Case Report

Verruciform xanthoma: A case report and a review of recurrent cases

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Abstract

Oral verruciform xanthoma (VX) is an infrequently encountered benign lesion in the oral cavity. We report an unusual case of VX on the left buccal mucosa presented as a red and white exophytic mass with a greyish white diffuse patch associated with it. A differential diagnosis of papilloma, verrucous carcinoma, and squamous cell carcinoma associated with leukoplakia was listed. Histopathological findings were suggestive of VX due to the presence of characteristic foam cells in the connective tissue papillae. Immunohistochemical analysis with CD68 showed strong positive immunoreactivity revealing expression of foam cells. After the excisional biopsy, the patient was followed up for the next 6 months with no recurrence. Follow-up is very essential in such a case as the exophytic lesion was associated with a potentially malignant disorder. A short review of reported recurrent cases of verruciform xanthoma is also discussed.

Keywords: Foam cells, IHC, verruciform xanthoma, verrucous lesions

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INTRODUCTION

Oral verruciform xanthoma (VX) is an infrequently encountered benign lesion of unknown pathogenesis, first described in the oral cavity by Shafer in 1971.[1] It predominantly affects the oral mucosa but can also affect cutaneous sites on the face, trunk, extremities, and genitalia.[2]

VX manifests as a solitary, sessile, or pedunculated lesion with a rough or pebbled, granular, or verrucous surface, which is an exophytic, sharply demarcated, and slightly raised plaque-like lesion and is generally asymptomatic in nature.[3] It is most commonly seen on the gingiva.[4] Its appearance may direct the clinician towards verruca vulgais and oral papilloma and with the high probability of getting it misdiagnosed as verrucous carcinoma both clinically and microscopically due to its analogous features like elongated rete pegs and deep keratin-filled clefts.[5] Numerous cases of VX have been detected and treated world-wide in the oral cavity in the past 50 years. Despite treatment, a few cases were found to recur in the existing literature.[6] The review that is discussed explores all the recurrent cases till the present date.

CASE HISTORY

A 58-year-old male patient presented with the chief complaint of missing teeth and wanted to get them replaced. He was unaware of his oral lesion. He had a habit

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of tobacco chewing 4–5 times a day for the past 25 years. There was no other known medical or family history.

On clinical examination, incidentally, a whitish red diffuse raised lesion, exophytic in nature, was noticed on the left buccal mucosa extending from the corner of the mouth to the depth of the vestibule, measuring 2 × 3 cm in size, and was found in the region of teeth 33 to 37, which had a cauliflower shape on inspection. Another secondary white diffuse patch was associated with a concerning lesion on the left alveolar ridge extending from the region. The lesion was asymptomatic and had a soft consistency. Lymph nodes were not palpable. Hence, a clinical diagnosis of papilloma was made associated with leukoplakia.

After clinical examination, an incisional biopsy was taken to rule out malignancy [Figure 1].

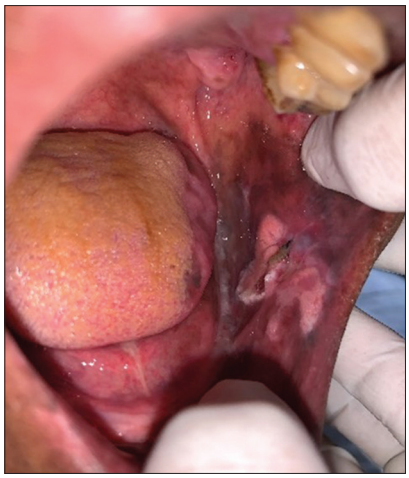

Figure 1: Clinical appearance of VX showing whitish red raised diffuse lesion with exophytic growth noticed on the left buccal mucosa

On histopathological examination, the haematoxylin and eosin (H&E)-stained sections showed an acanthotic epithelium covered by a thickened layer of parakeratin. A few clefts filled with parakeratin were seen between the epithelial projections. The epithelium was non- dysplastic with a few mitotic figures in the basal cell layers. The rete ridges were elongated to a uniform depth entrapping connective tissue papilla. The most conspicuous feature was the accumulation of numerous large macrophages with a foamy cytoplasm typically confined to the connective tissue papillae. Moderate chronic inflammatory cell infiltration was noted adjacent to the epithelium, along with many dilated and engorged blood vessels. The deeper region showed adipose tissue. Based on these features, a diagnosis of VX was given [Figure 2].

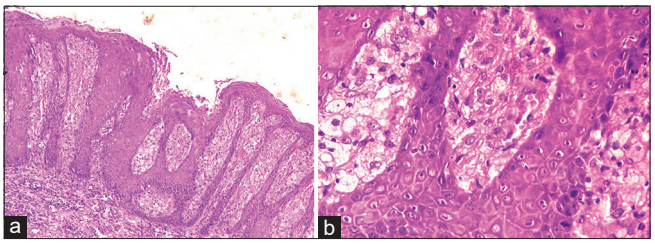

Figure 2: (a) Photomicrograph of VX (H&E; 10x) of the lesion exhibiting the uniform rete pegs with parakeratotic invaginating crypts and connective tissue filled with xanthoma cells (b) Photomicrograph of VX (H&E; 40x) showing the connective tissue exhibiting the accumulation of foam cells between the epithelial rete pegs

Immunohistochemistry (IHC) analysis with CD68 revealed positive expression by foam cells. This suggests its monocyte–macrophage lineage [Figure 3].

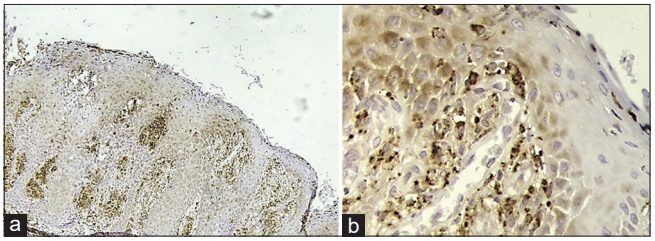

Figure 3: (a) Strong positive immunoreactivity to antibody CD-68 (10x) (b) Xanthoma cells showing strong positive immunoreactivity to antibody CD-68 (40x)

Excisional biopsy was done, and the patient was kept under follow-up. No recurrence has been reported for 6 months, and the patient is being followed up.

DISCUSSION

Looking at the existing reports of VX, it is considered as a rare benign proliferative lesion of the oral cavity. It usually appears clinically as a papule or single plaque and may be sessile or pedunculated showing a verrucous or papillomatous surface, with the colour varying from reddish pink to grey. It is asymptomatic, has a slightly low growth rate, and enlarges to sizes varying from 0.2 to 2 cm.[7] There is no gender predilection as such and most frequently occurs in the middle-aged group with a mean age of 40–50 years.[8] A review of literature showed that 70% were localised to anatomic regions, predominantly the masticatory mucosa.[9] A recent review confirms that 70.8% of the lesions were indeed seen on the masticatory mucosa, that is, gingiva and palate, and the second commonly encountered site was the lip (1.89%), and the floor of the mouth was an unusual location with 1.42%.[9] In the present review of oral VX cases (duration: 1996–2024), the male-to-female ratio is found to be 3:1 with the most common site as gingiva followed by hard palate, with the size of the lesion ranging from 0.1 to 2.5 cm. We have made a short table summarising the clinical features and IHC findings of oral VX cases reported from 1996 to 2024 [Table 1]. The present case was noted at an unusual site in the region of buccal mucosa. Ide *et al*.[10]

Table 1: Details of reported cases of oral VX (Duration: 1996 to 2024)

Author	Year	No of cases	Age/Gender	Site	Size (cm)	IHC Findings
Iamaroon A *et al*.[23]	1996	12	Age range- 14 to 83 years (mean 50.45 years)	Gingiva (*n*=6) Hard palate (*n*=6)	0.4 to 1.5 cm	The foam cells were of monocyte /macrophage lineage showing consistent positivity for cathepsin B in all the 12 specimens but were inconsistently positive for MAC387, Leu7, AACT, and PNA. The predominant cells in the inflammatory infiltrate were T-cells. The mean composition of the mononuclear infiltrate in the 12 specimens was 51.8±2% UCHLI-positive (pan T) cells, 13.8±7% L26-positive (B) cells, 1.0±l% Leu7-positive (NK) cells, 43.3±2% LN3-positive (HLA-DR) cells, and 4.8±4% cathepsin B- positive (macrophages) cells. S-100-positive (Langerhans) cells were occasionally found in the supra-basal layer of the epithelium. LN3-positive HLADR cells were found in the lamina propria. LN3-positive keratinocytes were frequently identified in the supra-basal layer of the epithelium at the sites of intense inflammatory infiltrates.
Shin HI *et al*.[24]	1997	2	30/M 57/M	Labial Gingiva of 45 Alveolar mucosa of 46	3×2.5 cm 0.9×0.6 cm	The foam cells of VX showed strong reactivity for antibodies to macrophage [CD 68(KPI)] and vimentin and faint reactivity for alpha-1-antitrypsin, and they reacted negatively for NES, desmin, S- 100, and keratin. A number of dendritic cells in epithelia and sub-basal lamina propria in VX were strongly positive for S-100.
Oliveira PT *et al*.[25]	2001	4	Age range- 23 to 46 years (Mean age=38.5 years)	Hard Palate (*n*=2) Gingiva (*n*=1) Floor of mouth (*n*=1)	The lesions ranged from 0.4 to 2 cm in diameter.	The foamy cells showed strong positivity for CD68. Some CD68-positive cells were also present in the epithelium. The CD68 label in the foamy cells appeared as delicate network throughout the cytoplasm.
Visintini E *et al*.[26]	2006	1	24/M	Ventral surface of the tongue	-	Foamy cells were positive for CD68-KP1, CD68-PGM1, alpha-1-antitrypsin, and vimentin and negative for desmin, cytokeratins, NSE, and S-100.
Rawal SY *et al*.[27]	2007	16	-	Gingiva (*n*=6) Palate (*n*=3) Mucosa (*n*=7)	-	Positive xanthoma cells immunoreactivity percentage with – RM3/1=100% 25F9=88% 27E10=50%.
Ide F *et al*.[10]	2008	36	Age range- 27 to 84 years (mean age 58.3 years)	Gingiva/alveolar mucosa (*n*=25 cases), Tongue (*n*=4), Buccal mucosa and palate (*n*=3 each) Floor of mouth (*n*=1). The gingiva/alveolar mucosa was the most frequent site.	-	Foam cells expressed for MSR-1, MCP-1, CCR2, and oxidized low-density lipoprotein (ox-LDL). VX epithelium showed expression of ox-LDL in the whole extension, MCP-1 was localised in the basal layer and IL-8 in the upper spinous layer. The pattern of keratinocyte HLA-DR was varachatiable, ranging from focal basal cell expression to entire thickness staining.
Aggarwal *et al*.[5]	2014	1	46/M	Extending from left interdental papilla between maxillary first and second premolar towards midline and left corner of the mouth.	1×2 cm	CD68 positivity for foamy macrophages
de Andrade BA *et al*.[21]	2015	6	Age range- 28–74 Years M:F Ratio: 3:2	Hard palate 30% Buccal mucosa 30% Gingiva 25% Floor of the mouth 10% Buccal vestibule 5%	0.1–2.5 cm (0.9 cm)	Foamy macrophages of all cases showed strong cytoplasmic positivity to CD68 (KP1 and PG-M1) and moderate staining for CD63 and CD163. CD138 and cytokeratin 14 (CK14) were strongly positive in the basal and supra-basal layers of the oral epithelium of all cases, with a membrane and cytoplasm pattern, respectively. The orange-coloured parakeratin layer exhibited granular membrane pattern of positivity for CD138, but it was negative for CK14 in all cases. CKs 8 and 19 were negative in all cases.
Garcia AS *et al*.[28]	2016	1	43/M	Hard palate	0.5 cm in diameter	Foam cells were positive for anti-CD68 Antibody, and anti-Ki-67 antibody was restricted to the basal layer of the oral epithelium and negative for foam cells.

Contd...

Table 1: Contd...

Author	Year	No of cases	Age/Gender	Site	Size (cm)	IHC Findings
Pereira T *et al*.[29]	2016	1	59/M	Lower lip	0.5×1 cm	Xanthoma cells showed a strong cytoplasmic positivity for CD68.
Hiraishi *et al*.[4]	2016	1	65/F	Left lateral border of the tongue	1.5×1	CD68 positivity for macrophage foam cells
Kimura M *et al*.[30]	2016	1	64/M	Lower gingiva	0.7×0.7	CD68 positivity for macrophage foam cells. Ki67 & p63 in basal and parabasal layers
Theofilou VI *et al*.[18]	2018	1	56/F	Left lateral lingual border extending to the ventral surface of the tongue	1×0.5×0.3	Foamy cells exhibited strong immunostaining for CD68 antibody.
Gannepalli A *et al*.[3]	2019	1	52/M	Attached gingiva	0.8×2	CD68 positivity (+++) for foam cells.
Preto KA *et al*.[31]	2024	2	30/M	Floor of the mouth	1 cm in diameter	The foamy macrophages were positive for CD163 and negative for S100.

suggested some oral agents such as periodontal pathogens, mechanical stimuli, tobacco, alcohol, drugs, or any allergic food stuff may be associated with local inflammation. These irritants can be considered as the initiators of the process, causing accumulation of epithelial breakdown products which induce inflammatory response and subsequent release of lipid material through the epithelium and finally scavenged by the macrophages, leading to formation of foamy macrophages.[5] On the molecular level, it all begins with monocyte chemotactic protein-1 (MCP-1) on the basal cells of VX and its shared receptor chemokine (C-C motif) ligand 2 (CCR2) on the macrophages. MCP-1 is considered to be a potent monocyte/macrophage attractor. The activated T-cells are recognised to modulate the production of these ligand-receptor pairs (MCP-1 and CCR2), which further upregulates the macrophage and T-cell trafficking into the sub-basal papillae region of VX. Both the ligand and receptor pair are expressed in the foam cells of VX. The activated T-lymphocytes due to the chronic inflammatory process recruit macrophages associated with CCR2 molecules, which sequentially upregulates the expression of macrophage scavenger receptor (MSR) on them. These macrophages identify, trap, and internalise the low-density lipoproteins (LDLs) from the epithelial cells and oxidise them, resulting in foam cells. The foam cells express MSR-1, which helps in self-sustenance and Ox (oxidized)-LDL as a chemoattractant for macrophages and T-cells.[11]

Most commonly, the lesion has a verruciform appearance, but it may also appear polypoid, papillomatous, sessile, or pedunculated. Our case showed verrucous appearance, which could be confused with other oral lesions. Hence, it is important to go through common lesions encompassed considering as differential diagnosis which may include squamous papilloma, verruca vulgaris, fibroma, leukoplakia, and squamous cell carcinoma.[5] Clinically, we can differentiate these lesions from VX to some extent. The majority of squamous papillomas are round in shape with a papillary surface with whitish colour and are pedunculated.[12] Verruca vulgaris is firm, circumscribed, and elevated with a papillomatous hyperkeratotic surface, while fibroma is just a smooth papule in the mouth.[12] Verrucous carcinomas appear as huge, exophytic–endophytic lesions with a typical cauliflower-like surface.[3] Leucoplakia has a typical white patch or a plaque with a rough texture.[13] Oral squamous cell carcinoma is a white or reddish ulcerated lesion and has rolled up margins with a central necrotic area.[14] As the lesion was associated with leukoplakia, the diagnosis was not very straight forward in our case. VX may coexist with oral discoid lupus erythematous,[15] graft versus host disease,[16] congenital epidermal nevi, and an STD.[17] VX could be associated with oral potentially malignant disorders such as oral submucous fibrosis,[3] lichen planus,[18] and erythroplakia,[16] though its association with leukoplakia is rare. The present case was associated with leukoplakia. This association of an oral potentially malignant disorder with VX could be a factor responsible for recurrence of the lesion.

The conclusive diagnosis of oral VX is accomplished through microscopic examination frequently succeeding an excisional biopsy.[9] Some features of VX may/can show similarity with other lesions like papillary or verrucous proliferation of the squamous epithelium, elongated rete ridges.[5] Hence, it is essential to check out for its primary distinguishing features, the orange hue and aggregation of "lipid-laden foamy cells" within the connective tissue for diagnosing every such lesion for definite diagnosis. A significantly condensed parakeratin with a frayed surface and a prominent "orange colouration" is seen, which displays a sharp delineation from the remaining epithelium. Parakeratin clefting into the underlying spinous layer and chronic inflammation of varying degrees in the subjacent lamina propria is also noted.[9]

Nowparast *et al*.[19] described three different architectural types viewed below the low power of oral VX. They are a) verrucous type, b) papillary type, and c) flat type which can mimic other papillary entities.[9] These variations in

epithelium might be due to the upward pushing effect by accumulated macrophages towards the epithelium, which further causes the thinning of epithelium giving verrucous or papillary appearance overlying the macrophages in connective tissue papilla. The verrucous types are hyperkeratotic, well-circumscribed, acanthotic, and usually elevated. The parakeratin arrangements invaginate crypts that extend deep into the acanthotic epithelium.[9] In some cases, the lateral rete ridges taper towards the centre, creating a cup-shaped appearance, mimicking verruca vulgaris and verrucous carcinoma, but do not display a thickened stratum granulosum. The papillary type has abundant finger-like projections of the stratified squamous epithelium with cores of connective tissue. Parakeratin-covered crypt-like spaces extend directly above the mucosal surface, analogous to a papilloma. In the last variety, the flat type, epithelial proliferation is seen beneath the surface. The rete ridges are slender and of uniform depth and exhibit inconstant elongation. The connective tissue papillae might extend next to the surface and are separated from the outer surface by a thin, slightly parakeratotic cell layer. Nowparast *et al*.[19] described a further intense parakeratosis in the verrucous and papillary types.

Former studies report that immunohistochemical markers are to be used to aid in the diagnosis of VX, including CD68, CD63, and CD163. Mostafa *et al*.[20] first described that the clone PG-M1 of the monoclonal antibody-CD68 identifies macrophages. Further immunohistochemical markers, CD63 and CD163 (monocytic/macrophage markers), expressed on the majority of circulating macrophages and monocytes are positive for the xanthoma cells as well. The xanthoma cells have been stated to stain negatively for S100, CK8, CK19, CK14, and pankeratin.[21] Table 1 shows different immunohistochemical markers used by different authors for foam cells in VX.

Treatment of VX comprises simple excision, and an excellent prognosis is reported in almost all cases.[22] In the period of 50 years, a few cases of VX were found to recur despite suitable surgical approaches [Table 2]. The recurrences are commonly seen in 35–45 years with slight male predominancy in the Caucasia population and more commonly presented on the palate followed by gingiva [Table 2]. As the evidence from the literature, palatal gingiva is more prone to masticatory trauma and irritation, which probably might be the reason of recurrence. The other reason probably may be associated with the thick keratinised rigid mucosa bound to the underlying bone causing difficulty for the surgeon during treatment. Cases recurred more commonly in the period of 2–5 years. The association of recurrent cases of VX with potentially malignant disorders or with tobacco habits should be evaluated. The lesions which are accompanying a potentially malignant disorder should be followed up after excision and checked for recurrence.

CONCLUSION

VX is considered as a benign lesion of the oral cavity. Unfortunately, after successful diagnosis and treatment, a few recurrent cases have been reported. It becomes the sole responsibility of the clinician and pathologist to diagnose it correctly by excluding all the possible entities similar to it clinically and histopathologically. The association of recurrent cases with potentially malignant disorders and tobacco habits needs an evaluation. This will aid the surgeon to adopt specific treatment modality and also keep a more thorough and fine approach while treating cases on the masticatory gingiva. Nevertheless, an appropriate follow-up track for such cases for any recurrence in the future is necessary.

Key Messages

The present case of verruciform xanthoma was associated with a potentially malignant disorder. Hence, follow-up is very essential in such cases.

Declaration of patient consent

The authors certify that they have obtained all appropriate

Table 2: Details of recurrent cases of oral VX

Author and year	Race, Age and Sex	Location	Size in cm	Histological appearance	Medical History	Initial treatment	Recurrence period
Nowparast B,[19] 1981	Caucasian, 39 years/F	Palate	1.3	Verrucous	-	Not available	-
Neville B,[32] 1986	White, 21 years/M	Palatal gingiva	-	-	-	Not available	After 2 years
A Iamroon,[23] 1996	Caucasian, 44 years/ M	Palate	1.5	-	-	Surgical excision	After 5 years
F Ide,[10] 2008	-	Gingival lesion	-	-	-	-	-
Anbinder AL,[33] 2011	Race not mentioned, 70 years/ M	Lip	-	-	Neurofibromatosis	Surgical excision	After 2 years
Ryu Da Jung,[17] 2013	Race not mentioned, 35 years/ M	palate	2.5×1	Papillary	Sexually transmiteed disease	Conservative surgical resection	After 4 years
Belknap AN,[9] 2020	Not mentioned	Lower lip	-	-	-	-	-
Belknap AN,[9] 2020	Not mentioned	Maxillary gingiva	-	-	-	-	-
Belknap AN,[9] 2020	Not mentioned	Mandibular vestibule	-	-	-	-	-

patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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