Plasma cell mucositis related to qat chewing: a report of 2 cases and review of the literature

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Plasma cell mucositis is a rare, benign mucosal condition with characteristic histological features of a dense polyclonal plasmacytic infiltrate. A variety of mucosal sites are affected and the presentation varies from a cobblestone to an intensely erythematous, lobulated appearance. Idiopathic cases are well documented and it has been attempted to define the entity as a hypersensitivity reaction, however reports show inconsistencies. The last two decades have highlighted an emerging association between qat (khat) chewing and plasma cell mucositis. This report provides a review of the most pertinent literature and describes two cases intimately related to qat chewing, whereby resolution occurred upon qat cessation. One case requiring systemic steroidal therapy due to severe symptoms. This highlights the need for an increased awareness amongst clinicians of a potential aetiological link between qat and plasma cell mucositis, emphasises the benefit of qat cessation and the scenarios whereby systemic steroidal therapy should be considered. (Oral Surg Oral Med Oral Pathol Oral Radiol 2021;131:e65–e70)

Plasma cell mucositis is a rare, benign condition of unknown aetiology with a hallmark histologic appearance of a dense polyclonal plasma cell infiltrate within the underlying lamina propria of mucosal tissue.[1] This histologic appearance was first reported by Zoon[1] in 1952 as affecting the glans penis and was described as Zoon's balanitis. Since 1952, similar pathologic changes have been seen at various mucosal sites, although simultaneous lesions affecting the genital mucosa and mucosa of the upper aerodigestive tract remain rarely reported.[2] Numerous terminologies have been used to document these clinicopathologic presentations, including plasma cell mucositis, plasma cell orificial mucositis, and mucous membrane plasmacytosis.[3,4] The term *plasma cell mucositis* has been suggested to improve the consistency of documentation of this proliferative polyclonal plasma cell infiltrate.[5] In this article, we describe 2 patients with plasma cell mucositis of the upper aerodigestive tract related to qat chewing who experienced resolution of symptoms upon cessation, and we review the pertinent literature in regard to other reported cases associated with qat.

Qat (khat), also known as *Catha edulis*, is a plant native to the eastern peninsula of Africa and Arabia. In these parts of the world, the plant is chewed fresh as part of cultural practice. Its chemical composition, including cathinone and cathine, produces stimulant and euphoric side effects.

Epidemiologically, plasma cell mucositis has a slight male predilection, with a male-to-female ratio of 1.2:1.[3] The initial average age of presentation is 56.6 years old.[3] Clinically, the appearance varies; intraorally, many sites can be affected, including the buccal and labial mucosae, tongue, hard and soft palate, and gingiva. Lesions can vary from a warty, cobblestone appearance[6] to an intensely erythematous, lobulated appearance.[5] Oral lesions often cause discomfort and pain, whilst lesions affecting the oropharynx and larynx can lead to symptoms of dysphonia and dysphagia. Symptoms at their most severe can lead to airway obstruction requiring tracheostomy.[7]

The aetiology of plasma cell mucositis is poorly understood and is defined by its histopathologic presentation of a dense, polyclonal, plasma cell infiltrate within the underlying connective tissue of mucosal epithelium.[4] The clinical differential diagnosis varies dependent on the clinical site and presentation. Previous reports have suggested this includes vesiculobullous disorders, oral lichen planus, fungal infections, sarcoidosis, granulomatosis with polyangiitis, lymphoproliferative disorders, dysplasia, and squamous cell carcinoma.[3,8,9] The diagnosis is ascertained via histopathologic examination and haematologic testing, including a full blood count and immunoglobulin levels, to help identify any causative underlying conditions. The histopathologic differential diagnosis includes multiple myeloma, plasmacytomas, and immunoglobulin G4 (IgG4)-related diseases. Further immunohistochemical testing, including kappa and lambda staining, is done to ensure plasma cell polyclonality.

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Attempts have been made to identify any underlying hypersensitivity as the cause of plasma cell mucositis. The oral presentation of this condition has been linked to allergies and sensitivities, including chewing gum, mints, dentifrices, and food substances, including chillies and cinnamon.[10-14] However, reports have shown inconsistencies with allergen testing, and many cases appear to have no easily identifiable cause.[15,16]

Since 2002, there have been 10 previously reported cases of plasma cell mucositis linked to qat chewing with evidence that cessation of qat results in a dramatic improvement of symptoms and clinical presentation.[17-19]

Here, we report 2 cases of plasma cell mucositis linked to qat chewing in which cessation of qat chewing resulted in an improvement of clinical symptoms. These cases are of importance because they highlight an emerging potential causative factor of plasma cell mucositis. The objective of this report is to discuss the link between qat chewing and the development of plasma cell mucositis in these cases, along with discussion and review of the current literature.

CASE REPORT 1

A 47-year-old man of Somali origin presented to an oral medicine clinic. He was referred by the ear, nose, and throat team. His main complaints consisted of a burning mouth sensation with recurrent bleeding and crusting of the lips, dysphonia, and dysphagia symptoms that had occurred on and off for many years. He was using a hydrocortisone cream with fusidic acid, which provided some relief, and had previously used 400-μg fluticasone nasule drops as a mouthrinse but had ceased using this. Furthermore, he reported recent weight loss, which he linked to decreased appetite as a result of his oral symptoms. He reported no other mucocutaneous lesions.

In regard to his medical history, he had schizophrenia, took 10-mg olanzapine and 30-mg mirtazapine once daily, and had no known drug allergies. He gave a varying smoking history of 3 or 4 cigarettes on alternate days and admitted to chewing qat for a minimum of 4 years several times per week.

Extraoral examination revealed a marked haemorrhagic, ulcerative appearance of the lips with erythema and crusting extending to the vermillion border. Ulceration was shallow, soft, not indurated, and of varying size. Intraoral examination showed widespread erythema, desquamation, and cobblestoning of the buccal mucosa and hard and soft palate with presence of generalised gingival inflammation. The lesions were soft on palpation and chronic in nature. He had a markedly fissured and lobulated tongue that bled easily (Figure 1).

Baseline blood tests were performed, including a full blood count, haematinics, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), immunoglobulin assay, complement, and angiotensin-converting enzyme. Blood tests revealed iron deficiency anaemia, raised white blood cell count (10.1×10^9 /dL), platelet count (578,000 /dL), and neutrophil count (7.8×10^9 /dL) with a polyclonal increase of IgG at 22.3 g/L (normal range, 6-16 g/L) and immunoglobulin A (IgA) at 4.42 g/L (normal range, 0.8-4.0 g/L) with normal immunoglobulin M (IgM) and raised ESR (45 mm/hr) and CRP (12 mg/L).

Several biopsies were taken from various mucosal sites, including the buccal mucosa, hard palatal mucosa, and tongue. A histologic specimen was submitted for haematoxylin and eosin and direct immunofluorescence staining. Microscopic examination revealed a dense chronic inflammatory infiltrate dominated by plasma cells throughout the underlying connective tissue (Figure 2). Immunohistochemistry for kappa and lambda light chains showed a polyclonal plasma cell population, negative staining for herpes simplex virus, and a normal total IgG4/IgG ratio. Direct immunofluorescence showed no convincing evidence of vesiculobullous disorder with strong positivity for fibrinogen and equivocal deposition of IgA along the basement membrane but negative results for C3, IgG, and IgM. Clinicopathologic correlation was consistent with the diagnosis of plasma cell mucositis.

Initial management involved qat and smoking cessation together with steroid and analgesic topical preparations; 400-μg fluticasone nasule drops as a mouthrinse 3 times daily, hydrocortisone cream with fusidic acid to be applied to the lips once daily, and 0.5% benzydamine hydrochloride mouthrinse as required. The patient's general practitioner prescribed iron supplementation to manage the iron deficiency anaemia.

At a 3-month review, the steroid topical preparations were found to have produced little effect. However, for a brief period, the patient managed to successfully cease qat chewing, and his symptoms drastically improved. Upon chewing qat again, the patient found that his symptoms worsened, returning to levels causing difficulty eating and drinking. The patient was unreliable in returning for follow-up visits; however, he was re-referred on several occasions. Most recently, further management involved further qat cessation and an 8-week reducing course of prednisolone starting at 20 mg and reduced by 5-mg increments every 2 weeks, which resulted in marked improvement of clinical appearance and symptoms. The patient has since not attended a further follow-up appointment.

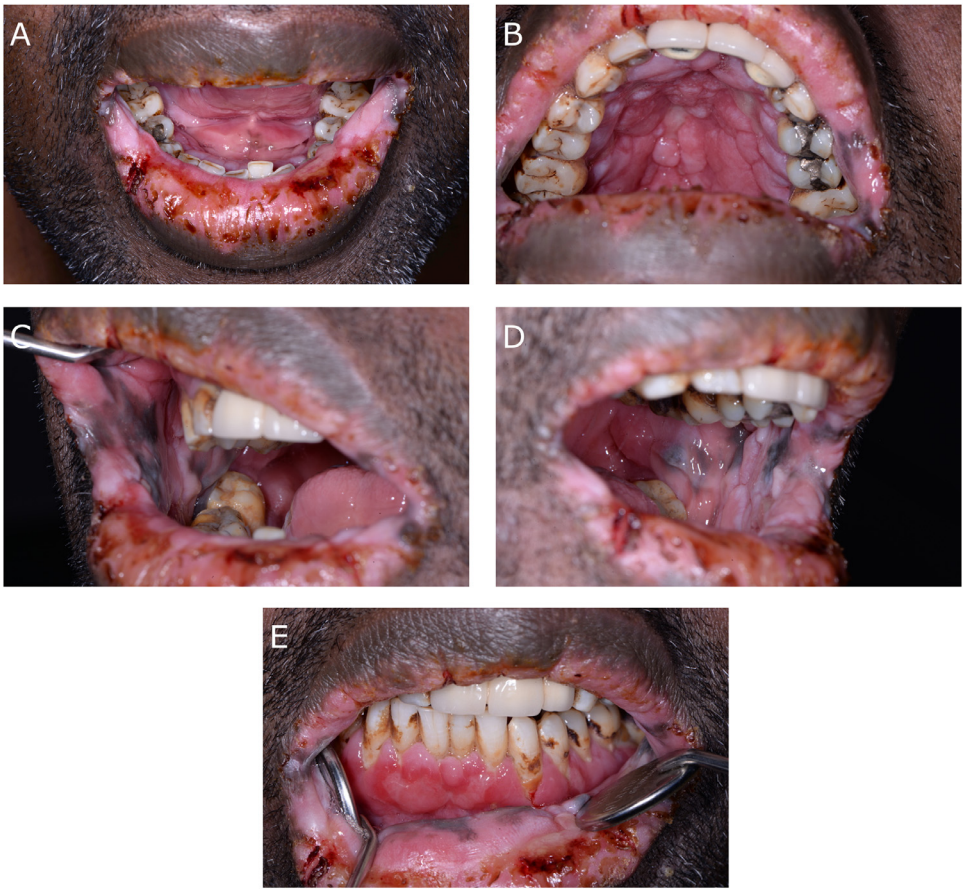

Fig. 1. (A–E) Case report 1. Across all 5 images, a marked haemorrhagic ulcerative appearance of the lips was seen with widespread intraoral cobblestoning of mucosa with ulceration, erythema, desquamation, and gingival overgrowth.

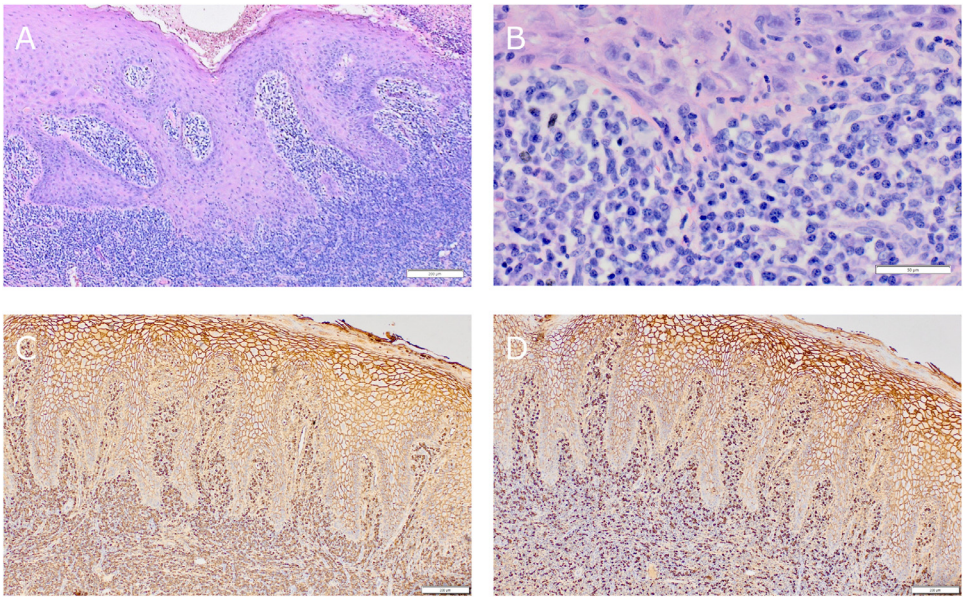

Fig. 2. (A) Oral mucosa exhibiting ulceration (top right) and a dense plasma cell infiltrate in the underlying connective tissue (haematoxylin and eosin [H&E] stain; original magnification, × 40). (B) Epithelium exhibits spongiosis and neutrophilic exocytosis and dense plasma cell infiltrate without atypia (H&E stain; original magnification, × 100). (C and D) Kappa (C) and (D) lambda studies showing positivity within plasma cells in a 2:1 ratio (in situ hybridisation kappa and lambda light chains; original magnification, × 40).

CASE REPORT 2

A 39-year-old man of Somali origin presented to an oral medicine clinic with a complaint of a sore mouth. He reported a 1-year history of oral soreness with ulceration. The soreness was felt throughout his mouth and throat and was worse with eating. Over the course of 1 year, he had lost 12.7 kg in weight, and he avoided spicy food and ate a soft, cool diet. He had no previous history of oral lesions or any cutaneous, genital, or ocular complaints. His medical history was unremarkable; he was fit and well, had no known allergies, and was not receiving any medication. He did not smoke or drink alcohol. He had come to live in the United Kingdom at age 10 years but had since had periods of time living in Asia, the United States, and the Middle East. He had recently been living in Somalia.

Extraoral examination revealed no abnormal clinical signs. Intraoral examination showed widespread, velvety-textured, erythematous mucosa on the maxillary and mandibular gingivae and the hard and soft palate. Superficial ulceration was present on the left maxillary buccal gingivae. No abnormal clinical signs were visible in the buccal mucosa, tongue, or floor of the mouth.

A swab was taken from the palate for culture and sensitivity testing, together with a full blood count, haematinics, random blood sugar, and immunology screening, including antinuclear antibodies and extractable nuclear antigen and an immunoglobulin assay. All results were negative or normal except a raised IgG at 25 g/L (normal range, 5.9-15.6 g/L) and raised IgA at 5.5 g/L (normal range, 0.6-5.0 g/L). Electrophoresis showed polyclonal immunoglobulins. An incisional biopsy was taken from the left maxillary buccal gingiva. Part of this biopsy sample was not fixed and was submitted fresh for direct immunofluorescence. The histopathology showed an oral mucosa with oedematous epithelium and an intense chronic inflammatory infiltration throughout the lamina propria, dominated by mature plasma cells. Direct immunofluorescence was negative for basement membrane and intraepithelial binding of IgG, IgA, IgM, and C3. Staining results for fungi and acid-alcohol-fast bacilli were negative. The appearance was consistent with plasma cell mucositis. Benzydamine hydrochloride 0.15% mouthwash was prescribed to be used on an as-needed basis for pain relief, and review was arranged.

The patient failed to attend review appointments for the next 3 months. Upon his return to the clinic, he told us he had been to Somalia, and, whilst there over Ramadan, his mouth had improved. Upon further questioning, he admitted to chewing qat on a once-daily basis for the past year, originally whilst living in Somalia and continuing to do so upon moving to the United Kingdom. During Ramadan, whilst in Somalia, he had stopped chewing qat. Upon his return to the United Kingdom, he had resumed qat use up to 3 times daily, and his mouth had once more become sore. Upon examination, the appearance of his oral mucosa was similar to what had previously been seen. He was asked to stop chewing qat completely. Upon review 1 month later, he reported that he had stopped using qat and that he had noticed a significant improvement in his oral soreness. He reported being able to eat a much wider diet than previously, without discomfort. Upon examination, a much less florid appearance of the oral lesions was noted with resolution of all previous ulceration. The patient was advised to continue to desist from using qat and requested to return for review. He failed to return for review in the clinic thereafter.

DISCUSSION

Here, we describe 2 cases of plasma cell mucositis linked with qat chewing. These 2 patients both are of Somali ethnic origin. Qat chewing is very common in Somalia and has been recognised as a national problem due to the adverse effects on individuals' health and the socio-economic development of the country.[20]

The qat leaf (*Catha edulis*) contains the chemicals cathinone and cathine, which produce a euphoric sensation similar to a mild amphetamine.[21] The leaves and stalks are chewed fresh as part of cultural practice and are held in the mouth for up to several hours.[22] The literature suggests qat is not often chewed with other substances, unlike betel quid, which can be chewed with many other substances, such as tobacco, spices, and slaked lime. Therefore, we consider the leaf itself as being a likely candidate for the plasma cell mucositis in our cases.

In 1980, the World Health Organisation classified qat as a drug of abuse that can produce psychological dependence. Until 2014, qat was a legal substance in the United Kingdom. Airfreight enabled the fresh and legal transportation of the leaf and allowed immigrant populations to continue their cultural and social practices. This contributed to the expansion of the qat production industry and in conjunction triggered international health concerns due to qat's amphetamine-like effects. In 2014, the United Kingdom joined many countries in labelling qat a class C drug, which makes it illegal to possess or traffic it. The criminalisation and banned importation of qat across the world has impacted the qat economy negatively with concerns that this may result in emerging organised crime and trafficking networks.[23] This highlights important economic and health impacts that the class C drug holds and which clinicians should be aware of.

In regard to the 2 cases of plasma cell mucositis described here, similar features are documented in the previous 10 case reports linked to qat chewing. The findings are summarised in Table I.[17-19] Firstly, the

Table I. Summary of plasma cell mucositis case reports related to qat chewing

Author	*Age (y)*	*Ethnicity*	*Medical history*	*Blood test results*	*Clinical presentation*	*Qat cessation, y/n*	*Improvement, y/n*
Rawal et al 2008[18]	40	Yemeni	No rmh	Not available	• Site: Gingiva • Erythema, oedema, and periodontal bone loss	Y	Y
Marker et al 2002[17]	30	Somalia	No rmh	Not available	• Site: Gingiva, buccal mucosa • Ulceration, desquamation, periodontal bone loss	Y	Y
Al-ak'hali et al 2015[19]	32	Yemeni	Not mentioned	Elevated IgG, positive ANA, lymphocytosis	• Site: Gingiva, tongue, palate • Erythema, oedema, ulceration	Not mentioned	N/A
Al-ak'hali et al 2015[19]	20	Yemeni	Not mentioned	Elevated IgG, lymphocytosis	• Site: Gingiva, palate • Inflammation, sloughing, cobblestoning	Not mentioned	N/A
Al-ak'hali et al 2015[19]	24	Yemeni	Not mentioned	Elevated IgG and ANA positive, lymphocytosis	• Site: Gingiva, lip • Erythema, sloughing, velvety surface, lip swelling	Not mentioned	N/A
Al-ak'hali et al 2015[19]	33	Yemeni	Not mentioned		• Site: Gingiva, lips • Erythema, oedema, lip angular cheilitis	Not mentioned	N/A
Al-ak'hali et al 2015[19]	24	Yemeni	Not mentioned	Lymphocytosis, increased ESR, IgG increased	• Site: Gingiva • Erythema, sloughing	Not mentioned	N/A
Al-ak'hali et al 2015[19]	23	Yemeni	Not mentioned	Lymphocytosis, IgG increased	• Site: Gingiva • Erythema, erosions	Not mentioned	N/A
Al-ak'hali et al 2015[19]	29	Yemeni	Not mentioned	Increased ESR and IgG	• Site: Gingiva, lips • Erythema, lip swelling	Not mentioned	N/A
Al-ak'hali et al 2015[19]	22	Yemeni	Not mentioned	Lymphocytosis, increased ESR, IgG increased	• Site: Gingiva, oropharynx • Erythema, oedema, dysphonia, and dysphagia	Y	Y
Our case 1	47	Somali	Schizophrenia	Raised IgG and IgA + ESR and neutrophils	• Site: Gingiva, buccal mucosa, tongue, palate, oropharynx • Erythema, oedema, desquamation, cobblestoning, ulceration, dysphonia, dysphagia	Y	Y
Our case 2	39	Somali	No rmh	Raised IgG and IgA	• Site: gingiva, buccal mucosa, palate • Erythema, oedema, ulceration	Y	Y

rmh, relevant medical history; *IgG*, immunoglobulin G; *ANA*, antinuclear antibodies; *N/A*, not applicable; *ESR*, erythrocyte sedimentation rate; *IgA*, immunoglobulin A.

ages of these 2 patients are both less than the average age of presentation for idiopathic plasma cell mucositis, documented as 56.6 years.[3] Furthermore, the mean age of presentation in all case reports linked to qat chewing, including our own, is 30.3 years.[17-19] There may be many factors that may account for this difference. The existing research has poor evidence to support the link between idiopathic plasma cell mucositis and allergens. However, in these cases, a potential allergen has been identified. Therefore, the younger mean age of presentation may, in part, be explained by the identification of allergen that is chewed habitually in those as young as 12 years of age.[19,22]

Furthermore, the clinical appearance described in our reports is similar to that in the existing literature, with velvety, erythematous, oedematous, superficially ulcerated gingivae, buccal mucosa, and palate. It is noted that the tongue is less often involved and symptoms of dysphonia and dysphagia are less often described. Dysphonia and dysphagia symptoms linked to qat-induced plasma cell mucositis have been described in 2 of 12 cases in the literature.[19] However, due to the limited number of cases reported, it is difficult to identify the true incidence of these symptoms.

It has also been documented that the worst-affected areas of the oral cavity often correlate with where the

qat is held in the mouth, and, occasionally, periodontal bone loss with radiographic evidence is also identified in these areas.[17-19] However, this was not noted in either of our cases.

Interestingly, in several reports of plasma cell mucositis, including our own, laboratory investigations have identified elevated IgG levels. It is difficult to ascertain the exact relevance of this related to qat and idiopathic plasma cell mucositis because these laboratory investigations are not always consistently undertaken or consistently reported in the literature.

In regard to the management of qat-related plasma cell mucositis, only 5 of 12 case reports in the literature mention the use and efficacy of qat cessation. As a result, it is difficult to assess the impact of qat cessation outside of these cases. However, the patients who were managed using this method experienced a vast improvement of clinical symptoms and presentation,[17-19] thus indicating a strong suspicion in these cases that qat was inducing the clinical and histopathologic presentations seen.

In addition to qat cessation, we have also described a case of a patient requiring systemic steroid therapy as a result of severe symptoms manifesting during unsuccessful qat cessation therapy. The steroid therapy was required in order to provide rapid resolution of symptoms. This highlights the severity of this qat-induced disease, which clinicians should be aware of.

CONCLUSION

In conclusion, we describe 2 further cases of plasma cell mucositis related to qat chewing. The cases described have similarities with existing case reports, and we believe that this contributes further evidence of a correlation between qat chewing and the presentation of plasma cell mucositis sufficient to warrant further research. Furthermore, these cases highlight the need for increased awareness amongst clinicians of qat chewing as an aetiologic factor in plasma cell mucositis in populations in which qat consumption is undertaken. A history of qat chewing should be explored in such patients, and, if found, cessation advice should be given.

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