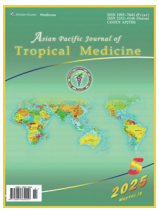

Case Report

Asian Pacific Journal of Tropical Medicine

apjtm.org

doi: 10.4103/apjtm.apjtm_45_25

Mucocutaneous leishmaniasis in Australia: A case report

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ABSTRACT

Rationale: Australia is the only inhabited continent, which is not endemic to leishmaniasis. There are some published articles reporting cutaneous leishmaniasis in travellers, immigrants and refugees. However, mucocutaneous leishmaniasis has not been reported previously from the continent.

Patient concerns: Lesions were present over the nasal septum and the oropharynx of a 34-year-old healthy non-indigenous male. Diagnosis was delayed as it took multiple biopsies as well as extensive discussions in a multidisciplinary team.

Diagnosis: Mucocutaneous leishmaniasis.

Interventions: Liposomal Amphotericin for 20 days.

Outcomes: The patient was symptomatically improved after 3 weeks' treatment.

Lessons: With international travel resuming after the pandemic, it becomes imperative that physicians in Australia are aware of this imported disease and its various presentations.

KEYWORDS: Leishmaniasis; Mucocutaneous leishmaniasis; Australia; Infectious disease

1. Introduction

Australia is the only inhabited continent that is not endemic to leishmaniasis. There are some published articles reporting cutaneous leishmaniasis in travellers, immigrants and refugees. However, mucocutaneous leishmaniasis has not been reported previously from the continent. We present a case of mucocutaneous leishmaniasis affecting a young healthy non-indigenous male.

2. Case report

A 34-year-old, previously fit non-indigenous man presented to the Ear, Nose & Throat Clinic in a regional hospital with recurrent nasal vestibulitis. He denied any other past significant medical history and was treated conservatively. He re-presented a few months later with recurring symptoms in his right nostril. A repeat swab did not grow any organisms. Due to the recurrent nature of nasal vestibulitis with a septal deviation and spur, he was consented and listed for a septoplasty surgery. Meanwhile, he also received a referral to the Drugs and Addiction services of our hospital for intranasal cocaine use which was thought to have contributed to his presentation.

He was seen a year later in the Ear, Nose & Throat Clinic as a pre-operative assessment before his elective septoplasty surgery when he mentioned ongoing dysphagia for 2-3 weeks. His right nasal vestibule had extensive crusting with dried clots and oral cavity revealed an ulceroproliferative lesion involving the uvula, soft palate and the left posterior pillar of tonsil (Figure 1A and 1B). There were no neck nodes and the airway was normal. A biopsy and swab for culture was taken from this lesion and he was also referred to the Infectious Diseases Clinic. At this time, a past history of being treated for cutaneous leishmaniasis (CL) 5 years ago interstate, became apparent. An extensive blood profile for autoimmune

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How to cite this article: Abraham A, Jacob M, Wright C, Culhane B. Mucocutaneous leishmaniasis in Australia: A case report. Asian Pac J Trop Med 2025; 18(5): 236-238.

Article history: Received 17 January 2025 Revision 12 May 2025
Accepted 13 May 2025 Available online 22 May 2025

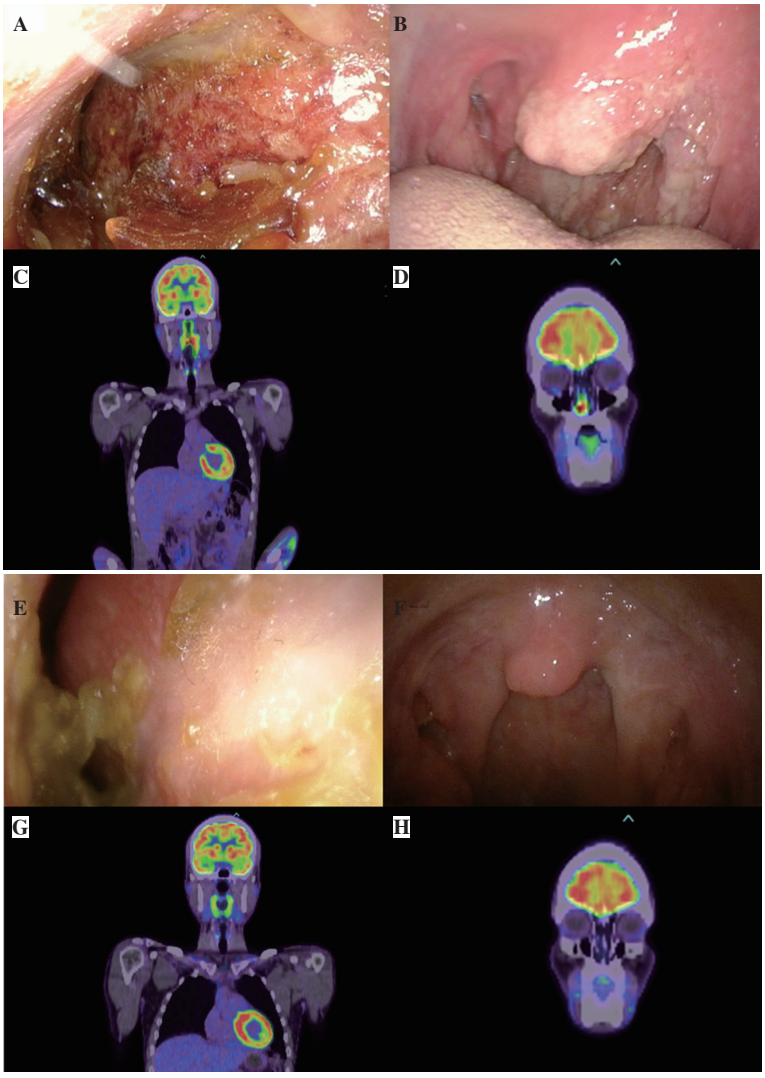

Figure 1. Lesions on the right nasal vestibule and oropharynx (A and B) along with PET scan images (C and D) in a 34-year-old male infected with mucocutaneous leishmaniasis before surgery. Picture of right nasal vestibule and oropharynx (E and F) along with PET scans (G and H) at the 10th week showing complete resolution of the lesions.

disorders, blood borne virus screen and sexually transmitted diseases screen was negative.

The biopsy of the lesion reported nonspecific granuloma and malignancy was ruled out. A possible antineutrophil cytoplasmic antibody negative sarcoidosis was suggested. Meanwhile, a second biopsy of the lesions were taken, this time under general anaesthesia. Tissues from both the oropharynx and nasal septum were sent separately in formalin and as a fresh sample to different pathology lab interstate. Rheumatology consultation suggested a possible cocaine induced midline destructive lesion as a differential. A positron emission tomography scan and another round of extensive blood tests for rare autoimmune disorders was sent for (Figure 1C and 1D).

Infectious diseases repeat review followed a third biopsy of the palatal lesion, which was sent for leishmaniasis PCR and Giemsa staining. Meanwhile, PCR testing on one of the previous biopsies taken from the nasal septum returned as positive for *Leishmania* (*L.*) *braziliensis*. The patient received liposomal Amphotericin for 20 days at 3 mg/kg/day. After 3 weeks, he had symptomatically improved. He had a repeat PET scan and a review at 10 weeks, which showed a complete response to therapy (Figure 1E-1H).

3. Discussion

Australia is the only inhabited continent that is not endemic to leishmaniasis. There are some published articles reporting CL in travellers, immigrants and refugees[1]. However, mucocutaneous leishmaniasis (ML) has not been reported previously from the continent[2–4].

Leishmaniasis is a vector borne protozoal disease transmitted by sandflies and caused by various species, 21 of which cause

infection in humans. The infection occurs in three forms; CL, ML and visceral leishmaniasis (VL). CL affects the skin as ulcers, ML involves the mucosal linings of the nose, oral cavity and larynx and VL affects organs like liver and spleen[5]. VL carries a high rate of morbidity and mortality while ML can lead to significant destruction of oropharyngeal lining and laryngeal cartilage with the possibility of airway compromise. *L. Viannia braziliensis*, *L. amazonensis*, *L. panamensis* and *L. guyanensis* causes localized ML in the New world and *L. infantum* and *L. donovani* in the Old World in cases of immunosuppresion[2].

Leishmaniasis is endemic in parts of the Old World (Europe, Asia and Africa) and the New World (the Americas). Annually 1.2 million cases of CL and 400 000 cases of VL are reported while ML is much rarer[6]. ML can occur several months or years after full clinical resolution of CL. It has been hypothesized that ML occurs due to an altered immune response in the early stages of CL[5]. In Australia, leishmaniasis is an imported disease seen more frequently in recent years due to travel from endemic areas, deployment and immigration. It is so far unreported as a locally acquired human infection although a few cases of CL have been reported in red kangaroos in the Northern Territory. This Australian leishmaniasis is propagated by the bite of the midges unlike the sandfly, which is a known vector for leishmaniasis worldwide[7].

It is pertinent to identify the *Leishmania* species causing infection as response to drug treatment is species dependent. The causative species may be presumed from travel history as species endemicity prevails; however, this may not always be accurate due to geographical overlap of species[8]. Our patient had a history of travel to Bolivia with a 3-month stay in the Amazon jungles 6 years prior, and being treated for CL 1 year later.

The treating clinicians had personally never seen nor encountered this disease previously. With international travel resuming after the pandemic and immigration rates rising, it becomes imperative that physicians in Australia are aware of this imported disease and its various presentations. It is likely that more such cases from travellers may present in future for management.

Conflict of interest statement

The authors declare that they have no conflict of interest.

Ethics approval and consent to participate

Informed consent was obtained from the patient for the publication of this case report and any accompanying images

Funding

The authors received no extramural funding for the study.

Authors' contributions

AA: Conceptualization, data curation, treating clinician, writing-review and editing; MJ: Treating clinician, review and editing; CW: Treating clinician, review and editing; BC: Data curation; review and editing.

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Edited by Pan Y, Lei Y, Zhang Q