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Monomorphous papulopustular rash

A case study

Keywords: acneiform eruptions

Case study

An otherwise healthy male patient, aged 16 years, presented with a mildly pruritic monomorphous papulopustular rash located on the neck, chest, back, shoulders and upper arms (*Figure 1*). Comedones were absent. The patient was a soccer player and had received an intra-articular injection of betamethasone dipropionate/betamethasone sodium phosphate in the left knee, 3 days prior to the onset of rash, for the treatment of post-traumatic knee pain. He reported malaise but denied fever, taking any other drugs, over-the-counter medications or 'natural remedies'.

Question 1

What is the diagnosis?

Question 2

What causes this condition?

Question 3

What drugs have been implicated in the origin of this type of eruption?

Question 4

How would you confirm the diagnosis?

Question 5

What are the differential diagnoses?

Question 6

What therapeutic measures would you recommend?

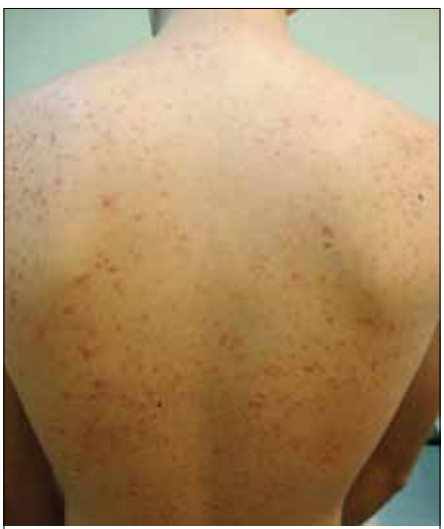

Figure 1. Lesions on the patient's back

Answer 1

The characteristics of the rash, the absence of other diseases or drug intake (other than intra-articular steroid) and the chronological relationship between intra-articular steroid injection and the rash onset suggest steroid induced acneiform eruption, steroid acne or steroid induced acne, or more specifically acneiform eruption induced by intra-articular betamethasone administration. Several other drugs have been associated with the development of similar eruptions, commonly known as acneiform eruptions, drug induced acne or acne medicamentosa. While these eruptions may superficially resemble acne vulgaris, their aetiology is thought to be different.[1]

Answer 2

Steroid induced acne is most commonly encountered in older patients after either topical

or systemic steroids, including patients with collagen vascular diseases or neurological pathology that require protracted courses of oral corticosteroids – particularly potent ones such as dexamethasone.[2] It is a follicular eruption beginning with an inflammatory lesion, usually a papule or pustule. Unlike acne vulgaris, comedones are later secondary lesions, a sequel to encapsulation and healing of the primary abscess.[1] Probably no single mechanism is implicated in the pathogenesis of the eruption. It has been suggested that glucocorticoids markedly enhance toll-like receptor 2 (TLR2) expression in human keratinocytes, which is further stimulated by *Propionibacterium acnes* and proinflammatory cytokines, resulting in acne vulgaris exacerbation and, possibly, in steroid induced acneiform eruption or rosacea-like dermatitis induction.[3] The direct effect of the steroid on the follicular epithelium appears to cause a focal degeneration with a localised intrafollicular and perifollicular neutrophilic inflammatory reaction. Contrary to acne vulgaris, follicular hyperkeratosis is not an early event.[1] It should be noted that acneiform eruption after intra-articular steroid injection appears to be a less common event, although it is probably under-reported in the literature. In fact, acneiform eruption is listed as a dermatological effect of several steroids used intra-articularly.[4] This is because a significant proportion of the injected drug will be absorbed systemically. Many of the side effects of intra-articular steroid injection are similar to those arising after oral or intravenous administration of steroids.[5]

Answer 3

Substances other than corticosteroids responsible for acneiform eruptions include: anabolic steroids such as danazol and testosterone (known as bodybuilding acne, doping acne, power athlete acne), corticotropin, iodides and bromides (halogen acne), epidermal growth factor receptor inhibitors, cyclosporine, azathioprine, anticonvulsants, antipsychotics, antidepressants, tumour necrosis factor-α blockers, tuberculostatic drugs, tetracyclines, quinidine and complex B vitamins.[1,6,7]

Answer 4

The diagnosis of steroid induced acneiform eruption is generally obvious if a high index of suspicion is maintained. It is essential to collect a thorough history from the patient including any recent illness and medications. As steroid induced acneiform eruption is a nonallergic reaction, skin testing is not useful for diagnosis. Important clues for diagnosis are:

- sudden onset within days after initiating steroid therapy
- widespread involvement
- presence of lesions in unusual locations (eg. distal parts of the extremities, buttocks, or restricted to the trunk)
- occurrence beyond teenage/young adult age
- monomorphous papulopustular eruption without comedones (or with secondary comedones only)
- signs of systemic drug toxicity with fever and malaise
- disappearance of the inflammatory lesions without scar formation after withdrawal of the offending medication, sometimes leaving secondary comedones
- some resistance to conventional acne therapy if the steroid is not interrupted.[1,6,7]

Answer 5

The main diagnoses to be considered in this patient's clinical scenario are acne vulgaris, Gram negative folliculitis, acne aestivalis and eosinophilic pustular folliculitis. Other differential diagnoses include acne necrotica, rosacea, perioral dermatitis and pseudofolliculitis barbae.[1,8]

Answer 6

Discontinuation of the steroid, when medically feasible, is first line treatment. Sometimes lesions may clear despite continuing the steroid medication. In cases where drug interruption is not possible, the lesions can be controlled with standard acne treatments, although some resistance to therapy can occur. In this case study, this was not a problem because the intra-articular steroid administration was episodic. However, we decided to treat the patient because of the extension of the lesions and the related psychological impact. Reassurance that the condition is self limited is critical. For their anti-inflammatory activity, oral tetracycline antibiotics (eg. doxycycline) and topical antibiotics could be helpful. Topical retinoids and benzoyl peroxide can also be used.[9] Recent observations suggest that oral isotretinoin could be an option for severe cetuximab induced acneiform eruption,[10] but there are no reports of its use in acneiform eruption induced by steroids.

Case follow up

The patient was treated with oral doxycycline (100 mg/day) and a twice daily application of erythromycin 4% + zinc acetate 1.2% over the affected area, with complete resolution of lesions within 15 days.

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Conflict of interest: none declared.

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