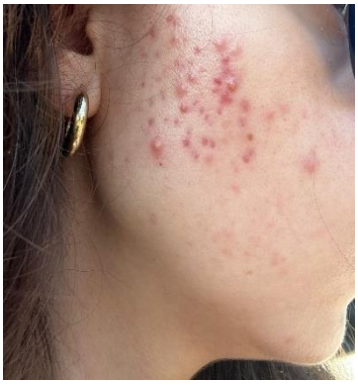

Figure 2. Severe inflammatory acne at onset: comedones, papules, pustules

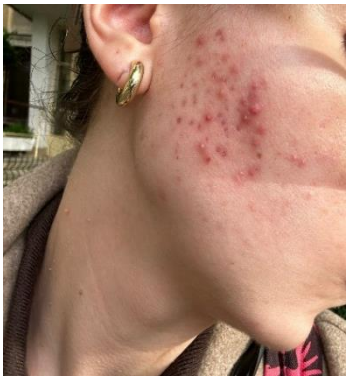

Figure 3. Severe inflammatory acne at initiation of dermato-cosmetic therapy

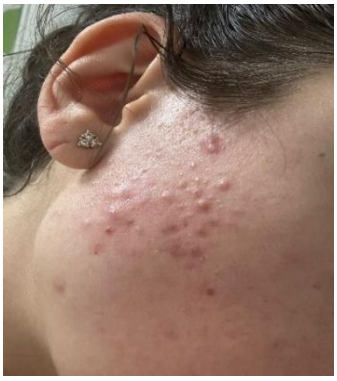

Figure 4. Partial improvement after several weeks; persistent severe lesions with comedones, papules, pustules, and scarring

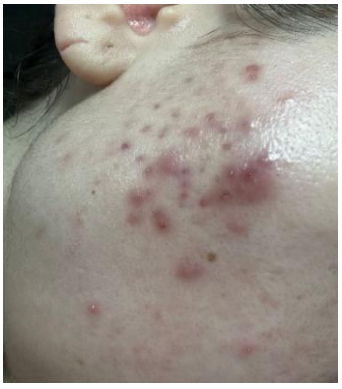

Figure 5. Periodic recurrences under stress or in luteal phase; severe acne with comedones, papules, pustules, and nodules

Figure 6. After new systemic protocol with isotretinoin: progressive improvement; moderate acne with comedones, scars, hyperpigmentation

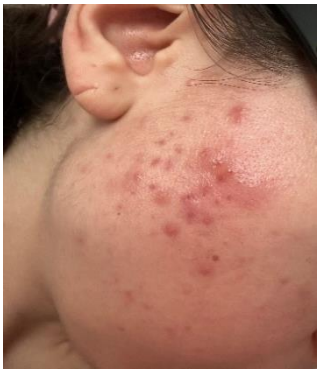

Figure 7. One month after new protocol: marked improvement; moderate acne with comedones, scars, hyperpigmentation

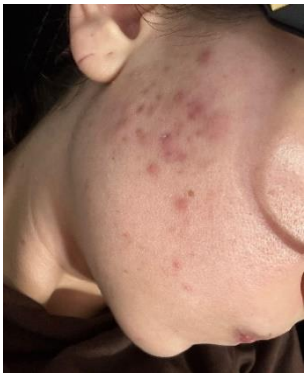

Figure 8. Two months after new protocol: visible improvement; mild acne with comedones, scars, hyperpigmentation

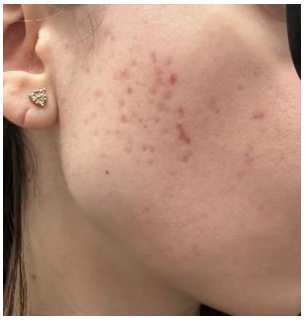

Figure 9. Five months after initiation of the new therapeutic protocol: visible improvement with no inflammatory acne lesions and no comedones for two months, but with residual scars and hyperpigmentation

CONCLUSIONS

Acne is a chronic, multifactorial dermatological condition with a major impact on aesthetics and quality of life. Understanding the pathogenic mechanisms allows for individualized treatment, which should be initiated early and tailored to each patient. A comprehensive approach—combining topical and systemic therapies, hygienic-dietary measures, corrective procedures, and, when needed, psychological support—is essential to reduce both clinical manifestations and psychosocial consequences.

The presented case highlights the complexity of severe late-onset acne in adults, where hormonal, inflammatory, and psychological factors can influence disease evolution. The patient's progressive improvement, absence of comedones for two consecutive months, and the integration of a laser therapy plan for scar correction demonstrate the effectiveness of an integrated strategy. The prognosis is favorable, provided adherence to treatment and careful clinical monitoring.

Optimal management of acne requires a multidisciplinary approach involving the dermatologist, endocrinologist, and psychologist, while early initiation of personalized therapy plays a key role in preventing scarring complications and psychosocial impact.

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