Localized post auricular annular lesion

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CLINICAL FINDINGS

A 72-year-old male presented with asymptomatic localized slowly growing nodular lesions on the head and neck for about 9 years. The lesion started as a small nodule on the post auricular area that slowly progressed downwards to the upper part of the mandibular region. The patient complained of some irritation on the borders of the lesion. In the last few months, some eroded areas appeared within the lesion, and started to bleed minimally. There was no symptom suggestive of any systemic disease, and there was no family history of similar lesions. Cutaneous examination showed large annular lesion localized to the right post auricular area and extended into the mandibular region. The lesion measured about 8x3cm and showed a raised border (Fig. 1). The central area of the lesion was almost atrophied (Fig. 2), while the border showed erosions and small pigmented nodules (Fig. 3). General examination was irrelevant while routine investigations revealed no significant abnormalities.

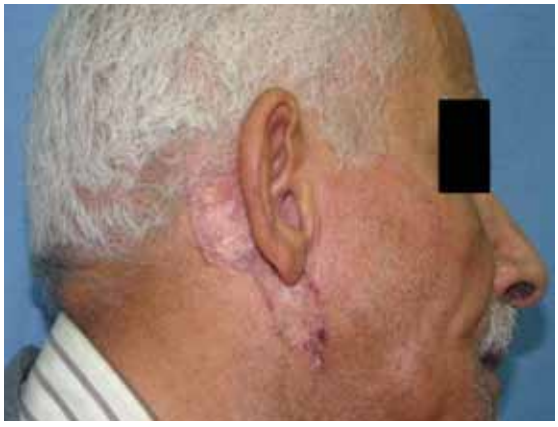

Fig. 1 A large annular lesion localized to the rt. post auricular area and extended into the upper mandibular region.

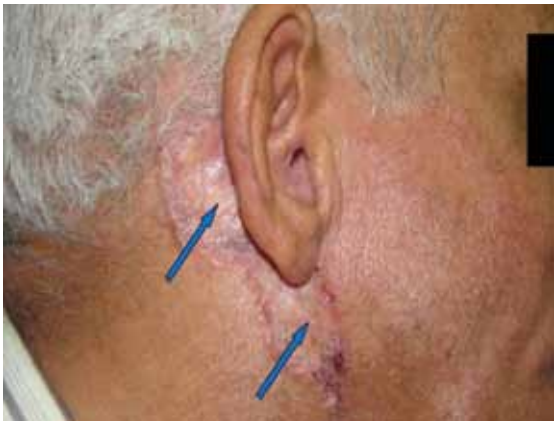

Fig. 2 The central area of the lesion was almost atrophied.

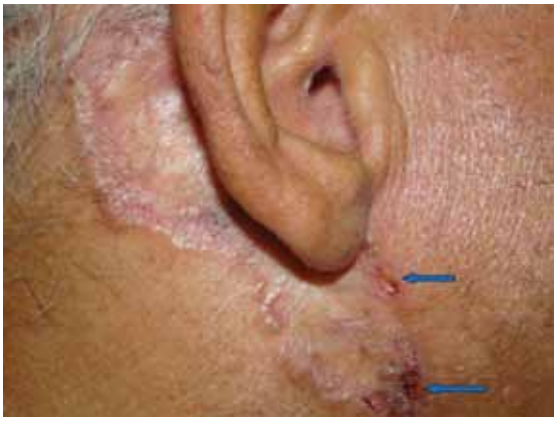

Fig. 3 The border of the lesion showed erosions and small pigmented nodules.

What is your clinical differential diagnosis?

Lupus vulgaris - Basal cell carcinoma - Annular sarcoidosis - Granuloma annulare - Lichen planus.

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PATHOLOGICAL FINDINGS

A skin biopsy from the border of the lesion showed large basaloid masses extended into the mid-dermis and connected to the epidermis in focal areas. The epidermis shows marked erosion with scale-crust formation (Fig. 4). These masses were formed mainly of basaloid cells and showed peripheral palisade arrangement (Fig. 5). There was minimal mitosis, and few atypical cells but melanin pigment was predominant in few areas. The surrounding stroma was retracted around some masses to show a clear cleft while the other areas of stroma showed mild inflammatory infiltrate with marked fibrosis and increased vascularity (Fig. 6).

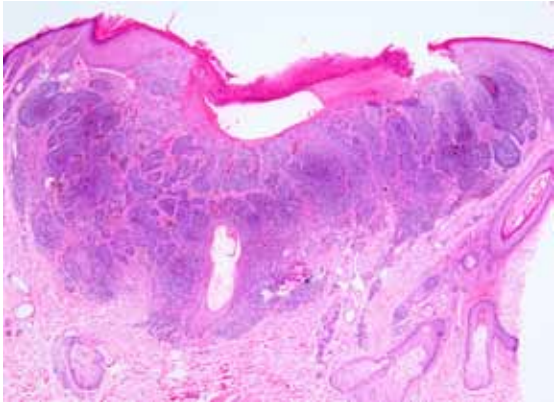

Fig. 4 Large basaloid masses extended into the mid-dermis and connected to the epidermis in focal areas. The epidermis shows marked erosion with scale-crust formation (H&E x20).

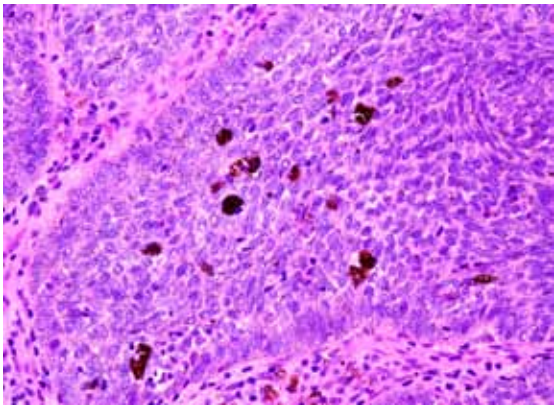

Fig. 5 These masses were formed mainly of basaloid cells and showed peripheral palisade. There was minimal mitosis and few atypical cells but melanin pigment was predominant in few masses (H&E x200).

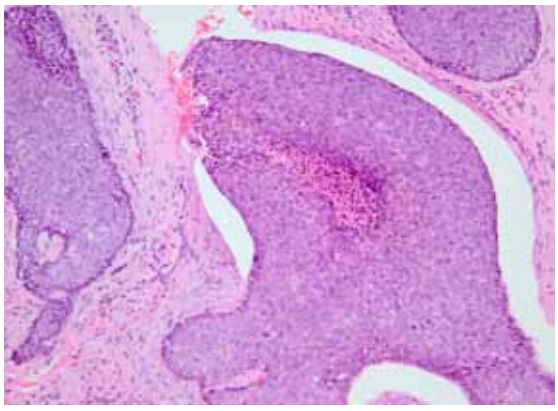

Fig. 6 The surrounding stroma was retracted around some masses to show a clear cleft while the other areas of stroma showed mild inflammatory infiltrate (H&E x400).

DIAGNOSIS

Basal Cell Carcinoma(BCC).

COMMENT

BCC comprises the majority of non-melanoma skin cancers (NMSCs) and is more common than all other human malignancies combined. Several lines of evidence suggest that the worldwide incidence of BCCs is increasing. In the United States, the diagnosis and treatment of NMSCs has increased dramatically with a growth rate of 77 % over the past two decades. The fastest growing group is in women under the age of 40 years.[1]

In other countries, such as Singapore where incidence of BCCs is monitored, the rate of BCCs has been rising over the past several decades as well. Overall, the reasons for this dramatic growth have been postulated to include the aging population, changes in sun exposure habits, environmental changes, migration patterns, and to a lesser extent, increased prevalence of immunosuppressant use.[2] Ultraviolet radiation (UVR) is the major etiologic agent in the pathogenesis of BCC. However, increased incidence of BCC among fair-skinned populations living in regions with different ambient UVR, highlights the importance of sun

exposure. The studies of personal sun exposure suggest lower levels of risk, with typically less than a doubling in risk with high self-reported levels of sun exposure.[3]

There is a plateau of BCC incidence with age and a reportedly stronger association with recreational than with occupational or total exposure, and it has been suggested that childhood might represent a critically important exposure window.[4] In addition, while BCC occurs predominantly on the head and neck, approximately 25% arise on less sun-exposed body sites such as the trunk. This is in contrast with squamous cell carcinoma (SCC), where only 8% of lesions occur on the trunk and the strongest risks seem to result from a lifetime of high sun exposure.[5]

Morphological classification of BCC includes: nodular (with micronodular), infiltrative (with morphoeic), superficial and mixed subtype. The nodular subtype occurs most commonly on the head (mainly the nose and forehead), neck and upper back, while the micronodular subtype occurs most commonly around the eyes. The morphoeic form localizes mainly on the nose, eye angles, forehead and cheeks and it is very rare on the trunk. The lesions of superficial BCC, usually multifocal, and localized on the trunk. In some cases superficial BCC may appear on the head, within the parietal part of the scalp.[6]

Histologically, BCC is composed of uniform cells with round or oval basophilic nuclei, larger and darker than nuclei of epidermal basal keratinocytes, with minimal cytoplasm. Cellular atypia is infrequent in most cases of BCC, excluding the rare pleomorphic (giant cell) type, but mitoses and apoptotic cells are frequent. Interestingly, nuclear atypia and multiple mitoses do not alter the clinical course of BCC.[7]

The most common histological type is nodular BCC with solid, well-bordered, irregular, lobulated tumor nests of various sizes surrounded by dense stroma with numerous fibroblasts and mucinous material, mostly hyaluronic acid. Typical for this type is empty peritumoral cleft due to retraction of the stroma. The tumor nests extend to the papillary and reticular dermis. The basaloid cells form a regular palisade at the periphery while their distribution in the middle is chaotic.[8] In the center of larger lobules, areas of necrosis may develop leading to formation of cystic spaces containing mucinous material (nodulocystic BCC). In some places keratin cysts with parakeratotic debris can be seen as an example of hair follicle differentiation potential (keratotic BCC).[9]

Highly efficient treatment modalities such as surgery that aims at complete excision, radiotherapy, curettage, cryotherapy, photodynamic therapy, and topical applications of imiquimod or 5-fluorouracil are available and effective for the great majority of BCC patients. However, the occurrence of locally aggressive and invasive tumors, a bleak prognosis upon metastatic spread, a significant rate of recurrence often associated with increased aggressiveness, as well as the multitude of tumors appearing in high-risk populations such as BCNS patients, provide compelling reasons to search for new preventive and therapeutic avenues.[10]

Treatment of basal cell carcinoma with Mohs micrographic surgery has the lowest recurrence rate. However, because of cost and limited availability, it is best considered for larger tumors (greater than 2 cm), for more invasive histologic subtypes (micronodular, infiltrative, and morpheaform), or for tumors at sites with higher risk of recurrence.[11] Smaller nodular

tumors that are located outside of the H region of the face, which includes the nose, eyelids, chin, jaw, and ear, can be appropriately treated with standard surgical excision. The recurrence rate for tumors treated with Moh's micrographic surgery is approximately 1 percent at five years, whereas standard surgical excision has an approximately 5% recurrence rate at five years.[12]

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The clinicopathological challenges of annular BCC

Diagnosis	Clinical	Pathological
Lupus vulgaris	• A well-demarcated skin-colored to erythematous plaque • Shows scarring after healing • Most common on the face and lower half of the body • Morphological variants include plaque or keratotic type, hypertrophic, ulcerative, atrophic and planar	• Typical epithelioid granulomas in the upper dermis • Dense lymphocytic infiltrate and may be plasma cells • Langhans giant cells could be seen in more than 80% of the cases • Epidermal changes include hyperkeratosis, papillomatosis and acanthosis.
Annular sarcoidosis	• Multisystemic disease with common pulmonary impairment • Described in all races, genders and age ranges • Nonspecific symptoms such as fever, malaise, fatigue in 1/3 of cases • Presented with erythematous-violet papules, some with central umbilication	• Nodular dermal granulomatous reaction • Well formed granulomas containing histiocytes with broad and vacuolated cytoplasm • Absence or minimal lymphocytes surrounding the tubercles • Negative Ziehl-Nielsen and PAS stains
Granuloma annulare	• Localized clusters of small papules, coalescing to form annular plaques • It often occurs symmetrically • Lesions are typically asymptomatic • More common in females • Morphological variants include generalized, papular, nodular, patch, perforating and subcutaneous	• The reaction is usually limited to the upper and mid dermis • Granuloma may be palisaded or interstitial • Mucin deposition is common in palisaded granuloma with central area of collagen degeneration • Epidermis is usually normal
Annular Lichen planus	• Presented with flat-topped violaceous papules covered by fine white lines • Affect the flexor surface of the extremities, the trunk, and the genitalia • May involve mucous membranes and nails	• Band-like lymphocytic infiltrate admixed with melanophages • Vacuolar alteration of the basal layer • Acanthosis with irregular elongation of rete ridges with 'saw tooth' appearance • Wedge-shaped hypergranulosis • Hyperkeratosis
Actinic keratosis	• Affect elderly, fair-skinned, sun-sensitive persons • Arise in sun-exposed areas • Presented as circumscribed red, scaly lesions • May be hyperkeratotic or pigmented	• Basal dysplasia and architectural disorders of the epidermis • Atypical keratinocytes with variable size and shape in the lower epidermis • Thin granular layer, hyperkeratosis and focal parakeratosis