

# Differential diagnoses of acne vulgaris (common acne)

Acne is a chronic inflammatory disease of the pilosebaceous follicle that involves three factors: excessive sebum production, abnormal keratinization of the pilosebaceous follicle and an anaerobic bacterium, *Cutibacterium acnes*. *Cutibacterium acnes*, along with the microbiome and innate immunity, is a key contributor to the development and maintenance of the local inflammatory response.

Only acne involves open and closed comedones. Differential diagnosis with folliculitis is thus based on an absence of comedones in papulopustular lesions. Below we will look at the differential diagnoses of acne involving the face and chest.

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## Folliculitis

### Gram-negative folliculitis

This is a follicular pyoderma, a complication of an overlong antibiotic treatment. Two types of Gram-negative folliculitis have been described:

- - Type I: the papulopustular form, where lesions are found predominantly around the nostrils and mouth.
- - Type II: the nodular form, which resembles acne conglobata. In most cases, bacterial cultures identify *Enterobacter*, *Klebsiella* or *Escherichia coli* for type I and *Proteus* for type II.

## Folliculitis of the back

These conditions present in the form of follicular papules with a gaping pilosebaceous orifice at their center that is sometimes inflamed (pustule), developing in seborrheic skin. There are no comedones. They are more likely to occur in young adults, more often in boys, and get worse in the summer. They can also be seen on the face.

The diagnosis is confirmed by a parasitological test, which in most cases reveals *Malassezia furfur*. More rarely, it will find *Demodex* (lipophilic mite that penetrates the dermis and causes inflammation), with clinical signs of follicular hyperkeratosis and pustules. It may also be bacterial folliculitis.

## Other forms of folliculitis

Eosinophilic pustular folliculitis (Ofuji disease) affects mostly men, with follicular papulopustules that reach the face, back and extensor surfaces of the limbs, accompanied by elevated eosinophils and blood IgE. The pustules are sterile.

Necrotizing lymphocytic folliculitis, or acne necrotica, is characterized by a crusted, pustular, inflammatory follicular lesion with a depressed center that evolves into a varioliform scar. The lesions are located around the periphery of the scalp (forehead, nape of the neck, temporal region) and are painful and often pruritic. Histological examination evidences distal necrosis of the pilosebaceous follicles, associated with a lymphocytic infiltration. This rare condition affects middle-aged women. There is no known etiology or treatment.

## Drug-induced folliculitis

This may involve the maintenance of acne by a medication or the induction of acne-like lesions by the medication. In this case, there are monomorphic or superficial inflammatory lesions (papules, pustules), with no comedones or microcysts. The lesions may be sited on the face, trunk and limbs.

There are many systemic treatments that can induce such lesions: systemic corticosteroids, B vitamins (B12), anticonvulsants, anti-tuberculosis drugs, lithium salts, halogenated compounds (bromides, iodides), etc.

Folliculitis induced by anabolic agents (bodybuilding, high-level competitive sport, etc.) resembles teen acne but is often resistant to treatment. It can be very inflammatory, and even assume the appearance of acne fulminans.

Acne caused by hormonal treatments, such as androgenic progestins taken alone or in combined pills, can induce or aggravate acne.

The pills most to blame are the first generation pills, which contain androgenic progestins that displace testosterone from its carrier protein. New-generation contraceptive pills contain progestins which pose less of a problem, some even declaring a certain level of efficacy in treating acne.

Recent oncology treatments, such as anti-EGFR therapies, appear to induce inflammatory folliculitis, sometimes causing patients to discontinue cancer treatment despite a therapeutic response.

## **Folliculitis associated with topical treatment or local causes**

### **Mineral oil**

The basic lesion is represented by oil folliculitis. These pseudo-acnes are observed following intoxication (poisoning) or occupational exposure to halogenated aromatic hydrocarbons, in particular those that are polychlorinated, such as naphthalenes and polyhalogenated bisphenols.

### **Chloracne**

This pseudo-acne begins with the appearance of open comedones, particularly in the zygomatic, retroauricular and scrotal regions. Then the lesions spread to the back, shoulders, chest and buttocks. The nose is generally spared. The axillary regions are only affected in cases of ingestion or inhalation. When poisoning is prolonged, microcysts, cysts and inflammatory lesions join the pre-existing comedones. In addition, general symptoms (digestive, pulmonary and neurological) may appear. When exposure ceases, the lesions regress over several months (on average 6 months) but can persist and in general cause scarring.

### **Cosmetic acne**

Cosmetic acne is caused by the presence of plant oils or liquid paraffin (Vaseline) in topical products. It is essentially comedonal, with obstruction of the infundibulum of the pilosebaceous follicle and secondary inflammation being key contributors.

### **Acne mechanica**

Repeated rubbing or occlusion can trigger a mechanical type of pseudo-acne (wearing a helmet, backpack, etc.), which is usually comedonal with thickening of the skin.

## Majorca acne, or summer acne

The sun usually improves acne lesions, particularly on the back, by reducing sebum production. But UVA rays also cause a thickening of the stratum corneum, which often causes a recurrence in the form of retentional lesions in the autumn.

## Other differential diagnoses of acne

### Acne excoriata

This is not actually acne but excoriations induced by a patient with a particular psychological predisposition. The acne lesions are combined with lesions caused by scratching and excoriations. This form of acne is maintained by continual handling of lesions. It is illustrative of a psychologically fragile and anxious disposition. The support of a psychiatrist is necessary in most cases.

### Rosacea

Lesions observed in rosacea are papulopustular, but there are no comedones or microcysts. The lesions are localized particularly on the cheeks and nose (centrofacial). Rosacea occurs at a later age (forties) and in individuals with a fair-skinned phototype.

### Perioral dermatitis

Lesions are exclusively inflammatory and are localized around the mouth. They are seen particularly in women and in most cases are linked to the misuse of cosmetics or the repeated use of topical corticosteroids.

### Papular sarcoidosis

The eruption is monomorphic and comprised of small papules localized on the face, distinguished by their orange or brownish-red color. They are sometimes translucent.

## Acne nevus

This corresponds to the appearance of acne lesions in a circumscribed area of the trunk and belongs to the group of functional skin nevi (Becker nevus, nevus anemicus, etc.).