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Acne mimickers: Differential diagnosis of open comedones: A short review

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ABSTRACT

Acne vulgaris is one of the most common disorders of the pilosebaceous unit, affecting around 9.4% of the global population. The principal or preliminary lesions of acne are comedones, which represent the follicular dilation and hypercornification secondary to androgen hypersensitivity. Although the diagnosis of acne is rarely difficult for an experienced dermatologist, we rarely come across various other dermatoses that clinically resemble comedones yet have a different pathophysiology and poorly respond to treatment. Not all disorders that have comedone-like lesions are acne. These disorders should be kept in mind when comedones occur in an unusual site, age, or pattern. This review article mainly focuses and highlights the salient features of multiple non-acne disorders that present with open comedone-like lesions along with an approach to the diagnosis and its management.

Key words: Open comedones; Acne; Keratin plugs; Black comedones

INTRODUCTION

Acne vulgaris is a disorder of the pilosebaceous unit, affecting around 9.4% of the global population, involving 90% of males and 80% of females in all ethnic groups [1]. The principal lesions are comedones, which are non-inflamed, primitive, and pathognomic lesions of acne. They represent follicular hyperkeratinization and dilatation that progress to form other lesions, such as papules, pustules, nodules, and cysts [2,3]. Various other disorders have similar presentations mimicking acne, which is briefly discussed in this article. Table 1 highlights the various types of comedones. Common disorders resembling open comedones include familial dyskeratotic comedones, nevus comedonicus, Favre–Racouchot syndrome, the dilated pore of Winer, and trichostasis spinulosa.

Familial Dyskeratotic Comedones (FDC)

Familial dyskeratotic comedones is a rare autosomal dominant disorder with no racial or sexual predisposition. It presents as asymptomatic, numerous, discrete, disseminated, symmetrical, crater-like depressions filled with keratin, resembling open comedones. When the keratin plugs are removed, they reveal a crater with minimal bleeding that heals with pock-like scars. The most common sites of predilection are the face and neck. However, these may also be generalized or diffuse. They are asymptomatic unless secondarily infected. On secondary infection, they form cysts and abscesses. The mucosa, palms, and soles are spared [4]. The three classical features of familial dyskeratotic comedones (FDC) are the presence of multiple, disseminate comedones, a family history of FDC, and histopathology showing dyskeratosis. The management of FDC is difficult as it has no tendency to regress and no response is satisfactory. However, topical and oral retinoids, CO_2 laser, and UVA may be attempted [5].

Nevus Comedonicus

This is a follicular keratotic nevus regarded as a hamartoma of the pilosebaceous unit. It is a developmental defect in which the follicle is unable to form a hair or sebaceous gland. It forms only keratin plugs. They are not true comedones.

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Table 1: The various types of comedones

No.	Comedones	Clinical features
1.	Open comedones	They are black papules, which represent dilated follicular openings < 1 mm. The black color is caused by the oxidation of melanin.
2.	Closed comedones	They are skin-colored, dome-shaped papules without a visible follicular opening, which is < 1 mm in diameter.
3.	Microcomedone	They are microscopic comedones invisible to the naked eyes. They represent the microscopic events such as follicular blockage with follicular hyperkeratosis and sebum secondary to androgen hypersensitivity.
4.	Macrocomedone	They are large comedones greater than 1 mm in diameter.
5.	Submarine comedones	These are deeper comedones, which are > 0.5 cm in diameter, better visualized when the skin is stretched. They are prone to forming inflammatory nodules and, hence, scarring. They are slightly resistant to treatment. Intralesional steroids and isotretinoin help in the management.
6.	Sandpaper comedones	Sandpaper comedones represent multiple closed comedones usually seen on the forehead and the T-area of the face. They are common in mid-childhood acne. It gives a roughed and gritty feel when touched. They are usually resistant to treatment, yet peels and oral and topical retinoids may help.
7.	Double-edged comedones	They are pseudo-comedones seen in hidradenitis suppurativa. They are dilated, keratin-filled, interconnected pores presenting as open comedones joined by a shallow tunnel.
8.	Polyporous comedones	They are comedones with multiple pores (keratin-filled), which is common in hidradenitis suppurativa.
9.	Missed comedones	They are comedones usually missed with normal examination, yet when the skin is stretched in the presence of good lighting, they become evident.
10.	Secondary comedones	They are secondary to inflammation or irritation, for instance, comedones secondary to waxing, chloracne, pomade acne.
11.	Solar comedones	They are comedones found on the cheeks and chin in elderly patients with chronic photodamage usually located on the lateral aspect of the periorbital region [Figure 1].

Figure 1: Solar comedones on the left cheek of a farmer with photoaging secondary to excessive sun exposure.

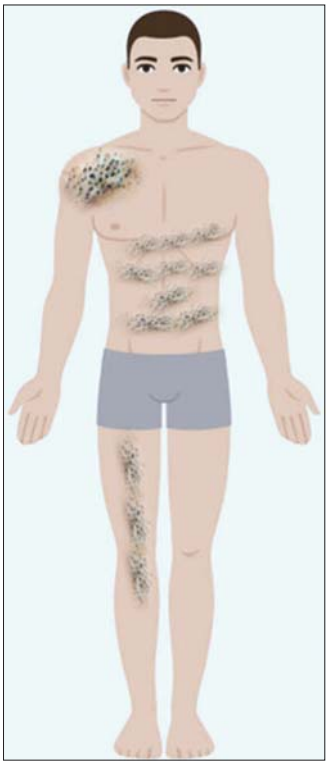


Figure 2: Types of nevus comedonicus in a) segmental, b) Blaschkoid, and c) linear distribution.

This nevus commonly occurs after less than ten years of age. The common sites of predilection are the face, neck, and trunk. They present as keratin-filled pits grouped together, resembling a honeycomb. They are mostly unilateral or segmental yet may also be bilateral, large, linear, or Blaschkoid (Fig. 2). They are asymptomatic and increase in size during puberty. In the case of secondary infection, they may form nodules or abscesses. They heal with scarring. When there is extracutaneous involvement of the spine, eyes, or CNS, it is termed *nevus comedonicus syndrome* [6].

Dermoscopic findings show multiple, dark brown areas studded with keratin plugs and numerous follicular openings [7].

The management includes the use of topical keratolytics such as tretinoin, salicylic acid, and ammonium lactate or destructive procedures such as ablative lasers and surgical excision of small lesions, yet the changes of post-procedure complications such as scarring and pigmentation are high [8].

Chloracne

Chloracne is a dioxin-induced hamartoma otherwise known as metabolizing acquired dioxin-induced skin hamartoma, abbreviated as MADISH. It is a form of occupational acne that produces secondary open comedones and cyst-like lesions due to the irritant effect. The various causative agents such as halogen, insecticides, pesticides, azobenzene, and naphthalene have been known to give rise to chloracne. The routes of transmission include direct contact, inhalation, and ingestion. Based on the quantity and duration of exposure to the chemicals, it may be classified as acute or chronic toxicity. Acute toxicity may cause GI disturbances, dizziness, pancreatitis, neuropathy, and liver disturbances. Skin manifestations are more common in chronic toxicity. Systemic effects may be associated with chronic toxicity as well [9,10]. Famous personalities, such as Ukrainian president Victor Yushchenko, were also victims of dioxin poisoning [11].

Clinically, it presents as numerous, diffuse, open comedones affecting almost all follicles, giving a slate-gray appearance. The most common sites include the face, retro-auricular region, axilla, chest, and trunk. Since these keratinous plugs are produced due to an irritant effect, the areas in which there is more chance of particle collection, such as the retro-auricular region and axilla, are involved. The T-area of the face and perioral region are spared. In severe cases, these areas may also be involved. It may also be accompanied by nodules and cysts. Multiple co-workers and family members are usually affected. It usually begins appearing within the first several weeks of one's occupation. It usually does not respond to the usual anti-acne medications, unless the cause is eliminated. Histopathological examination reveals dilated, cystic, follicular infundibula filled with keratin.

The differentiating points of chloracne vs. acne vulgaris are as follows: Chloracne is caused by an irritant effect, whereas acne vulgaris is caused by androgen hypersensitivity. There is sebaceous gland atrophy as the chemicals concentrate in the sebaceous glands and metabolize slowly, whereas there is sebaceous gland hyperactivity in acne. In chloracne, there is xerosis, as opposed to seborrhea seen in acne. In chloracne, there is a paucity of *Propionibacterium acnes*, whereas in acne vulgaris, there is a perfect anaerobic environment due to the breakdown of the free fatty acids, which facilitates the growth of *P. acnes*.

When it comes to the management of chloracne, the first and foremost step is the avoidance of occupational exposure to the causative agent. The other treatment modalities include the use of topical or oral retinoids and manual expression of the open comedone-like plugs.

Favre–Racouchot Syndrome

Favre–Racouchot syndrome is synonymous with senile comedones, solar comedones (Fig. 1), and nodular elastosis with cysts and comedones. It is characterized by multiple, large comedones, nodules, or cysts on a yellowish background on an actinically damaged face, which is more common in Caucasians and in the male sex [12]. The predisposing factors include chronic sun exposure, smoking, and aging.

The exact pathogenesis of Favre–Racouchot syndrome is unknown. However, excessive UV exposure and the harmful effects of smoke on Favre–Racouchot syndrome are well recognized.

UV Rays in Favre–Racouchot Syndrome

Elastic fiber degeneration leads to solar elastosis and excessive sebum production, leading to an increase in free fatty acids and squalene as well as squalene peroxidases, which promotes comedogenesis and dysregulated keratinization [13].

Smoking in Favre–Racouchot Syndrome

Smoking causes an increased amount of matrix metalloproteinase 1 and 3, which leads to the degeneration of collagen, reduced oxygen supply, and increased production of reactive oxygen species, which eventually leads to reduced synthesis and increased breakdown of collagen with reduced angiogenesis and poor wound healing [14].

Clinically, the patients present with large open comedones with yellowish papules or nodules on the face typically involving the periorbital region (the most common site involving the lateral and inferior aspects), forehead, temporal region, and rarely neck. They are bilaterally symmetrical, although unilateral occurrence has been described. The convexities of the face are the main targets due to UV damage. Due to chronic actinic damage, the surrounding skin shows multiple wrinkles and furrows, and the skin appears leathery [15]. Histopathological examination reveals

epidermal atrophy with actinic elastosis surrounding the cystic lesions. The dermoscopic findings include homogenous, circular areas with shades of light to dark brown with keratin plugs representing open comedones. Multiple, circular, yellowish-brown, homogenous, and structureless areas around the keratin plugs represent the closed comedones. Intervening normal skin shows a yellowish hue due to solar elastosis with arborizing vessels, which represents telangiectasia [16].

The differentiating points from acne are as follows: Acne vulgaris is not common in the elderly. The comedones in Favre–Racouchot syndrome are larger, along with cysts and nodules present on a photodamaged background, and these comedones lack inflammation associated with acne.

The management of this condition includes the elimination of risk factors, such as proper photoprotection, the liberal use of sunscreens, and the cessation of smoking. Retinoids, comedone extraction, peels, hyfrecation, or lasers may be attempted.

Dilated Pore of Winer

The dilated pore of Winer is a benign tumor of the follicular infundibulum commonly seen in middle-aged to elderly women. Clinically, it presents as a large, solitary, follicular, and crateriform depression filled with dark or black keratin, mimicking a giant open comedone (Fig. 3) with no signs of inflammation. The most common sites of predilection are the head and neck yet may occur at other sites as well.

Histopathology reveals a dilated follicular infundibulum with some amount of keratinous material with hyperplastic infundibular epithelium radiating small finger-like projections into the surrounding dermis. Histopathology resembles "a glass of red wine."

Dermoscopy shows central, bluish-black material (representing the keratin) with surrounding translucent, grayish-white margin (representing the epidermal hyperplasia of the follicular infundibulum) [17].

The differential diagnosis of the dilated pore of Winer is mainly pilar sheath acanthoma, which is almost always seen on the upper lip in the elderly. Histologically, pilar sheath acanthoma presents with a large, irregular cavity with multiple, lobulated masses extending to the surrounding dermis, in contrast to the finger-like projections seen in the dilated pore of Winer [18].

The management is only for cosmetic purposes. Surgical excision is curative. Lasers and cautery may also be attempted.

Trichostasis Spinulosa

This indicates the follicular dilation comprising multiple, trapped vellus hairs (around 5–60) presenting as asymptomatic, tiny, black, dilated pores (resembling open comedones) with spinous hair projection (Fig. 4). The most common site of predilection is in the centrofacial region and the most common site is the nose. Rarely, it may be pruritic and disseminated. This is commonly reported in middle-aged and elderly females (due to follicular dilation caused by elastolysis). Dermoscopy shows a paintbrush appearance (hence, it is also known as "pinselhaar") with a small tuft of hair emerging from the central punctum [19-21]. Treatment is mainly for cosmetic purposes, which includes topical

Figure 3: Dilated pore of Winer mimicking a large open comedone.

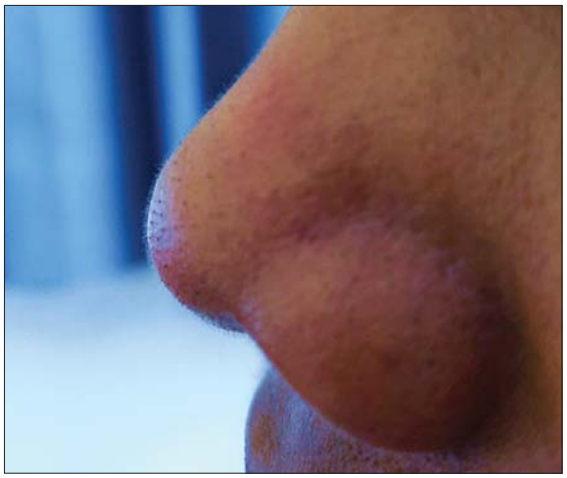
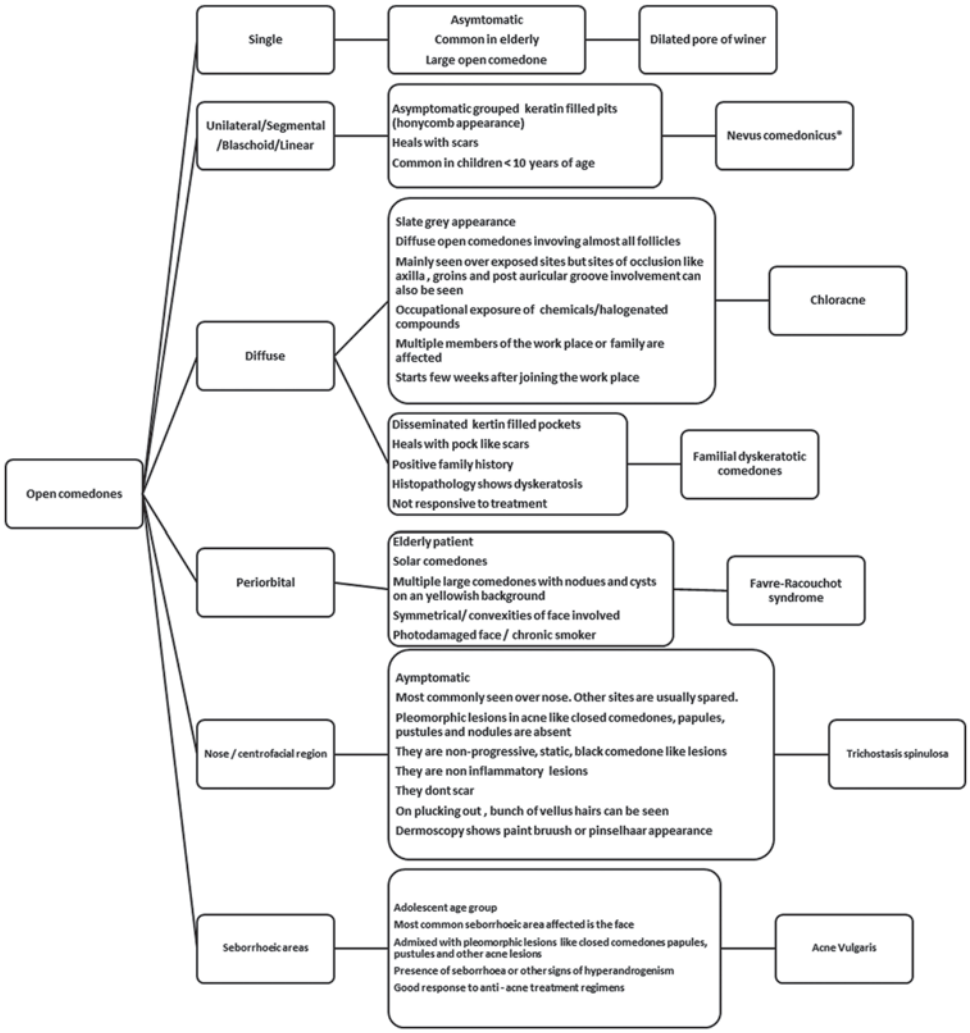


Figure 4: Trichostasis spinulosa mimicking open comedones on the nose.



Flowchart 1: The approach to the diagnosis of mimickers of open comedones. *Rule out other systemic involvement for nevus comedonicus syndrome.

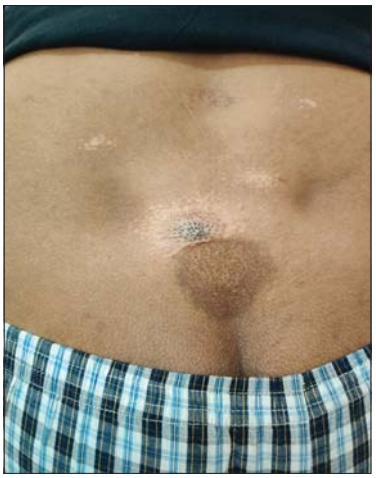


Figure 5: Lichen sclerosus et atrophicus with open comedone-like plugs.

keratolytics, manual extraction with a comedone extractor, tweezers, laser epilation, and adhesive tapes.

Table 2: Disorders with comedones at unusual sites [14]

1. Porokeratotic eccrine ostial and dermal duct nevus (PEODDN)
2. Familial dyskeratotic comedones (FDC)
3. Darier's disease
4. Acne mechanica
5. Nevus corniculatus
6. Comedo nevus
7. Neurofibromatosis
8.Follicular mycosis fungoides
9. Systemic amyloidosis
10. lichen sclerosus et atrophicus (LSEA) [Figure 4]

Table 2 shows disorders with comedones in unusual sites. Fig. 5 shows open comedones in association with lichen sclerosus et atrophicus. Flowchart 1 shows an approach to the differential diagnosis of open comedones.

CONCLUSION

Acne is one of the most common dermatological disorders encountered in our day-to-day practice.

However, when the open comedones are present in unusual sites, occurring in an unusual age group (prepubertal or elderly), not responding to treatment or presenting with a disseminated or Blaschkoid pattern, the other rarer disorders should be kept in mind while diagnosing.

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