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*Case Report*

# Basal cell carcinoma superimposed on a cutaneous leishmaniasis lesion in an immunocompromised patient

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## Abstract

Leishmaniasis is a protozoan infection due to organisms of the genus Leishmania. The differential diagnosis of cutaneous leishmaniasis includes arthropod bites, basal cell carcinoma (BCC) and other malignancies. BCC is the most common form of skin cancer. We present a case of cutaneous leishmaniasis resistant to standard intralesional glucantime injection in an immunocompromised patient, which was proved to be BCC after surgical excision.

**KEYWORDS:** Leishmaniasis, Basal Cell Carcinoma, Glucantime.

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Leishmaniasis is a protozoan infection whose diagnosis should be confirmed by the presence of the organism in dermal macrophages in skin biopsy, dermal scrapings and fine-needle aspirate (FNA). The differential diagnosis of cutaneous leishmaniasis includes arthropod bites, atypical mycobacteriosis, basal cell carcinoma (BCC) and other malignancies. Local therapy consists of excision, laser ablation,[1] cryotherapy,[2] local heat, electrotherapy, and local and intralesional drug administration.[3,4] BCC is the most frequent skin cancer. Sun exposure and anatomic site appear to be important in the etiology of BCC. It may also arise in burn or vaccination scars.

## Case Report

In 2009, a 52-year-old woman presented with a single lesion on her nose, which started as a papule, referred to Sedighe Tahereh Clinic, Isfahan, Iran. The lesion had existed for a period of 14 months and was slowly increasing in size, enlarging to a plaque. The diagnosis of leishmaniasis was confirmed with a positive smear of the lesion showing leishmania bodies about 1 year before. All five members of her family had had a history of proven leishmaniasis. In the past medical history, the patient was a renal failure case since 11 years before and received a renal transplant 4 years after the diagnosis of renal failure. She was also suffering from hypertension and hyperlipidemia. She was receiving oral mycophenolate mofetil (2 g daily) and cyclosporine (100 mg daily). Her skin type was determined as IV in the skin examination. No actinic keratosis was present. A 3×3 cm indurated ulcer with elevated borders was present on the tip of her nose (Figure 1). Her therapeutic plan was intralesional glucantime injection (approximately 1 ml of 1.5 g vial per week, intralesional injection). After completing a therapeutic course of 20 sessions receiving intralesional glucantime injections, she was considered as glucantime therapy resistant. Therefore, surgical excision was advised and performed under local anesthesia. The histopathology was that of a BCC.

## Discussion

The occurrence of malignant neoplasms in sites of scars is an infrequent but well-known phenomenon.[5] Although the coexistence of cuta-

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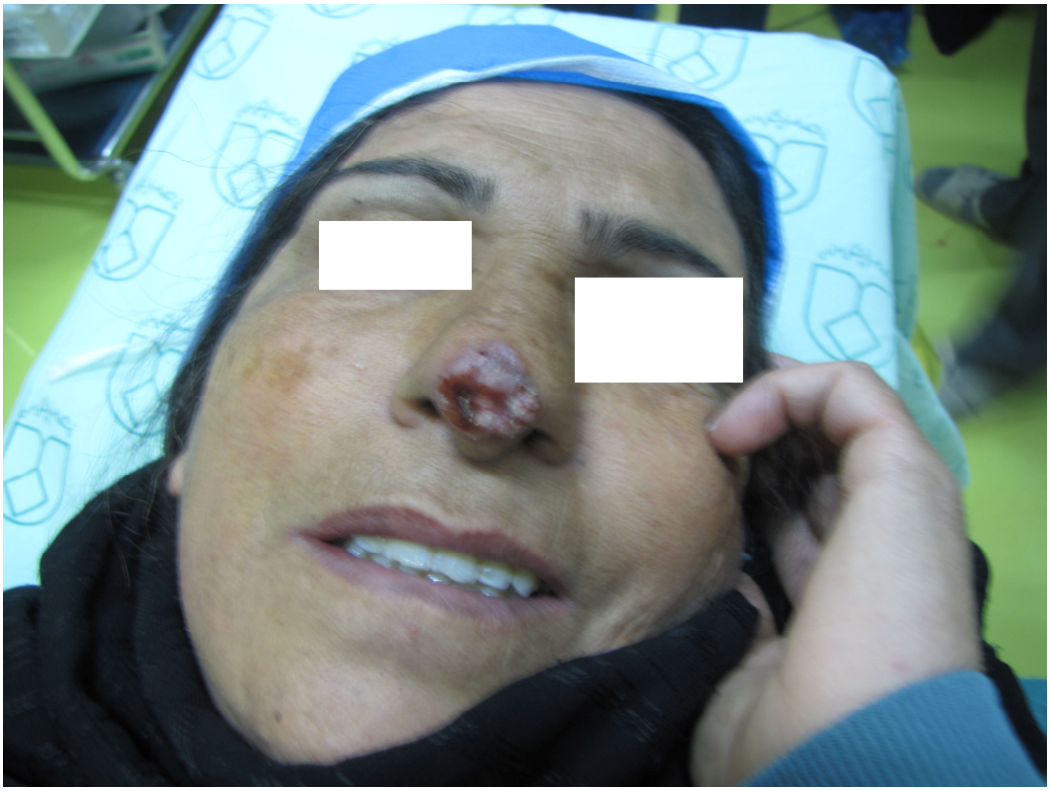

**Figure 1**. Superimposed basal cell carcinoma on a leishmaniasis lesion

neous leishmaniasis and BCC may have been coincidental, some studies suggest that an association between these two entities does exist.[6] Leishmaniasis can directly or indirectly alter the diagnosis and course of different malignancies.[7] There are reports of BCC in chronic leg ulcers.[8] Cases of BCC developing in a Leishmania scar have also been documented,[9] but to our knowledge, cases of both leishmaniasis and BCC in the same site and the same lesion are rare.[10] However, in this case, solid organ transplantation and long term immunosuppressive therapy should be considered as risk factors for malignancy. Advances in effective immunosuppression after organ transplantation have led to increased risk of malignancies, particularly skin cancers [11] including squamous cell carcinoma, basal BCC and malignant melanoma.[12] Thus, malignancies should be considered in the differential diagnosis of leishmaniasis lesions difficult to treat. The possible role of cutaneous leishmaniasis, as a predisposing factor for skin cancer, should also be kept in mind.

## Conflict of Interests

Authors have no conflict of interests.

## Authors' Contributions

AA was the main therapeutic physician and helped write the manuscript. IM and PK contributed in writing the manuscript. All authors have read and approved the content of the manuscript.

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