

Cutaneous leishmaniasis: an unusual cause of nasal obstruction

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Received 7 July 2014

Accepted 11 October 2014

The Egyptian Journal of Otolaryngology

2015, 31:71–72

Nasal and sinus-related illnesses are among the most common reasons for appointments to otolaryngologists and allergists. The usual causes include septal deviation, allergic rhinitis, polyps and so on. Apart from the common cold, allergic rhinitis is probably the most common cause of nasal obstruction. Allergic rhinitis is most common during adolescence and improves significantly during middle age and wanes during old age. The anatomical causes including septal deviation and adenoids constitute other common causes of nasal obstruction. The unusual causes include middle turbinate osteoma, concha bullosa of the inferior turbinate, congenital nasal pyriform aperture stenosis, and congenital inferior turbinate hypertrophy. We describe the case of a 44-year-old man with localized cutaneous leishmaniasis, a hitherto unobserved cause of nasal obstruction, cured with intravenous sodium stibogluconate.

Keywords:

cutaneous leishmaniasis, Leishman–Donovan bodies, nasal obstruction, unusual cause

Egypt J Otolaryngol 31:71–72

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1012-5574

Introduction

Nasal and sinus-related illnesses are among the most common reasons for appointments to otolaryngologists and allergists. Some clinicians consider nasal obstruction to mean a blockage within the nasal cavity; however, nasal obstruction is most commonly defined as a symptom consisting of a sensation of insufficient airflow through the nose [1]. The usual causes include septal deviation, allergic rhinitis, polyps and so on.

We hereby describe the case of a 44-year-old man with localized cutaneous leishmaniasis (LCL) – a hitherto unobserved cause of nasal obstruction.

Case history

A 44-year-old man was sent to the Dermatology Clinic from the Otorhinolaryngology clinic with a complain of progressive blockage and deformity of the left nostril (Fig. 1a).

The swelling, small to start with, had progressively grown in size over the months causing deformity and diminution of the left nasal orifice. There was no history of injury, fever, weight loss, cough, or night sweats. The patient was a school teacher by profession and none in his family suffered from similar illness.

On examination, edema and deformity of the left nare were found. A large (3.5 cm × 2.5 cm) dome-shaped swelling on the left upper lip was visible. The surface of the swelling was smooth, slightly erythematous, nontender, and of normal temperature. The hair was not easily pluckable and there was no local lymphadenopathy.

The left nasal ala was edematous and deformed; the left nasal mucous membrane was also edematous.

Results of routine blood examination and biochemistry were within normal ranges. Skiagram of the head (lateral view and anteroposterior view), chest radiograph, and mantoux readings were within normal limits.

The possibilities of lupus vulgaris, sarcoidosis, and rhinoscleroma were kept in mind before proceeding further.

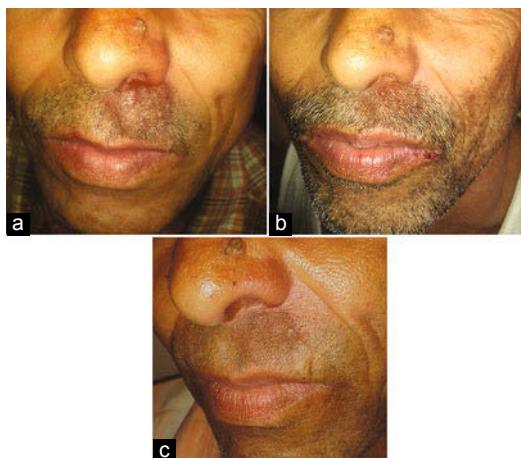
A full-thickness skin punch biopsy was obtained from the edge of the swelling and a part of it was sent for histopathological examination. The other part was used to make a touch preparation, which was air dried and stained with Romanowsky stain. On light microscopy, a mixed inflammatory infiltrate in the dermis (Fig. 2a) consisting of mononuclear cells, macrophages, and plasma cells was seen (Fig. 2b). On oil immersion view of the touch preparation, blue-colored intracellular inclusions identified to be Leishman–Donovan bodies were seen (Fig. 2c).

On the basis of the clinical picture and microscopy findings, a diagnosis of early LCL was made.

The patient was put on intravenous sodium stibogluconate therapy at a dose of 20 mg antimony/kg/day. Within 2 weeks, much of the lump had disappeared (Fig. 1b); by a month, the lump had resolved without scar or pigmentation (Fig. 1c).

Discussion

The affliction of cutaneous leishmaniasis has been known since olden times. Cunningham [2] observed

Figure 1

(a) Patient at presentation. (b) At 2 weeks of treatment. (c) After 1 month of treatment.

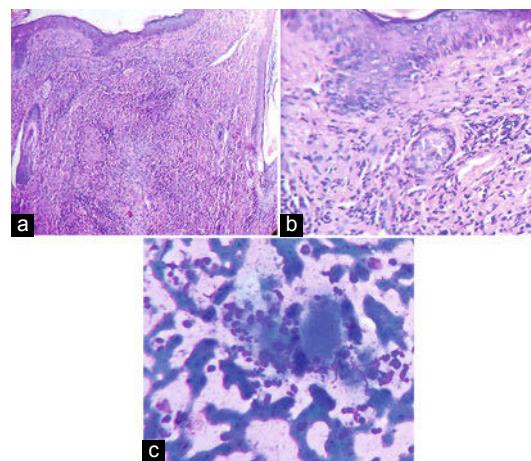
intramacrophage organisms from lesions known as 'Delhi boil'.

Cutaneous leishmaniasis can be classified as localized, diffuse, recidivans, and post-kala-azar varieties, each with different patterns of geographical distribution. The old-world parasites causing LCL are *Leishmania major*, *Leishmania tropica*, *Leishmania aethiopica*, and *Leishmania infantum*. LCL affects mainly unclothed areas of the body, thus affecting the face, neck, and the arms. These areas are easily bitten by the vector, the sand fly. The incubation period ranges from 1 week to 3 months; after which a red papule appears, which enlarges to a plaque or a nodule. Subsequently, this plaque or nodule breaks down to form an ulcer with a violaceous margin [3]. When left untreated, this ulcer regresses spontaneously within 6–12 months.

Inflammation of the nasal mucosa, irrespective of the cause, may induce nasal obstruction; as a result, this symptom is very commonly complained of. Apart from the common cold, allergic rhinitis is probably the most common cause of nasal obstruction [1]. Allergic rhinitis is most common during adolescence, improves significantly during middle age, and wanes during old age. The anatomical causes including septal deviation and adenoids constitute other common causes of nasal obstruction [1].

The *unusual* causes include middle turbinate osteoma [4], concha bullosa of the inferior turbinate [5], congenital nasal pyriform aperture stenosis [6], and congenital inferior turbinate hypertrophy [7].

The management of nasal obstruction begins with determination of the underlying cause. The current patient had presented with nasal obstruction; the swelling on the lip was of little concern to the patient as

Figure 2

(a) Mixed dermal infiltrate ($\times 40$, H and E) (b) same as (a), higher magnification ($\times 100$, H and E). (c) Oil immersion view of touch preparation showing Leishman-Donovan bodies ($\times 1000$, Romanowsky).

it was asymptomatic. The patient had been sent to the dermatology clinic for evaluation of the swelling, on the presumption that the nasal pathology and the lip swelling were of different pathologies. Radiological investigations ruled out bony causes. Biopsy and special stains confirmed cutaneous leishmaniasis as the etiology for both the swelling in front of the nose as well as the edema and blockage of the nasal orifice. This was further confirmed by the resolution of both by a common treatment.

A search of the existing literature revealed no similar instance in the past. This is probably the first case report of nasal obstruction caused by LCL. We intend here to emphasize upon the recognition of the same as one of the causes, although unusual, of nasal obstruction.

Acknowledgements

Conflicts of interest

None declared.

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