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Drug-induced acne

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Abstract A variety of drugs may provoke acne, with drug-induced acne (DIA) often having some specific clinical and histopathologic features. DIA is characterized by a medical history of drug intake, sudden onset, and an unusual age of onset, with a monomorphous eruption of inflammatory papules or papulopustules. The location of the acne lesions is beyond the seborrheic zone. Corticosteroids, anabolic steroids, testosterone, halogens, isoniazid, lithium, and some new anticancer agents are drugs with undoubted causal relationship to acne. The diagnosis of DIA is made by a detailed history with a record of drug onset, dosage regimen and therapy duration, absence of additional triggering factors, and clinical relationship between the introduction of the drug and the onset of an acne-like eruption. In all cases, the withdrawal of the drug should be followed by lessening of the acne lesions.
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Introduction

Drug-induced acne (DIA) is a common skin condition whose classic signs resemble true acne. DIA is provoked by a variety of drugs and has some specific clinical and histopathologic features. The culprit drug can be one orally administered, topically applied, or even inhaled. The earliest report of DIA dates from 1928, when acne-like lesions were described with the use of iodides and chlorinated hydrocarbons.[1]

DIA is very similar to common acne; yet, there are a number of differences to distinguish its clinical picture. DIA is characterized by a medical history of drug intake, sudden onset, unusual age of onset, appearance on the face and neck, and an unusual location of the lesions beyond the seborrheic areas. The eruption consists of inflammatory papules or papulopustules; comedones, if present, are secondary lesions[2] (Table 1).

The number of patients with DIA is currently rising. Considering their acne-inducing capacity, drugs related to acne fall into the following categories:

- Drugs with undoubted causal relationship to acne
- Drugs about which there are considerable, though insufficient, data
- Drugs occasionally reported to be associated with acne (Table 2)[3]

Corticosteroids

Systemic corticosteroids, inhaled steroid therapy, or use of huge amounts of topical corticosteroids is often the reason for the induction and exacerbation of acneiform lesions. Steroid-induced acne often appears in patients with collagen vascular diseases or with neurologic pathology that requires protracted courses of oral corticosteroids. Steroid acne has become more common after the advent of organ transplant surgery and chemotherapeutic regimens.[4]

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Table 1 Differences between DIA and acne vulgaris

Clinical signs	Acne vulgaris	DIA
Medical history	No history of drug intake	History of present illness and/or drug intake
Onset	Onset during teenaged years, or late onset beginning after the age of 25 years (adult acne)	Sudden onset (occurrence often away from acne age)
Acne localization	Appearance on the face, chest, and back	Appearance on the face and neck, unusual location of the lesions beyond the seborrheic areas
Acne lesions	Polymorphous eruption: Comedones, papules, pustules, nodules, and cysts	Monomorphous eruption: Inflammatory papules or papulopustules; no comedones or, if present, they are secondary lesions
Therapy	Conventional acne therapy	Resistance to conventional acne therapy
Prognosis	Acne can almost always be controlled with medication; however, results may not be seen for weeks or months.	Lesions disappear with the discontinuation of the inducing drug

DIA, drug-induced acne.

Factors predisposing to steroid acne are high concentration of the drug, application under occlusion, young adults below age 30 years, whites in preference to blacks, and application to acne-prone areas of face and upper part of the back.[5]

The exact pathogenesis of steroid acne is still uncertain. The accelerated chronologic progression of infundibular spongiosis, hyperkeratosis, microcomedo formation, and hair follicular rupture is significant for the development of the papules and papulopustules in steroid acne.[4] Several reports have shown that steroid-induced TLR2, together with *P. acnes*, plays an important role in the exacerbation of acne vulgaris.[6]

The severity of the acne eruption seems to depend on the dose, the treatment duration, and the medical history of acne. The eruption usually starts after several weeks of treatment. It is located predominantly on the trunk and extremities, with less involvement of the face. It is possible that the use of a facial mask may concentrate the exposure to inhaled corticosteroids, thus leading to acneiform eruptions in the midfacial region.[7]

The clinical picture consists of a monomorphous papulopustular eruption. Comedones may be present predominantly in cases with topical steroid application. Nodules and cysts are rarely observed. Steroid acne has been described with a similar clinical picture to *Pityrosporum* folliculitis.[8]

Steroid acne usually resolves after discontinuation of the drug. Conventional treatment for acne vulgaris is recommended if the steroid drug should be continued. Tretinoin topically is often preferable.[9]

Anabolic steroids

Anabolic-androgenic steroids (AAS) represent a class of synthetic steroid hormones related to the testosterone. The use of self-administered AAS by recreational bodybuilders is a well-recognized phenomenon. Acne is one of the most frequent adverse effects of these drugs, but for many users it has been an acceptable one.[10] DIA occurs in about 50% of

Table 2 Drugs with acne-inducing potential

Drugs with undoubted causal relationship to acne	Drugs with considerable, but insufficient, data	Drugs occasionally associated with acne
Corticosteroids	Cyclosporine A	Vitamin B6
Anabolic steroids	Tacrolimus, sirolimus	Vitamin B1
Testosterone	Vitamin B12	PUVA
Isoniazid	Vitamin D2	Propylthiouracil
Halogens (iodines, chlorides, bromides)	Phenobarbiturates	Anticancer agents (VEGFi, anti-TNF-α)
Anticancer agents (EGFRi, BRAFi, MEKi)	Disulfiram	Voriconazole
Lithium	Azathioprine	Dactinomycin
	Quinidine	Rifampicin
	Amoxapine	Etambutol
	TNF-α inhibitors	Sertraline
	Tetraethylthiuram	Muscle relaxant (dantrolene)
	Tricyclic antidepressants (amineptine)	
	Thyreostatica (thiouracil)	

BRAFi, BRAF inhibitors; EGFRi, EGFR inhibitors; MEKi, MEK inhibitors; TNF-α, tumor necrosis factor α; VEGFi, VEGF inhibitors.

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