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Oral Benign Fibrous Histiocytoma – An Enigmatic Entity and Its Journey across Six Decades.

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Introduction:

It all began way before the 1960's, however on one fateful day in the year of 1961, Kauffman ST and Stout AP [1] changed the way the world looked at fibrous soft tissue tumours by being the first to report about fibrous histiocytoma and recognising it as a separate clinical entity.

Benign fibrous histiocytoma depicts a group of quasi-neoplastic lesions that show both fibroblastic and histiocytic differentiation. However the precise origin and pathophysiology of the same remains largely undetermined [2].

Experts have hypothesized that the cells originate from tissue histiocytes with fibroblastic properties [3], while others have argued saying that immunohistochemical evidence shows factor XIIa positivity which is known to favour a dermal dendrocytic cell predilection [4]. In consequence of the controversies of origin, over the years, BFH has been designated several names hence, like sclerosing hemangioma, cutaneous hemangioma, fibroxanthoma and nodular subepidermal fibrosis [3].

BFH can be cutaneous and non-cutaneous in nature. Cutaneous BFH commonly originates in areas of skin exposed to sunlight. Non-cutaneous BFH account for approximately 1% of all benign FH lesions and occur most commonly in soft tissues in the lower extremities (50%), less frequently in the upper extremities (20%) and retroperitoneum (20%) [5].

Benign fibrous histiocytoma can also be categorised into superficial and deep forms. Deep benign fibrous histiocytoma is an extremely rare entity, comprising of less than 5% of all BFH tumours [6]. Fibrous histiocytoma as reported in literature can also present as malignant fibrous histiocytoma or benign fibrous histiocytoma involving soft tissue, hard tissue or occasionally both [1].

The incidence of BFH, however, in the oral cavity is rare. There are few cases across literature of evidence of presence of BFH in the buccal spaces, tongue, gingival or alveolar ridge, mandible, maxilla, lower and upper lip, soft palate and floor of the mouth. Rare occurrences also include nasal cavity and paranasal sinuses, larynx, trachea, temporomandibular joint and submandibular and parotid glands [5, 7].

The aim of this study is to trace the incidence of BFH across literature, discuss the diagnostic techniques, current protocols in treatment and incidence of metastasis or recurrences if any.

Discussion

Gray et al. found that the mean age of patients with oral and perioral BFH is around 55 years with the range between 12 to 71 years [11]. There is a definite female predilection. Bielamowicz et al. in their study of BFH in the head and neck region found the ratio of M: F as 2.5:1[12].

In literature, the clinical picture varies significantly depending upon the factors like location, duration and suspected aetiology. Speculations are rife on the aetiology of BFH namely secondary to trauma, infection and also immuno-suppression [22].

Clinically these benign tumours can be asymptomatic [5], solitary, gradually enlarging growth that is well-circumscribed, painless and does not show aggressive behaviour or damage to the overlying mucosa. The most common chief complaint a patient presents with is, swelling with possible facial asymmetry and occasionally pain [1].

In rare situations a patient may complain of nasal obstruction, nasal discharge, and episodes of epistaxis in case of involvement of the maxilla or dysphagia, dyspnoea and difficulty in speech if tumour is present over the lingual or palatine region [9, 13].

On oral examination, it can present as elastic-soft or elastic-firm in consistency [9], demarcated and painless mass with no ulceration or involvement of adjacent structures.

The diagnosis and analysis can be challenging and is usually based on a combination of diagnostic aids like histopathology, light microscopy and immunohistochemistry [5].

Histiology

The histopathological picture shows a fibro-histiocytic lesion that is frequently non-infiltrating in nature. The histopathology can be varied with interlacing fascicles of spindle cells, plump and vesicular nucleus, tapered and blunt ends arranged in a typical storiform pattern , densely proliferated histiocytes, spindle shaped tumour cells [8] or round histiocyte-like cells, lipidcontaining xanthoma cells, multinucleated giant cells, and scattered lymphocytes as well[7].

Table 1: Histiogenesis of BFH

Evidence In Support Of Histiocytic Origin	Evidence In Support Of Fibroblastic Origin
Presence of lysosomal and proteolytic enzymes	Histological resemblance to fibroblastic cells.
Affected cells exhibit phagocytic activity	Lack of expression of histiocytic marker (Langerhans granules).
Lipophilic cells.	
Multinucleated osteoclasts like cells.	

The differentiation between BFH and MFH on the basis of a histopathological picture can be made by the lack of cellular atypia [9], high mitotic activity, high pleomorphism of cells [12], hyperchromasia, atypicality of the nucleus and nuclear fission [8] all of which are the characteristic features of MFH.

There is still a marked deficit of specific markers for fibro-histiocytic lesions hence the diagnosis of BFH is usually made on the basis of the absence of markers for cells of other lineages. Diagnosis through exclusion. The immunohistochemistry diagnosis is carried out with the help of formalin fixed, paraffin-embedded sections using streptavidin-biotin-peroxidase complex labelling method. BFH shows immunostaining for vimentin (+), CD68 (+), CD34 (+), S100 (−), CD117 (−), Leu7 (−), desmin (−), and α- SMA (−) [10, 18].

Table 2: Stainability of immuno-histochemical staining.

- : no stain + : positive

Search antibody	Stainability
S-100	-
NSE	-
α 1-ACT	+
Lysozyme	+
CD68	+
Vimentin	+

CT scans can be of diagnostic aid in hard tissue fibro-histiocytic tumours which present as well defined, expansile lytic lesions that may or may not be associated with thinning or breaching of the cortical plates. MRI

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scans are used in case of soft tissue fibro histiocytic tumours which show up as heterogeneously hyper intense on T2-weighted image [16,17,19]. Role of PET scans is not tapped into as of now and may pave way for better imaging in the recent future.

Table 3: Review of cases of BFH of Soft tissues in chronological order.

SE: Surgical excision; FU: Follow-up; NED: No evidence of disease.

Journal	Authors	Age/Sex	Location	Treatment	FU time/ Recurrence	Year
Cancer	O'Brien and Stout[24]	50/F	Buccal Mucosa	SE	24M/ NED	1964
Journal of Oral Medicine	Hillis and Beasley [25]	52/M	Internal Left Cheek	SE	12M/NED	1975
Revue De Stomatologie et de chiururgie Maxillo-Faciale	Alonso del and Hayo et al.,[26]	68/M	Buccal Mucosa	*	12M/ NED	1976
	Hoffman and Martinez[27]	8/M	Buccal Mucosa	SE	14M/ NED	1981
	Weerapradist and Punyasingh[28]	50/F	Retromolar area	SE	*	1984
	Thompson and Shear [29]	49/F	Retromolar area	SE	10M/ NED	1984
	Thompson and Shear[29]	36/M	Maxillary Gingiva	SE	12M/ NED	1984
	Thompson and Shear[29]	44/F	Base of Tongue	SE	11Y 7M/ NED	1984
	Thompson and Shear[29]	49/F	Palate	SE	7M/ NED	1984
	Thompson and Shear[29]	17/M	Buccal Mucosa	SE	7M NED	1984
	Fielman and	11/M	Soft Palate	SE	8M/ NED	1989

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	Morrow[22]					
	Triantafyllou et al. [30]	70/M	Tip of Dorsal Tongue	SE	7Years/ NED	1985
	Fletcher [18]	45/M	Subcutaneous Face	SE	*	1990
	Fletcher [18]	31/M	Intramusular scalp	SE	*	1990
	Fletcher [18]	56/M	Intramuscular cheek	SE	*	1990
	McLeod and Jones [31]	22/F	Lower Lip	SE	18M/NED	
	Gray et al.[11]	45/M	Upper Lip	SE	12M/NED	1992
	Gray et al. [11]	42/M	Buccal Mucosa	SE	12M/NED	1992
	Gray et al.[11]	65/M	Buccal Mucosa	SE	12M/NED	1992
	Gray et al.[11]	37/F	Tongue	SE	24M/NED	1992
	Gray et al.[11]	50/F	Dorsum of Tongue	SE	14M/NED	1992
	Gray et al.[11]	71/F	Buccal Mucosa	SE	14M/NED	1992
	Gray et al.[11]	45/F	Lower lip	SE	12M/NED	1992
	Gray et al.[11]	49/M	Maxillary Vestibule	SE	12M/NED	1992
	Gray et al.[11]	70/F	Buccal Mucosa	SE	14M/NED	1992
	Gray et al. [11]	60/M	Mandibular Vestibule	SE	14M/NED	1992
	Gray et al. [11]	68//F	Buccal Mucosa	SE	10M/NED	1992
	Gray et al. [11]		Mandibular Vestibule	SE	7M/NED	1992

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	Gray et al. [11]	66/F	Mandibular Vestibule	SE	12M/NED	1992
	Gray et al. [11]	37/F	Maxillary Gingiva	SE	14M/NED	1992
	Bielamowicz et al. [12]	25/M	Buccal Mucosa	SE	24M/ NED	1995
	Bielamowicz et al. [12]	49/M	Submandibular Region	SE	17years/ NED	1995
	Shrier et al.[53]	Newborn (1 day)	Nasal Cavity	SE	*	1998
	Dardo et al.[54]	44/M	Floor of mouth	SE	14M/ NED	1998
	Menditti et al.[13]	44/M	Lingual Mucosa	SE	10years/ NED	1998
	Menditti et al.[13]	34/M	Tongue	SE	10years/ NED	1999
	Dardo et al.,[54]	34/M	Tongue	SE	12M	1999
	Hong et al., [32]	74/F	Floor of mouth	SE	9M/ NED	1999
	Femiano et al.,[10]	32/M	Buccal Mucosa	SE	*	2001
	Ide and Kusama [33]	50/F	Mandibular Gingiva	SE	20years/ NED	2002
	Yamada et al.,[34]	6m/M	Upper lip	SE	*	2002
	Alves et al., [35]	26/F	Buccal Mucosa	SE	24M/ NED	2003
	Hidaka et al.,[36]	2y8m/M	Maxillary Gingiva	SE	4M/ NED	2005
	Skoulakis et al.,[55]	19/M	Cheek	SE	*	2007
	Toyohara et al.,[37]	76/F	Upper Lip	SE	4years/ NED	2008
	Lee et al.,[38]	41/F	Upper lip	SE	*	2010

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	Giovani et al.,[5]	36/M	Buccal Mucosa	SE	12M/ NED	2010
	Bage et al.,[39]	59/F	Right Cheek	SE	14M/ NED	2010
	Pia et al.,[57]	8/F	Tongue	SE	*	2011
	Lopez Lornet et al.,[40]	8/F	Dorsum of Tongue	SE	*	2011
	Nur et al.,[58]	10/F	Extrnal Auditoy canal	SE	12M/ NED	2012
	Bindhu et al.,[41]	20/F	Hard Palate	SE	*	2012
	Rullo et al.,[23]	9m/M	Tongue	SE	*	2012
	Caldeira et al[42]	29/F	Hard palate	SE	*	2012
	Himanshu et al.,[58]	62/F	Buccal Mucosa	SE	12M/ NED	2012
	Rajathi et al.,[43]	23/M	Gingiva	SE	*	2013
	Priya et al.,[9]	30/F	Dorsum of Tongue	SE	3Y/ NED	2013
	Pandey et al.,[45]	26/M	Tongue	SE	*	2013
	Narendra et al.,[59]	26/M	Tongue	SE	*	2013
	Pradipta et al., [60]	45/M	Submandibular space	SE	14M/ NED	2013
	George et al.,[7]	37/F	Maxillary Gingiva	SE	18M/ NED	2014
	Srikanth et al., [20]	27/M	Subcutaneous- cheek	SE		2014
	Prisse et al.,[2]	48/F	Lower lip	SE	7M/ NED	2015
	Prisse et al.,[2]	75/M	Palate	SE	14M/ NED	2015

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	Prisse et al.,[2]	81/M	Soft and Hard Palate Junction	SE	18M/NED	2015
	Eun Jo et al.,[6]	36/F	Buccal Mucosa	SE	7M/NED	2015

There seems to be a consensus across literature on the treatment protocol of Benign Fibrous Histiocytoma. The treatment is surgical en-bloc resection of the tumour with a safety margin of 5mm and regular follow up upto 3 years. BFH has a malignant form, which is more often encountered in the literature, MFH is described as having a local aggressiveness and a low rate of metastasis [23, 11]. MFH is believed to be a primitive, pleomorphic sarcoma consisting partly of fibroblastic cells and partly histiocytic cells. Reported incidence of BFH to malignant transformation is 1% [24]

MFH has been an enigma since no true cell origin has been determined. WHO declassified MFH as a formal diagnostic entity and renamed it as undifferentiated pleomorphic sarcoma [21].

The prognosis of oral BFH is usually very good. A rare case of metastasis has been documented with the angiomatoid variant of Oral BFH [23].

There also is a case report of a malignant transformation of oral benign Fibrous Histiocytoma lesion which was treated with aggressive surgical management and chemo/radiotherapy [24].

We have carried out an exhaustive research of all the Oral Benign Fibrous Histiocytoma tumours documented in literature since 1961-2015 and we have tabulated the findings received (Table 3).

Table 4: Review of cases of BFH of Hard tissues in chronological order.

No. of cases	**Authors**	**Age/Sex**	**Location**	**Treatment**	**FU time/ Recurrence**	**Year**
1.	Cale et al., [46]	13/M	Posterior Maxilla	SE	14M/ NED	1983
2.	Ertas et al.,[47]	13/F	Anterior Mandible	SE	12M/ NED	2003
3.	Heo et al.,[48]	42/M	Posterior Mandible	SE	*	2004
4.	Kishino et al.,[49]	49/M	Posterior Mandible	SE	7M/ NED	2005

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5.	Katagiri et al.,[50]	48/M	Mandible-Condyle	SE	12M/ NED	2008
6.	Wagner et al.,[51]	41/M	Posterior Mandible	Piezoelectric assisted SE	10M/ NED	2011
7.	Gupta et al.,[52]	24/F	Posterior Mandible	SE	12M/ NED	2011
8.	De-ming Ou et al., [44]	31/M	Posterior Mandible	SE	14M/NED	2012
9.	Saluja et al.,[15]	23/F	Maxilla	SE	24M/ NED	2014
10.	Shoor et al.,[14]	30/F	Posterior Mandible	SE	24M/ NED	2015

Conclusion

An oral and maxillofacial surgeon may frequently encounter these tumours in the scope of their clinical practice. To the best of our understanding, oral BFH tumours have excellent prognosis and lesser chances of recurrences on management with complete surgical en bloc resection. These benign tumours show good loco regional behaviour post- surgical management. Chemo or Radiotherapy currently has no role in their management.

Thorough clinical history, prompt and correct diagnosis, complete excision with pathological margin clearance and regular follow up is imperative in the management of BFH.

However a complete understanding, knowledge and awareness of the innate behaviour of these tumours is an indispensable trait in a Maxillofacial Surgeon.

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