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CASE REPORT

Oral mucosal involvement as the sole or main manifestation of linear IgA disease: case report and review of the literature

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Abstract

Background: Linear IgA disease is a rare autoimmune subepithelial blistering condition that predominantly affects the skin. Lesions also commonly occur on mucosal surfaces, including the oral mucosa. Demonstration by direct immunofluorescence of IgA deposits in a linear pattern along the basement membrane zone is required for diagnosis.

Case report: A case of linear IgA disease in which oral mucosal involvement remained the sole manifestation of the disease for twenty-three years is presented.

Literature review: A review of the literature revealed seventeen cases in which the disease manifested itself principally in the oral mucosa. Desquamative gingivitis or gingival erythema appears to be the most common manifestation of the disease. Vesicles and bullae, shallow ulcers, erosions, white striae and white patches are also commonly seen. There may be considerable clinical and histological overlap with other diseases, principally mucous membrane pemphigoid and lichen planus.

Conclusion: Although a number of case reports have been published describing instances in which oral mucosal involvement was the only or main manifestation of the disease, the fact that linear IgA disease can manifest itself as a predominantly oral mucosal condition is not widely appreciated by clinicians. This may lead to potentially serious delays in arriving at the correct diagnosis in these cases.

Clinical relevance

Linear IgA disease can mimic other entities, especially mucous membrane pemphigoid and lichen planus, both clinically and histologically. Although principally a dermatological condition, oral mucosal lesions may rarely be the main or sole manifestation of the disease. This is not widely appreciated and as a consequence, potentially serious delays in establishing the correct diagnosis can occur. Repeat biopsies and repeated direct immunofluorescence may be appropriate in instances when the histological findings are not entirely consistent with the clinical picture.

Introduction

Linear IgA disease is a rare autoimmune subepithelial blistering condition that was first recognised as a separate entity in 1979[1]. Demonstration by direct immunofluorescence of IgA deposits in a linear pattern along the basement membrane zone is required for diagnosis[1–3]. The disease affects both adults and children and the majority of cases are idiopathic although some appear to have been induced by certain drugs, physical trauma, infections, underlying malignancy or certain inflammatory diseases[4–11]. Two cases developed after the placement of metal-ceramic bridges; however, it is possible that this association was coincidental[12].

The vast majority of patients present with skin lesions, predominantly an itchy blistering rash which has variable features[1,2,4]. Oral mucosal involvement was first described in 1980[13] and is now considered to be a common occurrence. Most authors report oral mucosal lesions in up to 70% of patients (range in literature: 26–100%)[14–17]. Very rarely, oral mucosal involvement may be the first or only manifestation of the disease. Linear IgA disease is regarded by most clinicians as a dermatological condition and the now well-established fact that the disease can be limited to the oral cavity is not widely appreciated. This may result in considerable delay in arriving at the correct diagnosis. We present a case in which oral involvement remained the only manifestation of the disease for 23 years. A literature review that brings together 17 cases in which oral involvement was the sole or predominant manifestation of linear IgA disease is also presented in order to enhance awareness of this particular manifestation of the disease.

Case report

A 44-year-old man first attended the Charles Clifford Dental Hospital, Sheffield, UK in 1985, complaining of sore gums. He had been prescribed metronidazole 1 month previously and used a chlorhexidine mouthwash but these apparently had no effect on his gingival condition. Clinical examination revealed red erosive areas with some white striae on the gingivae in the region of the lower right canine and premolar. The rest of his oral mucosa was unremarkable and no skin or ocular lesions were present. His general health was unremarkable. Poor oral hygiene was noted. The patient was prescribed metronidazole again but no improvement was noted after 1 month. A clinical diagnosis of erosive gingivitis, probably erosive lichen planus, was made. A gingival biopsy was carried out and microscopic examination showed heavily chronically inflamed and focally ulcerated mucosa with an area of subepithelial clefting. Direct immunofluorescence failed to reveal any deposition of immunoglobulins or complement components. The histological picture was considered to be inconclusive and a differential diagnosis of mucous membrane pemphigoid or erosive lichen planus was offered.

The patient was prescribed bethamethasone mouthwash, but after 3 months there was no appreciable improvement and his condition was clinically considered to be erosive lichen planus. The patient reported that his gingival condition underwent periodic phases of exacerbation followed by remission. Chlorhexidine mouthwash appeared to bring some measure of relief.

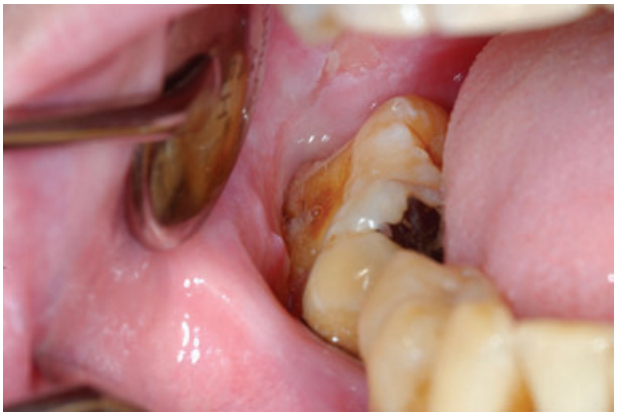

Figure 1 White striae with erosions on the right buccal mucosa.

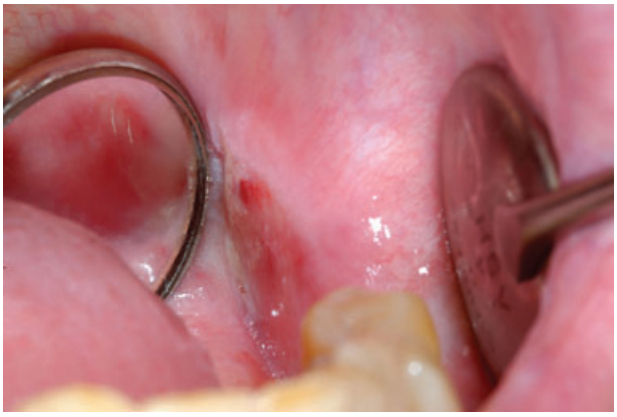

Figure 2 Erythematous erosive lesion with focal white areas on the left buccal mucosa.

His oral hygiene remained poor in spite of regular oral hygiene appointments. During a review appointment in 1997, it was observed that lesions were by now also present on both right and left buccal mucosa as well as on the maxillary (edentulous) alveolar mucosa.

In 2008, the patient presented with bilateral bullous lesions on the buccal mucosa (Figs 1 and 2) as well as erosive lesions on the gingivae. A biopsy was taken from the left buccal mucosa and submitted for routine histological examination and for direct immunofluorescence. Microscopic examination showed chronically inflamed mucosa with separation of the surface epithelium from the underlying lamina propria in several places. In one area, a sub-epithelial bulla filled with neutrophils and occasional eosinophils was present (Fig. 3). Direct immunofluorescence showed linear deposition of IgA at the basement membrane zone with no evidence of IgG, IgM or C_3 deposition (Fig. 4). A diagnosis of linear IgA disease was therefore made.

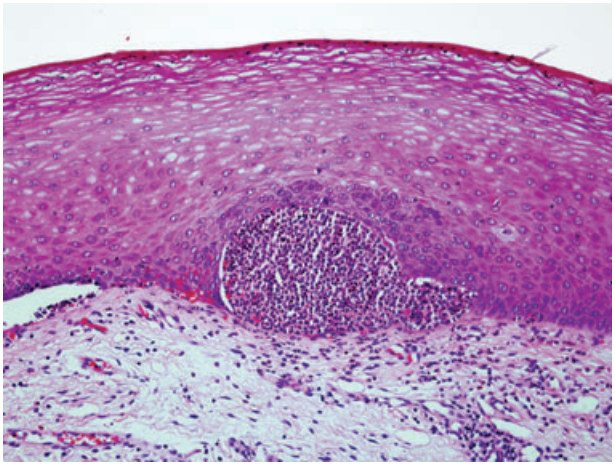

Figure 3 Photomicrograph of lesional buccal mucosa showing subepithelial clefting and a subepithelial vesicle containing a neutrophil microabscess. A chronic inflammatory cell infiltrate that includes eosinophils is present in the superficial lamina propria.

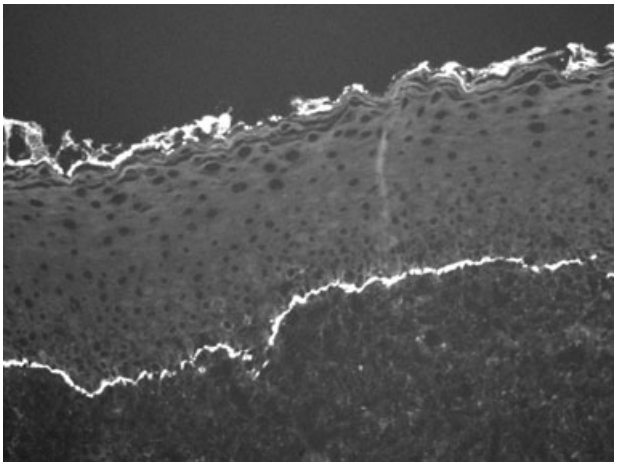

Figure 4 Direct immunoflurescence showing linear IgA deposition at the basement membrane zone.

Discussion

The most common acquired predominantly mucosal subepithelial blistering disorders are mucous membrane pemphigoid and bullous (erosive) lichen planus. Although predominantly a dermatological condition, linear IgA disease should be included in the differential diagnosis of mucosal blistering disorders as it may rarely present as a predominantly mucosal disease. Subepithelial blisters are also seen in bullous pemphigoid, dermatitis herpetiformis and epidermolysis bullosa acquisita; however, skin lesions are always present in these conditions.

Mucous membrane pemphigoid, bullous or erosive lichen planus and mucosal linear IgA disease can have overlapping clinical and histological features and the diagnosis is usually made on direct immunofluorescence results. In mucous membrane pemphigoid, direct immunofluoresence reveals linear IgG and/or C_3 deposition at the basement membrane zone. This may be accompanied by linear IgA or, more rarely, by IgM deposition in a number of cases[14,18,19]. In lichen planus, direct immunofluorescence shows a linear band of fibrinogen deposition in the basement membrane area together with deposition of C_3, immunoglobulins (most commonly IgM) and fibrinogen in cytoid bodies. In a minority of cases, C_3 and/or immunoglobulins are also detected at the basement membrane zone in a granular or discontinuous linear pattern in addition to fibrinogen[20]. A diagnosis of linear IgA disease should be made when there is linear IgA deposition at the basement membrane zone. This is usually the sole immunoreactant present; however, focal deposition of IgG and C_3 may also be seen in some patients[1–3,19].

It is postulated that in linear IgA disease, as in other autoimmune disorders, an environmental trigger such as a virus or drug leads to the production of autoantibodies against components of the basement membrane in genetically predisposed individuals. Immunoelectron microscopic studies have showed IgA deposition in the lamina lucida, in the sub-lamina densa or in both regions[15,21–23]. The autoantibodies in linear IgA disease are mainly directed against the extracellular domain of the hemidesmosomal glycoprotein BP180 (type XVII collagen) and against collagen VII[24–28]. There is some overlap between the target antigens in linear IgA disease, mucous membrane pemphigoid, bullous pemphigoid and epidermolysis bullosa acquisita. Some authors have referred to the sub-lamina densa type of linear IgA disease as the IgA type of epidermolysis bullosa acquisita[28,29]. It should also be noted that because the main clinical features of both mucous membrane pemphigoid and mucosal linear IgA disease are similar and as there is considerable overlap in the target antigens, it has been proposed that these diseases can be grouped together and that mucosal linear IgA disease should be considered a variant of mucous membrane pemphigoid[30]. To date, this proposal has not gained wide acceptance.

In most cases of linear IgA disease, the dermatological manifestations predominate and mucosal involvement, although common, usually constitutes only a minor feature of the disease[14,16]. Oral mucosal manifestations of the disease include vesicles and bullae, painful ulcers and erosions, desquamative gingivitis and white patches with erythema[13,14,16,21,31–34]. White

Table 1 Signs and symptoms in 17 patients with predominantly oral involvement by linear IgA disease

	Sex	Age at presentation (years)	Oral lesions	Other mucosal involvement	Skin lesions at presentation	Skin lesions developed after presentation
Kumar *et al*.[13]	Female	65	Blisters, diffuse shallow ulcers	Ocular	No	No
Hietanen *et al*.[21] (case 2)†	Female	61	Erosions, scarring	Nose, genital, ocular	No	Yes, ? after 4 years
Leonard *et al*.[14] (patient 2)†	Male	65	Desquamative gingivitis	Ocular	No	Not known
Leonard *et al*.[14] (patient 24)†	Male	59	Not described	Ocular	No	Not known
Fine *et al*.[35] (patient 10)†	Female	83	Not described	No	Yes – rare umbilical lesions	
Chan *et al*.[36]	Female	76	Blisters, ulcers, erosions	Larynx, vagina	No	Yes, after 5 years
Porter *et al*.[37]	Male	69	Gingival soreness and blistering	No	No	No
Porter *et al*.[38]	Male	29	Gingival soreness, ulcers preceded by blood-filled blisters	Ocular	No	No
Kirtschig *et al*.[34†]	Male	38	Generalised gingival erythema with fixed white patches, positive Nikolsky's sign	Penis – coronal sulcus and glans	No	No
Cohen *et al*.[33] (case 1)	Female	57	White, reticular striae on buccal mucosa and gingivae, diffuse gingival erythema, later blisters, Nikolsky's sign initially negative but later positive	No	Yes	
Cohen *et al*.[33] (case 2)	Female	78	Ulcers, gingival erythema, later white reticulate area, vesicles, white plaque	No	No	No
Smith *et al*.[39]	Female	60	Erosive gingivitis, palatal ulcers	Ocular	Yes – area of small vesicles on one leg	
Eguia del Valle.[12] (case 1)	Male	43	Gingival erythema and ulceration, positive Nikolsky's sign	No	No	No
Eguia del Valle.[12] (case 2)	Female	72 (at time of referral)	Gingival erythema and ulcers, desquamative gingivitis, blood-containing bullae, positive Nikolsky's sign	No	No	No
O'Regan *et al*.[40]	Male	50	Desquamative gingivitis, widespread ulcers, blister	Larynx, pharynx, oesophagus	Yes	
Lewis *et al*.[41]	Female	79	Ulcers, bullae, erosions	No	No	No
Angiero *et al*.[42]	Female	56	Ulcers, bullae, gingival erosion and erythema	No	No	No

†Published as a case of mucous membrane pemphigoid.

reticular striae similar to those seen in lichen planus have been described[32]. Mucosal scarring may ensue; however, this feature is difficult to assess in the oral mucosa[17,19,21,34].

When the oral lesions are not accompanied by skin lesions or where the oral lesions are severe and dominate the clinical picture, the disease may be clinically and histologically confused with other entities, especially with mucous membrane pemphigoid and erosive lichen planus with which there may be considerable clinical overlap. A search of the literature revealed 17 cases in which oral mucosal involvement was the main or only manifestation of linear IgA disease, at least initially. The clinical features of these cases are summarised in Table 1. Some of the cases included in this review were diagnosed and published as cases of

cicatrical or mucous membrane pemphigoid; however, as far as the authors can establish from the description of the histological picture and direct immunofluorescence pattern, these cases could possibly represent linear IgA disease because linear IgA deposition at the basement membrane zone as the sole immunoreactant was described in these cases.

From these 17 cases, it appears that the most common clinical presentation of oral disease is gingival erythema or desquamative gingivitis which was seen in at least 11 patients and was the sole oral manifestation of the disease in six patients[12,14,33,34,37–40,42]. Recently, Leao *et al*. published a retrospective analysis of 187 patients with desquamative gingivitis which also included three patients with linear IgA disease[43]. These three patients apparently did not have any extraoral manifestations of the disease; however, they are not included in Table 1 because of the paucity of detail provided. It is however clear that linear IgA disease should be included in the differential diagnosis of diseases manifesting as desquamative gingivitis.

Two patients had white striae or white reticulate lesions which suggest a lichen planus-like appearance[33]. One further patient had fixed white patches in addition to gingival erythema, a picture which can also be consistent with a clinical diagnosis of lichen planus[34]. Our patient also had a combination of gingival erythema and white areas and striae which were not limited to the gingivae, a clinical picture that was felt to be most consistent with a diagnosis of lichen planus even though the histological picture was never entirely typical of this disease.

The treatment of oral linear IgA disease can be difficult and, unlike mucous membrane pemphigoid and lichen planus, linear IgA disease frequently fails to respond to topical or systemic corticosteroids alone. Failure of oral vesiculobullous disease or of desquamative gingivitis to respond to corticosteroid therapy has been cited as being suggestive of the diagnosis[40]. Most cases respond to dapsone, either on its own or in combination with systemic corticosteroids[44]. Other drugs that have been successfully used to control oral disease include sulfapyridine[38], sulfamethoxypyridazine[34], cyclophosphamide[39] and mycophenolate[41]. Colchicine and a combination of tetracycline with niacinamide have also been used to control skin lesions but the efficacy of these drugs in the treatment of oral lesions is uncertain[45,46].

The frequent failure of linear IgA disease to respond to the established treatment protocols for mucous membrane pemphigoid and lichen planus highlights the importance of establishing the correct diagnosis in these cases so that treatment protocols that are more likely to control the lesions can be initiated. More importantly, the frequent occurrence of ocular lesions and scarring in patients with linear IgA disease also necessitates prompt recognition of the disease. A diagnosis of linear IgA disease should imply referral to an ophthalmologist to assess ocular involvement which may be symptomless[14].

The case we have presented went unrecognised for 23 years, partly because the initial direct immunofluorescence test failed to reveal any deposition of immunoglobulins or complement components. The case reported by Chan *et al*.[36] also had initial negative direct immunofluorescence. This highlights the fact that on occasion repeat biopsies, with direct immunofluorescence being carried out on each biopsy, may be necessary especially when the clinical picture is not explained by the histological features. The case presented by Kumar *et al*. had histological features which were identical to those seen in lichen planus; however, the clinical picture was not typical of this entity[13]. This case also highlights the importance of clinicopathological correlation. It is perhaps worth noting that Leao *et al*. pointed out that patients with desquamative gingivitis may experience considerable delay before their condition is correctly diagnosed[43]. Some of the patients in their study were diagnosed 25 years after developing clinical signs and symptoms. O'Regan *et al*. also noted that there was a (relatively short) delay in diagnosing the patient they presented[40].

Our case could have been recognised earlier as linear IgA disease if more weight was put on the repeated histological finding of subepithelial clefting from the underlying lamina propria, on the histological picture in general which was not entirely typical of lichen planus and on the fact that the lesions never really responded to corticosteroid therapy. This should have prompted more rigorous consideration of a vesiculobullous disease as opposed to erosive or bullous lichen planus in spite of the initial negative direct immunofluorescence result. Unfortunately, only three biopsies were submitted fresh for direct immunofluoescence over a stretch of 23 years; the first was negative, the second consisted of epithelium only and was therefore unsuitable (even though direct immunofluorescence was still carried out), while the third, which was submitted in 2008, finally revealed linear IgA deposition at the basement membrane zone.

Conclusion

The case we have presented as well as the other cases we have reviewed show that oral lesions can dominate the clinical picture in some cases of linear IgA disease.

Recognition of this is essential in order to prevent potentially serious delays in diagnosing this condition.

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