Solitary erythematous firm noduloplaque on nose

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CLINICAL FINDINGS

A 36-year-old man presented with asymptomatic red firm noduloplaque on bridge of nose for the last 4 months. It started as a small papule and rapidly increased in size in last couple of months. There was no history of previous similar lesions or other skin problems. The patient had no systemic complaints and there was no family history of similar lesions.

Local examination of the skin revealed well defined firm erythematous papulonodular lesion on bridge of nose. (Fig. 1). Hair, nails and mucous membranes were not affected and showed no significant abnormalities.

Routine laboratory investigations were all within normal limits. Chest x-ray was normal.

What is your clinical differential diagnosis?

Angiolymphoid hyperplasia
Nodular Basal cell carcinoma
Granuloma Faciale
Keratoacanthoma
Tumid lupus erythematosus
Jessner's Lymphocytic infiltrate of skin

Pathological findings

A wedge incision biopsy was taken from the periphery of the lesion. It showed dense dermal granulomatous inflammation. The infiltrate typically contained predominantly histiocytes, lymphocytes and plasma cells. Histiocytes contained small oval organisms with bar shaped paranuclear kinetoplast. (Fig. 2, 3). Immunohistochemical staining showed positive staining for using G2D10 antibody (Fig. 4)

DIAGNOSIS

Cutaneous Leishmaniasis

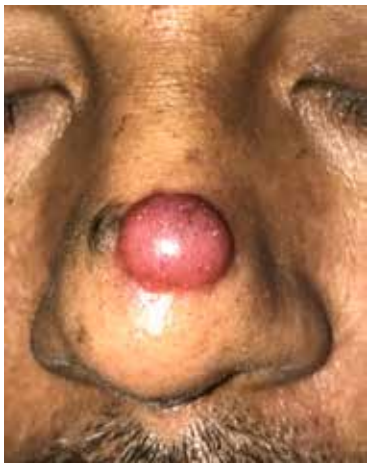

Fig. 1 Well defined erythematous nodule on bridge of the nose.

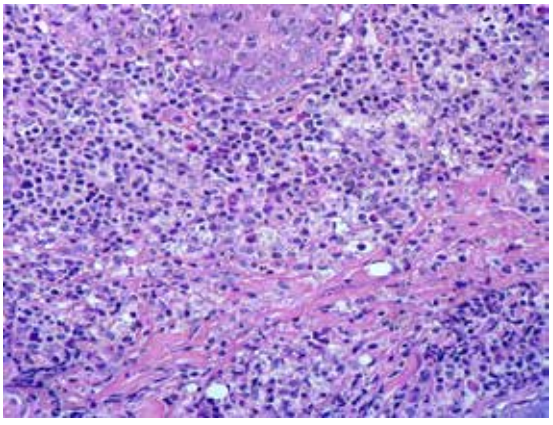

Fig. 2 Diffuse dermal granulomatous infiltrate containing histiocytes and plasma cells (H&E x40).

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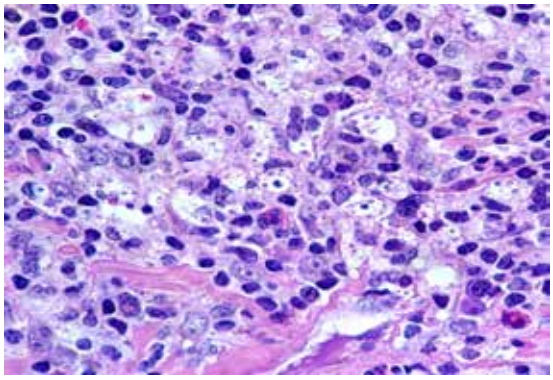

Fig. 3 Showing LD bodies in amastigote forms in the macrophages (H&E x 100).

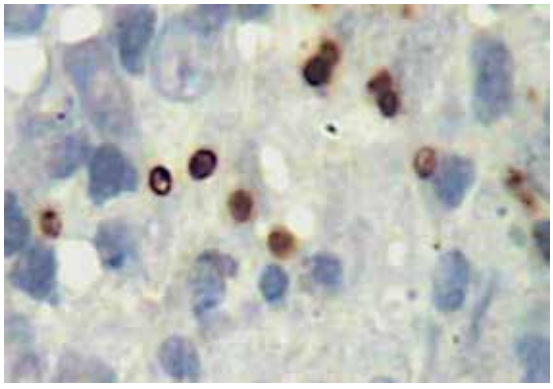

Fig. 4 Immunohistochemistry showing G2D10 positive LD Bodies (IHC x400).

COMMENT

Leishmaniasis is a disease caused by an intracellular protozoan parasite (genus Leishmania) transmitted by the bite of a female phlebotomine sandfly.[1] Leishmaniasis is endemic in more than 70 countries worldwide and affects an estimated 12 million people. There are several clinical forms of leishmaniasis. The clinical manifestation of the infection depends on the species of Leishmania, which varies with geographical area and the host's immune response. The clinical spectrum of leishmaniasis ranges from a self-resolving cutaneous ulcer to a mutilating mucocutaneous disease and even to a lethal systemic illness.

Cutaneous leishmaniasis (CL) typically occurs at the site of inoculation. The presentation and prognosis will vary depending on the species involved.

Old World cutaneous leishmaniasis

- Middle East, North Africa, Asia.
- L major, L tropica, L infantum, L donovani.
- Synonyms: oriental sore, Baghdad boil, Delhi boil, saldana, Aleppo button, granuloma endemicum.

New World cutaneous leishmaniasis

- Central and South America.
- L mexicana, L braziliensis, L amazonensis.
- Synonyms: chiclero ulcer, uta, ulcera de Bauru, forest yaws, pian boi, bejuco.

Clinical features

CL is the most common form of leishmaniasis.[2] It is typically characterized by solitary lesions. It starts as a small red papule, which gradually enlarges up to 2 cm in diameter, and eventually ulcerates in the center. The lesions appear on exposed areas of the skin, especially the face and extremities. These are usually painless and most often resolve spontaneously leaving behind atrophic scar in 2 months to more than 1 year time.[3] The incubation time between an infected sandfly bite and lesion development is typically 2 weeks to 6 months. However, chronic disease can occur and there is a risk of dissemination in immunocompromised patients.

Diagnosis

The diagnosis of cutaneous leishmaniasis is usually based on the history and clinical appearance of the lesion. A detailed travel history, including historical travel to endemic areas is important due to the long incubation period. The diagnosis can be confirmed by identifying the parasite on biopsy or split skin smear.[4-5]

The clinicopathological challenges of solitary cutaneous leishmaniasis nodule.

Diagnosis	Clinical	Histologica
Angiolymphoid hyperplasia with eosinophilia	• Uncommon, vasoproliferative, idiopathic condition that manifests in adults as isolated or grouped papules, plaques, or nodules in the skin of the head and neck. • Most patients present with lesions in the skin of the periauricular region • May be associated with pain or pruritus • Extracutaneous sites have also been noted.	• Shows marked proliferation of blood vessels with distinctive large endothelial cells. • These blood vessels are accompanied by a characteristic inflammatory infiltrate that includes eosinophils. • Inflammatory cells may penetrate into the lumen of the blood vessels, blocking or rupturing them.
Nodular Basal cell carcinoma	• The most common type of BCC • Usually presents as solitary, shiny, nodule with large telangiectatic vessels on the surface • Slow growing, usually asymptomatic • It is often seen older adults • Commonly observed on the head and neck area • May spontaneously bleed or ulcerate	• Consist of the key feature of a basaloid epithelial tumour arising from the epidermis. • The basaloid epithelium typically forms a palisade with a cleft forming from the adjacent tumour stroma. • Centrally the nuclei become crowded with scattered mitotic figures and necrotic bodies evident. • A useful distinguishing feature from other basaloid cutaneous tumours is the presence of a mucinous stroma. • Some tumours may also show foci of regression, seen as areas of eosinophilic stroma with lack of basaloid nests.
Granuloma Faciale	• lt is characterized by single or multiple papules, plaques or nodules, most often occurring on the face. • Most often affects healthy middle-aged white men • Usually asymptomatic • Varies in color, can be skin colored, to reddish-brown, blue or purple • Size can vary from a few millimetres to several centimetres • Elevated and soft • Characterized by prominent follicular opening on the surface	• Dense cellular infiltrate, often with a nodular outline, occupying the mid dermis • Deep dermis and subcutaneous fat may be involved in some cases • Typically spares the immediate subepidermis and hair follicles, forming a Grenz zone • Infiltrate is polymorphic, containing eosinophils, neutrophils and an admixture of plasma cells, mast cells and lymphocytes • Red cell extravasation is often present • Blood vessels appear dilated and their walls are infiltrated by eosinophils and fibrin deposition • Older lesions may show fibrosis and hemosiderin deposition
Keratoacanthoma	• Typically solitary. However, familial cases may be multiple • Usually 80% patients are males • Seen primarily on sun exposed skin of face • Arises from normal skin, grows rapidly for 4 - 8 weeks, then regresses over 6 months • Heals with depressed, annular scar. • It can rarely metastasize, usually in immunosuppressed patients • Gryzbowski type: multiple eruptive lesions • Ferguson-Smith type: multiple ulcerating tumors with atypical distribution • Subungual keratoacanthoma: arise from nail matrix; rapidly growing mass in tip of finger or toe	• Composed of well circumscribed solid lobules of large, pale squamous cells with little keratinization • Distorted follicular infundibulum • Mild atypia can be seen. • Central crater filled with keratin but no granular layer • Old lesions are characterized by large and more irregular infiltrating squamous nests and islands, accompanied by marked inflammatory infiltrate with lichenoid features and eosinophils but plasma cells are characteristically absent
Tumid lupus erythematosus	• Strictly photosensitive, affects both sexes equally • Presents classically with erythematous, edematous plaques • Often occurring independently of the other forms of LE either, cutaneous or systemic • It resolves with normal skin, leaving no residual scarring, or pigmentary alterations • ANA may be detected in only 10% of the patients	• It lacks the characteristic epidermal changes namely; hyperkeratosis and follicular plugging characteristic of discoid LE; and dermo-epidermal interface changes, which are hallmark of most of the cutaneous forms of LE. • Increased amounts of mucin can be identified on staining with alcian blue • Direct immunofluorescence, shows linear IgG, C3, and fibrin deposits along the basement membrane zone

Jessner's Lymphocytic infiltrate of skin	• Also called benign lymphocytic infiltration of the skin • It is a rare, benign dermatosis with a self-limiting course and an excellent prognosis. • It typically presents as erythematous papules and plaques primarily located on the face, neck, or upper back. • Usually painless and do not itch • The clinical course varies with remissions and exacerbations. • The lesions may persist indefinitely, and some may disappear spontaneously without sequelae.	• No distinct epidermal changes • Dermal infiltrate consists of dense perivascular and periadnexal lymphocytes and plasmacytoid monocytes in reticular dermis • Mucin deposition between collagen bundles may be seen • Immunohistochemistry shows a predominance of T cells, mostly CD4+

Laboratory diagnosis of leishmaniasis can include the following

- Isolation, visualization, and culturing of the parasite from infected tissue.
- Serologic detection of antibodies to recombinant K39 antigen.
- Polymerase chain reaction (PCR) assay for sensitive, rapid diagnosis of Leishmania species.[6]

Other tests that may be considered include the following

- CBC count, coagulation studies, liver function tests, peripheral blood smear.
- Measurements of lipase, amylase, gamma globulin, and albumin.
- Leishmanin (Montenegro) skin testing (LST) (not FDA approved in the United States).

Treatment: Treatment options for CL lesions include[7-9]

Self-healing (simple lesions only)
Topical non-antimonial treatments
Cryotherapy
Heat therapy
Photodynamic therapy
Imiquimod
Topical paromomycin (also known as aminosidine)
Intralesional antimonials
Sodium stibogluconate
Meglumine antimoniate
Non-antimonial systemic therapies
Amphotericin B
Miltefosine
Pentamidine
Azole antifungal drugs: itraconazole, fluconazole, ketoconazole
Zinc sulfate
Allopurinol
Systemic antimonials (intravenous or intramuscular)
Sodium stibogluconate
Meglumine antimoniate.

Prevention

No vaccines or drugs are available to prevent infection. The best way for travelers to prevent infection is to protect themselves from sand fly bites. Personal protective measures include minimizing nocturnal outdoor activities, wearing protective clothing, and applying insect repellent on exposed skin. In general, prevention and control measures must be tailored to the local setting and typically are difficult to sustain. Control measures against sand fly vectors or animal reservoir hosts might be effective in some settings.

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