

Asymptomatic papules over central and pericentral areas of the face

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ABSTRACT

We report the case of a 24-year-old college girl, presenting with numerous acneiform papular eruptions over the central part of the face.

Key words: Acneiform eruption, cutaneous lymphoid hyperplasia, pseudolymphoma

INTRODUCTION

Facial papular lesions sometimes pose a diagnostic challenge. We report an unusual case of central and pericentral facial papular lesions mimicking acne vulgaris in a 24-year-old female.

CASE REPORT

A 24-year-old college-going female presented to the outpatient department with the complaint of numerous asymptomatic, small, raised lesions over the central part of the face (cheekbones, nose, forehead and chin) since 7–8 years [Figure 1]. She reported that while the old lesions persisted, new lesions appeared as crops over the same site. She perceived a mild burning sensation of the face upon going out in the sun and also complained of occasional, mild itching. There was no significant history of drug intake, topical application or infection. A provisional diagnosis of acne vulgaris had been made earlier and she had been treated with antiacne drugs without any improvement. Physical examination revealed multiple firm, dome shaped and skin colored papular lesions over malar region, nose, forehead and chin on a mildly erythematous base. The lesions were nontender, nonscaly and the size varied between 2 to 8 mm. The surface was hairless without telangiectasia. She was otherwise healthy. All routine blood investigations including complete blood count, liver function test, urea, creatinine and chest radiograph were within normal limits. The clinical differential diagnosis we considered were acne vulgaris,

sebaceous hyperplasia, acneiform drug eruption, syringomas, trichoepitheliomas, milia, papular sarcoid and lupus miliaris disseminatus faciei. On histopathological examination (HPE), biopsy of a papular lesion revealed that epidermis was unremarkable except for focal atrophy over the lymphoid follicles, with an underlying ill defined grenz zone. Dermis showed nodular infiltration in the upper and mid dermis comprising mainly of lymphocytes admixed with plasma cells and histiocytes. Pilosebaceous units and adnexal structures were also unremarkable. No atypical cell or granuloma was evident [Figures 2 and 3]. Immunohistochemical analysis displayed more than 90% CD3 (Pan T-Cell marker) positivity among the cells [Figure 4]. Considering all the above features we reached at a diagnosis of T-Cell predominant cutaneous lymphoid hyperplasia (CLH) [Table 1]. We treated our patient with a mid potent topical corticosteroid (mometasone furoate) for three weeks and most of the lesions disappeared [Figure 5].

DISCUSSION

Cutaneous lymphoid hyperplasia (CLH) is usually a benign, reactive, dermal B- or T-lymphocytic inflammatory response of unknown cause that may mimic lymphoma. It is commonly seen in adults and less often in children with a slight predilection for women. It was first described in 1894. CLH manifests clinically with isolated, or rarely few multiple nodules, mainly on the face and extremities.^[1] CLH is mainly a histopathological condition rather than a clinical entity.^[2]

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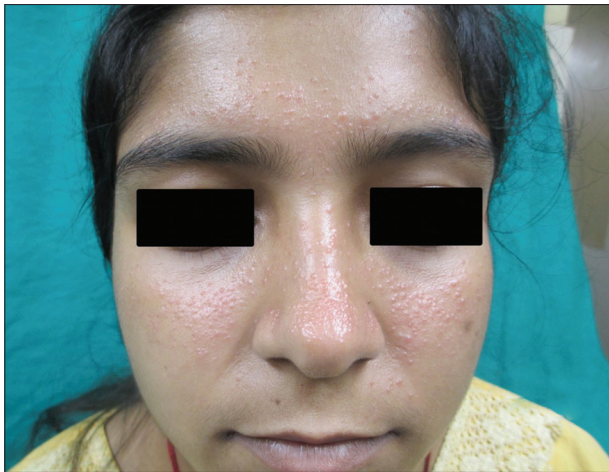


Figure 1: Numerous papular lesions on erythematous base over central part of face

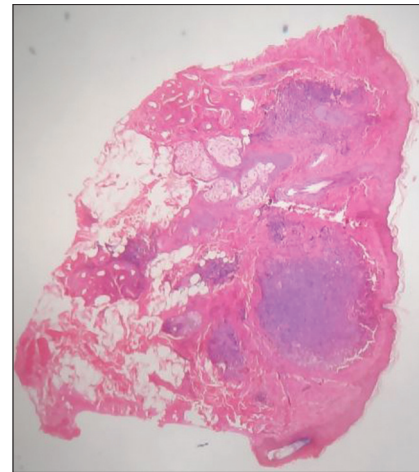


Figure 2: Photomicrograph showing lymphoid follicles in the upper and mid dermis (H and E, ×40)

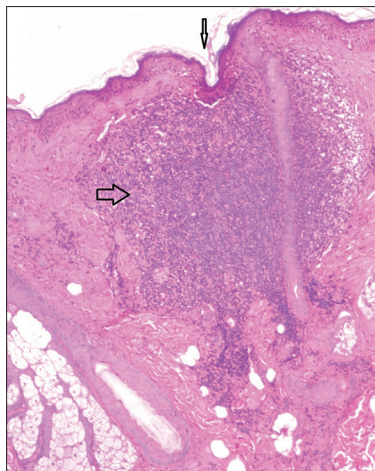


Figure 3: Photomicrograph showing a dense lymphocytic infiltrate in the upper and mid dermis (H and E, ×100) (Arrows indicating focal atrophy of epidermis and lymphoid follicle)

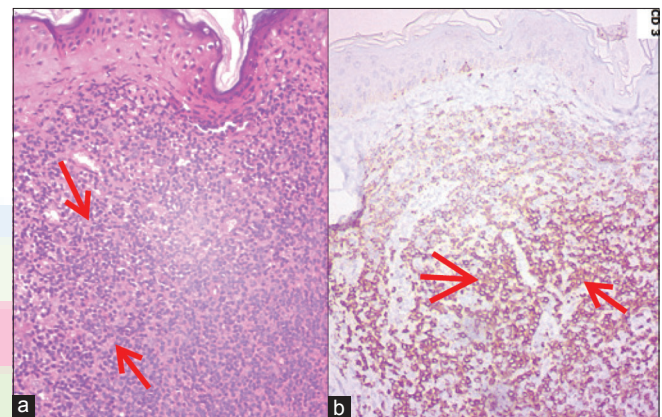


Figure 4: (a) Photomicrograph showing polymorphic infiltrate comprising mainly of lymphocytes, histiocytes, and plasma cells (H and E, ×400), (b) immunohistochemical staining demonstrates numerous CD3⁺ T-cells in the dermis (×400)



Figure 5: Central part of face after 3 weeks of treatment with topical steroid showing disappearance of lesions

Depending on the predominant cell type in the infiltrate, CLH may be B-cell predominant (borreliolymphocytoma cutis,

tattoo-induced lymphocytoma cutis and post-zoster scar lymphocytoma) or T-cell predominant (lymphomatoid drug reaction, lymphomatoid contact dermatitis and persistent nodular arthropod-bite reactions). A variety of etiological factors may be involved, including parasitic infections such as *Borrelia burgdorferi* and *Leishmania panamensis*, viral infections, trauma, drugs, arthropod bites, tattoos, zoster and vaccinations. However, the cause of the disease most of the time remains obscure. Clinically, both T- and B-cell CLH may occur as isolated or multiple cutaneous firm, erythematous to violaceous papules or nodules without surface changes, mainly on head, neck, or upper extremities. Patients do not generally have any systemic involvement.^[3] The peculiarity in our case was that the patient presented with numerous papular lesions appearing in crops over the central part of the face with mild erythema, which was a very unusual presentation of CLH.

On HPE, CLH often reveals a superficial and deep dermal, nodular or diffuse polymorphic infiltrate comprising mainly of lymphocytes, and histiocytes and occasional plasma cells,

Table 1: A differential diagnosis of the case

Disease	Clinical feature	HPE	Immunohistochemical
Acne vulgaris	Open or closed comedones, papules, pustules, nodules and cystic lesions. Respond to antiacne treatment	Various stage of progression of the lesions, mainly involving pilosebaceous unit	-
Sebaceous hyperplasia	One or several elevated, small, soft, yellowish, slightly umblicated papules over the central and upper face	Consist of single enlarged sebaceous gland composed of numerous lobules	-
Milia	Multiple white tiny papules, mainly over the face	Small epidermal cyst with stratified squamous epithelium lining and laminated keratin cyst	-
Trichoepitheliomas	Multiple skin colored papules or nodules, over the face, mainly nasolabial folds	Presence of well circumscribed small symmetric lesion in upper dermis, horn cyst and basaloid epithelial formation	CD34+ Bcl2+
Syringomas	Multiple, small, skin colored papules, classically over periorbital area	Numerous tubular structures, lined by 2 layered epithelial cells, embedded in a dense collagenous stroma	-

HPE: Histopathological examination

dendritic cells and eosinophils. A clear grenz zone may be present. In deep dermis, well defined germinal centers, often surrounded by a mix of B and T cells may be seen with central large lymphoid cells with abundant cytoplasm. CLH needs to be distinguished from cutaneous lymphomas by clinico-histopathological correlation, histochemical studies, and in some cases, gene re-arrangement studies. Immunohistochemical studies might be helpful further to demonstrate the polymorphic nature of infiltrate, including CD3-positive T lymphocytes, CD20-positive B lymphocytes and CD68-positive histiocytes.^[3,4] In our case, suggestive HPE and >90% CD3⁺ cells in the infiltrate were indicative of T-cell predominant CLH.

Most cases resolve spontaneously within a few weeks to months. A conservative approach should be considered initially. A variety of treatment options are available including topical or intralesional steroids, cryotherapy, thalidomide, interferon- α , laser and surgical excision. In documented CD20⁺ lesions, intralesional rituximab may be tried.^[1,5] Topical corticosteroids, which aggravate acne, remain the mainstay of treatment of CLH.^[6]

Cutaneous lymphoid hyperplasia should always be suspected whenever a patient presents with numerous recalcitrant small

papules over central part of the face for a considerable time period.

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