CASE REPORT

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Extensive Palatal Necrosis Secondary to *Chrysomya bezziana* Myiasis: A Case Report

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ABSTRACT

Oral myiasis, a rare condition characterised by infestation of dipterous larvae in the oral cavity, poses unique diagnostic and therapeutic challenges. Herein, we present a case study of a 35-year-old quadriplegic man presenting with oral myiasis localised to the palatal region, likely attributable to malnutrition and suboptimal oral hygiene. Manifestations comprised oral bleeding, fever, and necrotic palatal ulcers infested with maggots. Initial diagnostic considerations encompassed mucormycosis and neoplastic lesions; however, further scrutiny revealed primary palatal myiasis caused by *Chrysomya bezziana* species. Therapeutic interventions entailed bedside maggot removal and meticulous wound care, with topical hyaluronic acid application and oral hygiene guidance. This case underscores the significance of uncommon conditions such as palatal myiasis, especially among patients with intricate medical backgrounds. A comprehensive diagnostic approach and optimal treatment ensure favourable outcomes.

Keywords: *Chrysomya bezziana; myiasis; necrosis; oral cavity; palate*

INTRODUCTION

The palate, also known as the 'roof of the mouth', forms the barrier between the nasal and oral cavities. The palate has two distinct regions, the hard palate and the soft palate. The hard palate is immobile and consists of bony structures lined by a thick parakeratinised stratified squamous epithelium. Soft palate, meanwhile, comprises muscle fibres covered by a mucous membrane, and it can be elevated to close the pharyngeal isthmus during swallowing, preventing the food bolus from entering the nasopharynx (Hansen, 2022). It is rich in blood supply from the greater palatine artery bilaterally and the nasopalatine artery centrally. Due to these anatomical properties and functional requirements, it is a robust part of the mouth that is highly resistant to infectious diseases.

Myiasis, a noun derived from Greek (mya, or fly), was first proposed by Hope (1840) to define diseases of humans caused by dipterous larvae, as opposed to those caused by insect larvae in general. Myiasis has since been defined as the infestation of live vertebrates (humans and/or animals) with dipterous larvae (Hall & Wall, 1995). It is commonly reported in rural areas, primarily affecting livestock but also impacting vulnerable humans. In humans, the sites most commonly affected are skin, nose, ears, eyes, anus, vagina, and oral cavity. Oral myiasis is uncommon, and when it occurs, it mostly affects the gingiva (29%), followed by the palate (25%) and the lip (21%) (Dos Passos *et al.*, 2021). Here, we report of a rare case of oral myiasis with palatal mucosal destruction, with details on the clinical features and therapeutic management of such presentation.

CASE PRESENTATION

A 35-year-old Malay gentleman presented to the emergency department with a presentation of bleeding from his oral cavity for the past two days. He was brought in by his parents as he was quadriplegic due to a previous traumatic brain injury secondary to a motor vehicle accident in 2007. Specifically, he sustained bilateral subdural hematoma in the accident. He was also on percutaneous endoscopic gastrostomy (PEG) tube for long-term feeding since the accident. In addition to the complaint of oral bleeding, he also had low-grade fever with chills, and rigours. It was found that his oxygen saturation upon presentation to the emergency department was also low (SpO_2 82% on room air). Written informed consent has been obtained from the patient's parents to publish this paper.

On general examination, it was noted he was cachexic and bed-bound in supine posture with the head tilted slightly upwards, with his mouth persistently open. Facial examination shows no obvious swelling or asymmetry. There was no palpable cervical nodes. Intraoral examination revealed full sets of permanent dentition, but with poor hygiene. In the palatal area, multiple and extensive ulcerations and hematomas were observed (Fig. 1). Parts of the bony hard palate were exposed due to the ulceration and necrosis of the palatal mucosa. In the depth of the ulceration, a significant number of maggots were present. The wound edges were necrotic and friable, without active bleeding or pus. The multiple palatal ulcers were connected by burrows and tunnels between them.

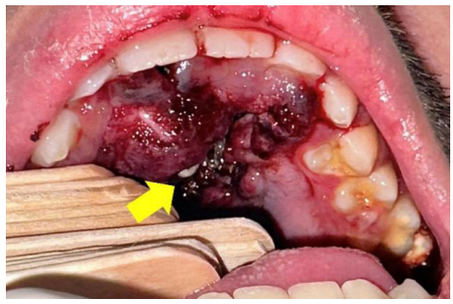

Fig. 1 Extensive palatal hematoma with maggot infestation (yellow arrow) and burrowing beneath the palatal mucoperiosteum was observed. No pus discharge was noted from the lesion.

Blood investigation showed an increase in the C-reactive protein (CRP) of 17.23 mg/dl and slightly lower albumin and creatinine levels of 33 g/l and 41 μmol/l, respectively. Otherwise, both his haemoglobin and white blood cell count readings were within the normal range of 15.2 g/dl and 4.5×10^9/l, respectively. At this point, the two initial differential diagnoses for the palatal ulcer were palatal necrosis secondary to mucormycosis and palatal neoplastic lesion. He was admitted and referred to the respiratory physician due to the low oxygen saturation. Based on clinical auscultation findings and consolidation observed in the chest radiograph, orthostatic pneumonia was suspected, and intravenous Co-amoxiclav, oral Azithromycin, and oral Bromhexine were initiated. Oxygen therapy was started, and oxygen saturation of 98% was achieved. He was also started on intravenous Amphotericin B once a day, in view of the working diagnosis of palatal mucormycosis.

Non-enhanced computed tomography (CT) scan was performed on the same day to assess the extension and bony involvement of the lesion (Figs. 2 & 3). It showed intact bone with no associated bone erosion. There was a mixed soft tissue thickening and destruction at the hard palate area, extending to the soft palate, with the presence of air locules.

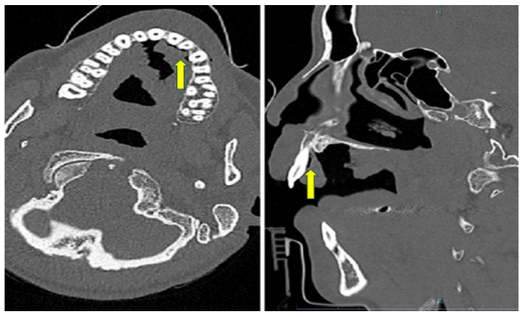

Fig. 2 Axial and sagittal views of the non-enhanced CT revealed soft tissue thickening with breaching of the palatal mucosa. No associated bone erosion or sclerosis was observed. Air locules (yellow arrows) were noted in the anterior part of the soft tissue thickening.

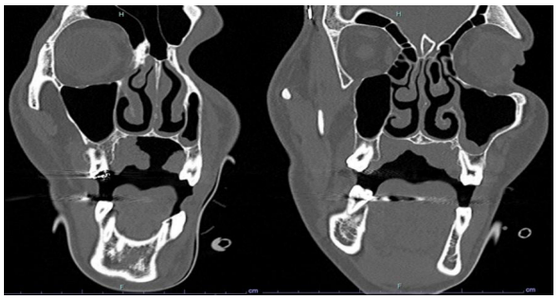

Fig. 3 The coronal view of the non-enhanced CT showed a combination of soft tissue thickening and palatal mucosal destruction. The maxillary and nasal floors remained intact.

Bedside maggots' removal, oral toilet were performed twice daily from the first presentation. A total of 32 maggots were removed on the first day. On the second admission day, an incisional biopsy was done under general anaesthesia, where multiple tissue samples were taken from the necrotic wound tissues over the palatal mucosa. Samples were sent for histopathological examination (HPE) and also for bacterial and fungal tissue cultures. A further total number of 9 maggots was also able to be removed. These collected maggots were placed in a 70% alcohol solution and sent for microscopic identification.

The patient was stable on day 3 and was kept in the ward for close monitoring and intravenous antimicrobial treatment. A total number of 16 maggots on day 3; followed by another 18 maggots on day 4 were identified and removed (Fig. 4). On day 5, an additional 8 maggots were removed. After six days of daily oral toilet, no more maggots were retrieved from the palatal wound. The palatal mucosa necrosis worsened exposing approximately 4 cm × 4 cm of the underlying hard palate (Fig. 5). The tissue culture and sensitivity (C&S) test showed a mixed growth of three different types of organism (2 gram negative rods, 1 gram positive cocci) with no predominant colony. The blood C&S test indicated there was no anaerobic and aerobic growth.

Fig. 4 Collection of maggots within specimen bottles.

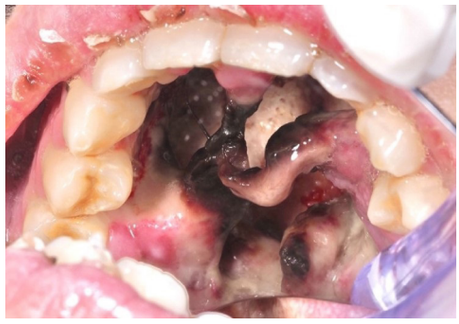

Fig. 5 A crater-like necrotic palatal wound with exposure of the underlying hard palate was observed, along with soft tissue thickening but no signs of bleeding or pus discharge. By day 6, no further maggot infestation or burrowing was noted beneath the palatal mucoperiosteum.

On day 8 of admission, the fungal culture reported that no fungi were isolated from the wound (bacterial growth only). The IV Amphotericin B was then discontinued. Malignancy of the palate was ruled out, as the HPE revealed necrotic tissue with acute and chronic inflammation, along with parasitic infestation, but no evidence of malignancy. These results confirmed the diagnosis of oral myiasis.

Granulating tissue was observed on the palatal wound two weeks after the admission (Fig. 6). Both the C-reactive protein and white blood cell count levels showed a reducing trend from baseline to day 16 of admission, with readings of 0.38 mg/dl for C-reactive protein and 3.8 × 10^9/l for white blood cells, respectively.

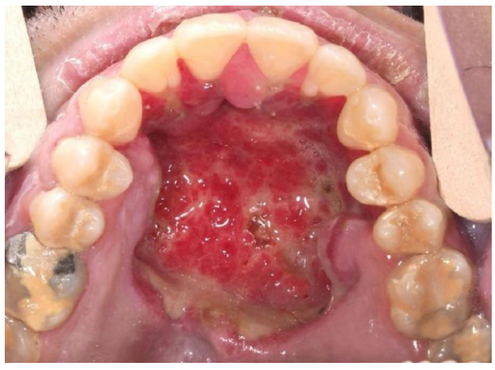

Fig. 6 Palatal wound after 2 weeks (day 14) of daily wound toilet with 0.12% chlorhexidine gluconate and mechanical removal of maggots.

The patient was discharged home after 18 days. Topical hyaluronic acid 0.2% spray (GENGIGEL®) was started, to be applied twice a day. The caretaker was educated on the proper method of application over the palatal granulation tissue, and reinforcement of oral hygiene was given. Review appointment was given, and upon review, one month since being admitted, the palatal wound bed showed progressive healing of palatal mucosa (Fig. 7).

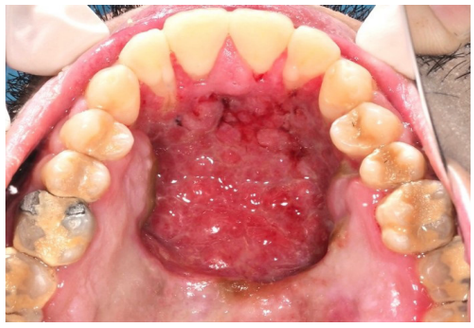

Fig. 7 On review after one month (day 30), new palatal mucosal tissue was observed over the palatal wound bed.

The results of the microscopic examination of the maggots found that the maggots were of *Chrysomya bezziana* species (Figs. 8 & 9). Based on this finding, along with the previous results from the HPE, C&S, and imaging studies, the final diagnosis was primary palatal myiasis, likely due to malnutrition coupled with poor oral hygiene.

Fig. 8 *C. bezziana* third instar larva. The third instar is approximately 10 mm in length with strong mouth hooks (green arrow) and a prominent segmental black band.

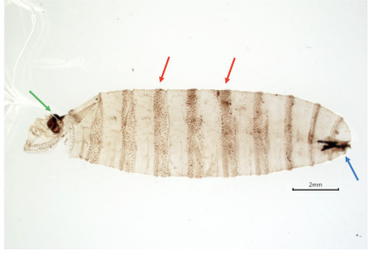

Fig. 9 *C. bezziana* third instar larva. Prominent black cephalopharyngeal skeletons (blue arrow) and posterior spiracles (green arrow). The heavy bands of dark, robust, thornlike spines (red arrow) are very prominent.

DISCUSSION

Palatal ulceration is rather common and can occur from diverse potential causes such as inflammation, infection, neoplasms, drug-related, iatrogenic, and traumatic events. Infectious causes of palatal ulceration are wide-ranging, from leprosy, tertiary syphilis, tuberculosis, naso-oral blastomycosis, actinomycosis, histoplasmosis, coccidioidomycosis, and diphtheria (Saroch & Pannu, 2016). Deep fungal infections are especially associated with extensive tissue destruction, including mucosal necrosis and ulcerations. Mucormycosis, especially, has been reported previously with palatal involvement as the initial presentation (Mora-Martínez *et al.*, 2023). The rhinocerebral form of mucormycosis typically involves the hard palate, as fungal spores inhaled through the nose can infect the maxillary sinus directly. Common oral manifestations include exposed bone, ulcerations, halitosis, purulent discharge, gingival swelling, and periodontitis (Mora-Martínez *et al.*, 2023). When treating with any deep, severe fungal infection, amphotericin B is usually the first line of treatment (Mora-Martínez *et al.*, 2023). The clinical utility of Amphotericin B has been significantly limited by the high frequency of infusion-related reactions, such as fever, chills, and nausea, and by the dose-limiting nephrotoxicity associated with the conventional formulation containing deoxycholate (Cavell, 2020). This formulation can cause kidney damage, requiring close monitoring of renal function and often limiting its use to severe, life-threatening fungal infections. Alternative formulations, like lipid-based formulations (e.g., liposomal Amphotericin B), have been developed to reduce these side effects, especially nephrotoxicity, but they are generally more expensive (Groll *et al.*, 2019).

Inflammation with ulceration can also result from physical (i.e., faulty denture and orotracheal tube) or chemical traumatic insults (i.e., sodium hypochlorite solution) which can lead to foreign material penetrating the deeper tissues of the oral (Gursoy *et al.*, 2006; Jain *et al.*, 2009; Bartlett *et al.*, 2013; Pinheiro *et al.*, 2017). Besides superficial causes of palatal ulceration, underlying bone pathology should also be considered and included in the differential diagnosis, especially when associated with exposure of the underlying hard palate. Common causes such as osteomyelitis and osteonecrosis of the jaws can be ruled out from detailed history taking and clinical findings (Pauli *et al.*, 2021). Cancer of the oral cavity should also be considered, especially when there are risk factors such as smoking or alcohol abuse. The most common neoplasms of the hard palate are minor salivary gland tumours and squamous cell carcinoma for the soft palate (Abu Rass *et al.*, 2018). Leukaemia in particular commonly causes oral ulceration secondary to neutropenia and may be accompanied by other oral symptoms such as gingival bleeding and/or inflamed boggy gingiva (Francisconi *et al.*, 2016). In this reported case, bony pathology was ruled out by the findings from the CT scan. The final diagnosis was only confirmed after the exclusion of any neoplasm or fungal/bacterial causes from the HPE and microbial culture results.

The incidence of oral myiasis is relatively rare compared to cutaneous myiasis, as the oral mucosa is not exposed to the external environment. A recent global review on oral myiasis reported that India (41%) had the most cases, followed by Brazil (29.5%) (Dos Passos *et al.*, 2021). Most of the oral myiasis cases occur over the gingiva and palate, specifically the anterior portion of it (Ramli & Rahman, 2002; Sharma *et al.*, 2015; Hassan *et al.*, 2019; Nath & Pulikkotil, 2019). A total of 13 fly species have been associated with oral myiasis. The most frequent species was *Cochlyomia hominivorax*, responsible for 35 reported cases. *Chrysomya bezziana* was also identified in 24 reports and *Musca domestica* in 17 (Dos Passos *et al.*, 2021). The factors associated with this condition were poor hygiene, neurological disabilities, residence in rural or subtropical

areas, absence of lip sealing and/or mouth breathing, bedridden or locomotor disabilities, previous medical comorbidities, periodontal disease, low socioeconomic status, previous injuries/trauma, halitosis, malnutrition, drug use and/or alcoholism and homelessness (Dos Passos *et al.*, 2021). The predisposing factors for oral myiasis are divided into local, systemic and environmental factors (Hassona *et al.*, 2014). The local factors includes poor oral hygiene, periodontal diseases, suppurating lesions, mouth breathing, anterior open bite, lip incompetence, thumb-sucking habit, halitosis, and trauma. Systemic factors such as mental impairment, cerebral palsy, epilepsy, neurological deficits, poor general hygiene, alcoholism, mental health issues, senility, and breastfeeding from mother with breast myiasis can also predispose an individual for oral myiasis. Finally, the examples of environmental factors are poor food hygiene, low socioeconomic status, humid, warm weather, and travel to endemic areas. The reported case here had several risk factors such as malnutrition, poor oral hygiene, bedridden with neurological disability and persistence of mouth opening. Furthermore, being in Malaysia with its warm tropical climate, there is a wide variety of flies in the order Diptera. Previous local reported cases of myiasis were also associated with mentally or physically disabled patients with locomotor or neurological deficit, and the involved sites were the anterior palate (Ramli & Rahman, 2002; Hassan *et al.*, 2019).

The species identified in this patient is *Chrysomya bezziana* based on its morphological features. *C. bezziana* transmission usually happens when the eggs are laid on the edges of wounds or in body orifices in masses of up to 250. The eggs are cemented tightly together like a shingled roof and hatch in approximately 12 to 14 hours (10.5 hours at 37°C) (Animal Health Australia, 2017). After hatching, the larvae undergo three stages of development as they feed on blood and wound exudates, and aggressively destroy the living tissues (Zhou *et al.*, 2019). The first-stage larvae start feeding on wound fluids on the tissue surface. Within 24 hours of hatching, the larvae then penetrate the wound and exuviate into the second stage. The larvae enter their third and final stage approximately two days after hatching. There are three instar stages in the development of *C. bezziana* larvae. The first two instars each occupy one day, while the third and final instar lasts three to five days. Larval development occurs over five to eight days. Most have evacuated the wound after seven days of feeding and fall to the ground where they pupate and later emerge as adults (Animal Health Australia, 2017). The clinical presentation seen correlates to the developmental stages of the larvae as they feed on the host. The larvae initially destroy living tissues, causing a presentation of deep ulcerative lesions with bleeding and a serosanguinous purulent discharge. Following this, secondary infections may set in, causing fever and inflammation as seen in this reported case. Further infestation leads to enormous soft tissue destruction and wound extension. The larvae can destroy bones, nasal sinuses, orbital cavities, hard palate, eyeballs, hearing apparatus, and teeth (Zhou *et al.*, 2019).

A review of myiasis in humans described myiasis into two possible classifications, namely the anatomical and ecological classifications (Francesconi & Lupi, 2012). Ecological classification considers the level of parasitism of the parasite and the host. It is divided into obligatory and facultative types. Parasites in the obligatory species are dependent on the host for part of their life cycle, thus causing myiasis. The larvae must infest living tissue in order to complete their life cycle. For facultative types, the larvae normally feed on decaying organic matter, such as dead animals or decomposing tissue. The larvae of *C. bezziana* are obligatory wound parasites, which typically specialise to infest living tissue (Singh & Singh, 2015). This may result in deeper penetration and potentially more severe tissue damage, which leads to the removal of *C. bezziana* larvae from living tissue becoming more

challenging. Early and proper treatment is essential to help wound healing and stop the rapid and destructive process of *C. bezziana* myiasis (Zhou *et al.*, 2019). This explained the extensive destruction of the palatal mucosa in this case and also justified the need for surgical wound debridement.

An anatomical classification of myiasis is based on the specific location within the body where the infestation occurs. These include dermal myiasis, subdermal myiasis, wound myiasis, cavitary myiasis, accidental myiasis, nasopharyngeal myiasis, ophthalmic myiasis, and aural myiasis. The case reported here falls under the 'cavity myiasis' as the maggots infest the oral cavity, which is a natural body cavity that can also occur in the nose or ears. Several ways of myiasis transmission have been proposed. Some flies can deposit their maggots in, on, or near a wound. After hatching, the maggots develop and burrow into oral tissues, causing progressive damage (Kamboj *et al.*, 2007). With the condition of the patient in this reported case, it is not surprising for flies to gain access to the oral cavity to initiate the transmission of the disease.

Treatment of oral myiasis comprises a combination of local and systemic measures with no specific treatment modalities. Systemic measures include broad-spectrum antibiotics such as penicillin, metronidazole, clindamycin, amoxycillin with clavulanic acid (Antunes *et al.*, 2011). Ivermectin regimes and other anthelmintics such as albendazole administered by different routes were indicated in several cases reported as well (Shinohara *et al.*, 2004; Sharma & Hedge, 2010; Ashour, 2019). Ivermectin is a broad-spectrum antiparasitic medication commonly used to treat various parasitic infections by killing the larvae causing the infestation. It works systemically to disrupt the parasites' nervous system, often combined with mechanical removal of the larvae for effective treatment. Its use in oral myiasis would generally be considered in severe cases or when larvae are widespread (Ashour, 2019). Local measure involves mechanical removal of maggots. The mechanical removal alone can be insufficient as it does not allow the complete removal of maggots because it uses its hook to grip the tissues and it buries deeper in the tissues. Hence, a number of asphyxiating agents have been suggested as a method to force the maggots out of the host tissue. Among these substances, creoline, turpentine oil, and lidocaine spray were used (Antunes *et al.*, 2011). Turpentine is a volatile mixture of hydrocarbon isomers. Turpentine is an irritant to the skin and mucous membrane and can also cause skin sensitisation and affect the central nervous system, gastrointestinal system, and the urinary system. Ingestion of turpentine causes a burning pain in the mouth and throat, nausea, vomiting, diarrhoea, abdominal pain, excitement, ataxia, confusion, stupor, seizures, fever and tachycardia, and may also cause death due to respiratory failure (Gopalakrishnan *et al.*, 2008). In this case, due to the location of the myiasis being within the mouth and the condition of the patient, we decided that turpentine was not suitable to be used.

Instead, chlorhexidine gluconate irrigation solution was used. Chlorhexidine is a relatively milder form of solution choice, and its use has been proven to be effective in many cases (Saha *et al.*, 2017; da Câmara *et al.*, 2023; Singh *et al.*, 2023). In this case, it plays a supportive role in managing myiasis by acting as an effective antiseptic to disinfect wounds, reduce bacterial load, and minimise the risk of secondary infection. While it does not directly kill larvae, its ability to clean the affected area and remove necrotic tissue facilitates the mechanical removal of larvae. From our experience in managing this case, we found that daily wound irrigation with it to be effective in compelling out the maggots from the tissues, including those deeply buried underneath. Together with repeated mechanical removal of the maggots, the wound became maggots-free within six days while allowing proper healing of the tissue bed. Once the wound bed was clear of maggots, application of hyaluronic acid was used in this case to hasten healing.

Hyaluronic acid has been shown to promote oral mucosal healing, reduce inflammation, and provide relief from discomfort by forming a protective layer over damaged tissue (De Lauretis *et al*., 2024).

CONCLUSION

This case report highlights the rare occurrence of oral myiasis with palatal mucosal destruction due to infestations of *Chrysomya bezziana*. In this case, the *C. bezziana* maggots caused serious but reversible tissue damage over the posterior palate. Oral myiasis is prone to occur among people with mental and physical disability, with immobility and poor manual dexterity, which causes difficulties in maintaining good oral hygiene. A step-by-step investigative process is required to achieve a diagnosis, following which a combination of local and systemic measures is needed for successful management.

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