



Tonsillar p16-Positive Follicular Dendritic Cell Sarcoma Mimicking HPV-Related Oropharyngeal Squamous Cell Carcinoma: A Case Report and Review of Reported Cases

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Abstract

Follicular dendritic cell sarcoma (FDCS) is a rare entity which can share morphologic features with non-keratinizing squamous cell carcinoma. Recent reports suggest that up to half of FDCSs show immunohistochemical positivity for p16 (Zhang et al., in *Hum Pathol* 66:40–47, 2017), a stain that is conventionally used in the risk stratification of oropharyngeal squamous cell carcinoma (OPSCC). Herein, we report a case of p16-positive FDCS with clinical and histomorphologic overlap with human papilloma virus (HPV)-related OPSCC.

Introduction

Follicular dendritic cells contribute to the stroma of lymphoid tissues, where they are found in germinal centers and function to present antigens to B cells during the immune response. They are immunohistochemically characterized by positivity for follicular dendritic cell markers such as CD21, CD23, and CD35. Follicular dendritic cell sarcoma (FDCS) is a very rare malignant proliferation of follicular dendritic cells first described by Monda et al. in 1986 [1]. FDCS afflicts men and women equally, with a median age of 50 years [2] at the time of presentation. FDCS often presents as a painless, slow-growing nodal mass, although it can also arise in extranodal locations [3]. This malignant proliferation may arise in association with the hyaline vascular variant of Castleman Disease, but most frequently appears de novo. Rarely, it is associated with myasthenia gravis. Pathogenic alterations in genes involving the NFκB pathway and *BRAF* V600E are often present [4–6]. There is no standard treatment for FDCS, although most patients undergo complete surgical resection with or without subsequent radiation

and/or chemotherapy. Although FDCS typically follows a protracted clinical course with a median survival of > 10 years in patients with localized disease [2, 3], nearly half of patients experience local recurrence and/or distant metastasis after initial treatment. Herein we report an unusual case of FDCS of the tonsil with histologic p16 positivity mimicking a human papilloma virus (HPV)-related non-keratinizing oropharyngeal squamous cell carcinoma (OPSCC).

Case History

A previously healthy 39-year-old non-smoking man presented with 3 months of posterior throat fullness, excessive snoring during sleep, and episodic night sweats. He had no personal or family history of cancer. Physical exam revealed asymmetric tonsillar hypertrophy with an ill-defined mass of the right tonsil. A CT scan showed asymmetric enlargement of the right palatine tonsil to 2.8 cm in diameter, which was felt to be non-specific, representing either a mass or reactive hyperplasia. The remainder of the pharynx was normal and there was no lymphadenopathy (Fig. 1). A bilateral tonsillectomy was performed. Intraoperative examination of the oropharynx revealed a grade 4 right tonsil and a grade 2.5 left tonsil. The soft palate and uvula were unremarkable.

Gross examination of the tonsillectomy specimen revealed that the right tonsil measured 4.0 cm in maximum dimension and displayed a tan to pink, fleshy, smooth, homogenous cut surface, as opposed to the cerebriform texture of the grossly normal left tonsil. There was no evidence

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Fig. 1 A CT scan showed non-specific enlargement of the right palatine tonsil up to 2.8 cm in diameter. The oropharynx and neck were otherwise unremarkable

of a clonal lymphoproliferative disorder in either tonsil by flow cytometry.

On histology, the right tonsil contained a well-circumscribed but unencapsulated mass measuring 2.0 cm. The mass was composed of sheets and poorly formed fascicles of medium-sized epithelioid to ovoid cells with eosinophilic and somewhat fibrillary cytoplasm (Fig. 2). The cells had elongated nuclei with dispersed chromatin, occasional small nucleoli, and nuclear pseudoinclusions. Occasional multinucleated giant cells were also present. The mass contained a mild infiltrate of small, mature-appearing lymphocytes which clustered around the small vessels scattered throughout the lesion. There was no necrosis and mitotic figures were rare.

Immunohistochemical stains revealed that > 70% of the atypical cells were p16-positive (with p16-positive cells showing both cytoplasmic and nuclear staining), but negative for epithelial (OSCAR, pankeratin, p63, EMA), melanoma (HMB45, Mart 1, SOX10), muscle (desmin, myogenin, SMA), schwannoma (S100), neuroendocrine (synaptophysin, chromogranin), lymphoid (CD45), and Langerhans cell (CD1a, langerin) markers (Fig. 3). INI

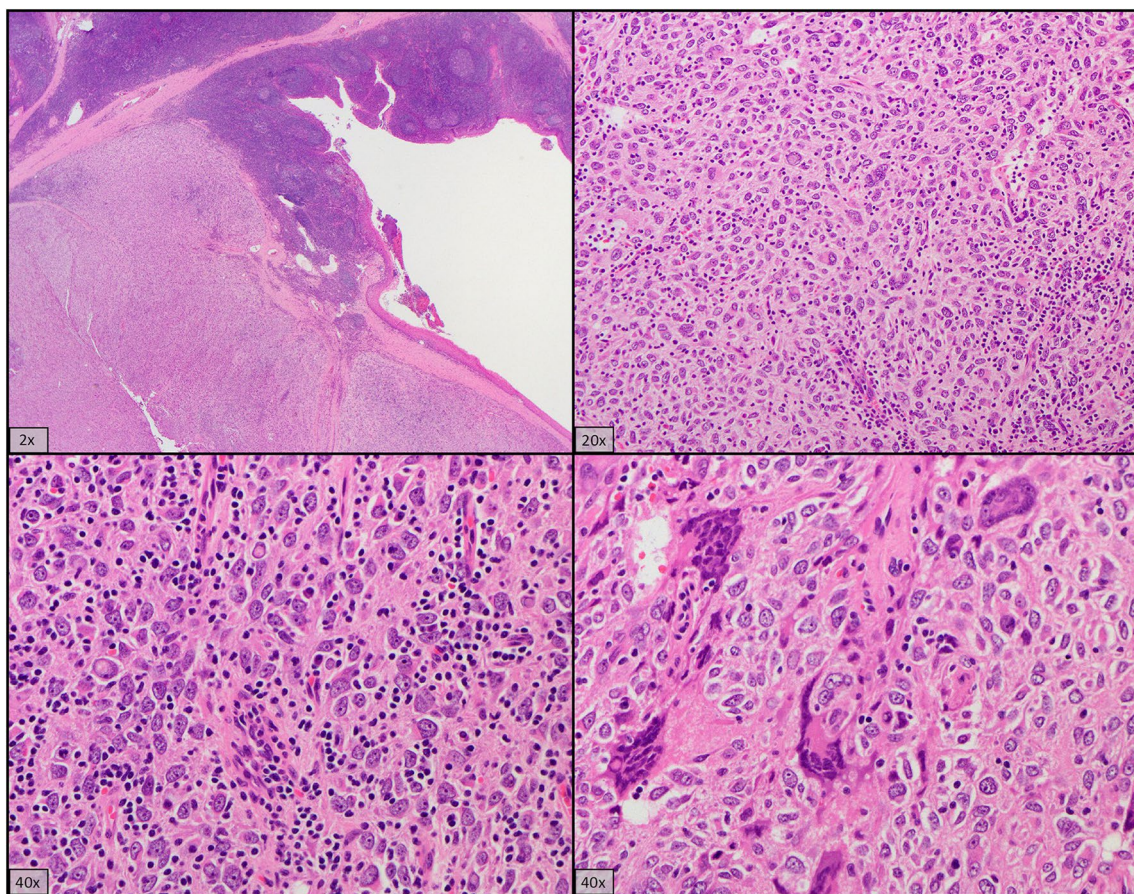


Fig. 2 The well-circumscribed mass consisted of sheets of epithelioid cells with pseudonuclear inclusions. Patchy lymphocytic infiltrates and giant cells were present

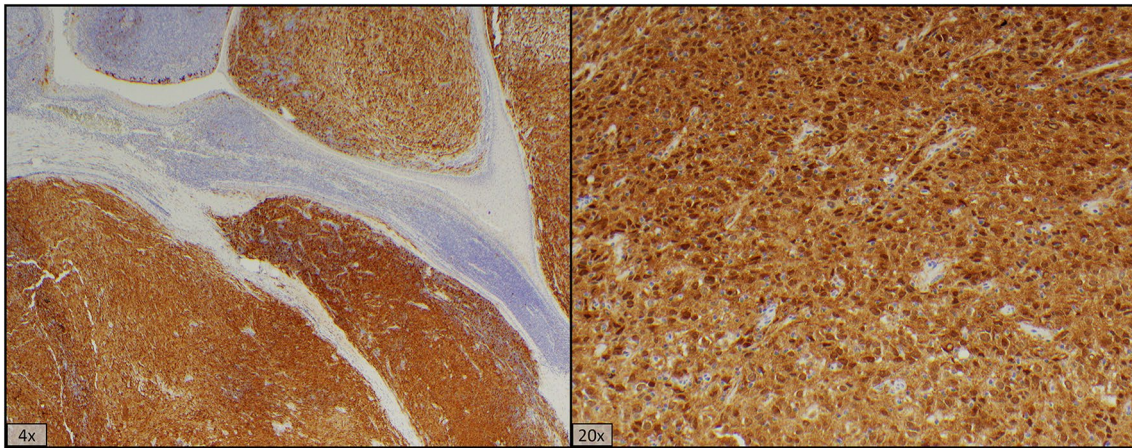


Fig. 3 p16 immunostaining shows cytoplasmic and nuclear positivity in > 70% of lesional cells

expression was retained. In situ hybridization (ISH) revealed no evidence of Epstein-Barr virus (EBV) by EBER or HPV 16/18. The proliferation index was 30% via Ki67 staining. PCR for high-risk HPV was negative.

Although initial suspicion for HPV-related OPSCC was high given the congruent clinical presentation, histology on routine stains, and p16 positivity, the lesion was negative for keratins and showed no evidence of HPV by ISH or PCR, making this diagnosis unlikely. Upon further examination, the fibrillary quality of the cytoplasm and the nuclear pseudoinclusions seen within the lesional cells, as well as the presence and pattern of the lymphocytic infiltrate within the lesion raised suspicion for FDCS. Immunohistochemical stains for follicular dendritic cell markers CD21, CD23, and CD35 were added and were strongly and diffusely positive (Fig. 4).

On this basis, the patient was diagnosed with follicular dendritic cell sarcoma. Follow-up immunohistochemical staining for BRAF V600E was negative. An additional stain revealed retention of Rb within the lesional cells.

Following initial resection of the mass, re-excision of the tumor bed was performed revealing no residual tumor. The patient was screened for Castleman's Disease and myasthenia gravis and was negative for both. Plans were made to monitor closely via imaging.

Discussion

Follicular dendritic cell sarcoma is a rare entity whose clinical and histologic presentation as a tonsillar mass can closely mimic a non-keratinizing oropharyngeal squamous cell carcinoma (OPSCC). Both entities frequently present as a painless mass in the head and neck of a middle-aged male, as in this case. Moreover, a FDCS composed primarily of epithelioid dendritic cells, as presented here, exhibits

histomorphologic overlap with non-keratinizing squamous cell carcinoma. This poses a diagnostic pitfall, particularly in the pharyngeal region where carcinomas are much more common.

A literature review performed by Duan et al. found that 58% of cases of extranodal FDCS were initially misdiagnosed, often mistaken for squamous cell carcinoma or undifferentiated carcinoma [7]. Further complicating the distinction between FDCS and epithelial lesions, a subset of FDCSs show focal positivity for epithelial membrane antigen (EMA; not present in this case) [8].

Lymphoepithelioma-like carcinoma also shares some histologic features with FDCS, although this tumor is usually EBV-related (and therefore EBER-positive) and includes a more prominent inflammatory component within the carcinoma. In the case reported here, the diffuse negativity of the lesional cells for keratins helped exclude carcinoma from the differential diagnosis.

The histologic differential diagnosis in this case also included metastatic melanoma (excluded by immunohistochemical negativity for S100, HMB-45, Mart 1, and SOX10). Negativity for S100, along with CD45, also helped exclude interdigitating dendritic cell sarcoma, which has significant cellular morphologic overlap with FDCS, including the presence of admixed of small lymphocytes. Langerhans cell histiocytosis was also excluded by negativity for S100, as well as for CD1a and langerin. Ultimately, the strong and diffuse immunohistochemical positivity for follicular dendritic cell markers CD21, CD23, and CD35 confirmed the final diagnosis.

While the epithelioid morphology of the lesional cells in this case resembled carcinoma, neoplastic follicular dendritic cells are often spindled, invoking a differential of other spindle-cell sarcomas. Yet another differential that warrants consideration is the inflammatory pseudotumor-like variant of FDCS, which is composed of follicular dendritic cells

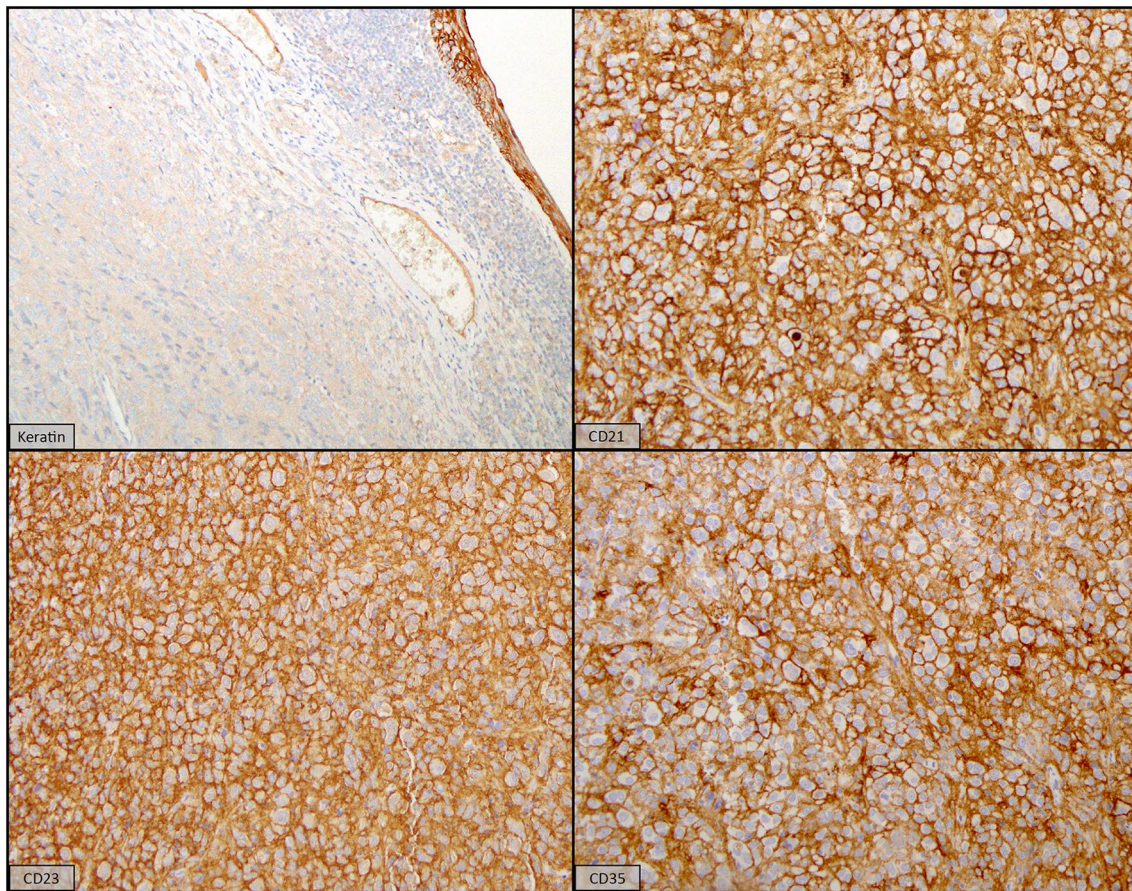


Fig. 4 The lesional cells were negative for cytokeratin and were strongly positive for dendritic cell markers CD21, CD23, and CD35

dispersed in a heavily inflammatory background of lymphocytes and plasma cells and often shows fibrinous changes of intratumoral vasculature. This variant arises most frequently in the spleen or liver of female patients and unlike other FDSCs, is frequently associated with EBV (and is thus EBER-positive by immunohistochemistry and ISH) [9]. Occasionally, the cytomorphology of the lesional cells in FDSC may resemble the Reed-Sternberg cells of Hodgkin lymphoma. These lesions are distinguishable from true Hodgkin lymphoma by a paucity of granulocytes in the inflammatory milieu as well as the diffuse architecture of atypical cells, which would be fewer and more dispersed in Hodgkin lymphoma.

The differential diagnosis of FDSC is broad, but it can often be limited by anatomic location of the lesion. In the case of a tonsillar lesion, an OPSCC is a particularly salient exclusion. FDSCs are characteristically indolent tumors, with histologically low-grade FDSCs having better clinical outcomes than their high-grade counterparts. Namely, intra-abdominal location, size ≥ 6 cm, the presence of coagulative necrosis, the presence of ≥ 5 mitotic figures per 10 high power fields, and prominent nuclear atypia have all been

demonstrated to portend a poorer prognosis [2, 10]. This patient's tumor lacked these characteristics, and as such, treatment with complete surgical excision alone would be appropriate. In contrast, an HPV-related OPSCC may be treated with radiation, chemoradiation, or surgical excision with or without radiation depending upon tumor size, margin, and lymph node status [11].

HPV-related non-keratinizing OPSCC is increasing in incidence in the United States and worldwide, affecting younger, healthier patients and showing a significantly better prognosis than non-HPV-related OPSCC [7]. Because of this, the American Joint Committee on Cancer (AJCC) and the College of American Pathologists (CAP) recommend immunohistochemical staining for p16 on all OPSCCs to determine HPV status [12, 13]. In order to be considered positive, the p16 must stain with at least moderate intensity in $> 70\%$ of lesional cells [13], including both nuclear and cytoplasmic staining.

A recent case series by Zhang et al. demonstrated that 50% of the eight FDSC cases studied showed some p16 positivity by immunohistochemical staining, while 25% showed p16 staining of a strength and quantity sufficient to meet

Table 1 Cases of oropharyngeal follicular dendritic cell sarcoma in the current English language literature

References	Patient age (years)/sex	Tumor location	p16 IHC
Chan et al. [14]	63/F	Soft palate	Not reported
Chan et al. [14]	44/M	Left tonsil	Not reported
Perez-Ordóñez et al. [15]	62/F	Tonsil	Not reported
Naylor et al. [16]	18/F	Tonsil	Not reported
Chan et al. [17]	32/M	Right tonsil	Not reported
Biddle et al. [18]	48/F	Left tonsil	Not reported
Biddle et al. [18]	48/M	Right tonsil	Not reported
Biddle et al. [18]	33/M	Right oropharynx/nasopharynx	Not reported
Vargas et al. [19]	54/F	Left tonsil and cervical lymph node	Not reported
Tisch et al. [20]	51/M	Left palatine tonsil	Not reported
Satoh et al. [21]	16/M	Right palatine tonsil	Not reported
Idrees et al. [22]	77/F	Palatine tonsil	Not reported
Grogg et al. [23]	57/F	Tonsil, cervical lymph node, axillary lymph node	Not reported
Domínguez-Malagón et al. [24]	48/M	Left tonsil	Not reported
Chou et al. [25]	61/M	Soft palate	Not reported
Bothra et al. [26]	40/M	Left tonsil	Not reported
Bothra et al. [26]	45/M	Right tonsil	Not reported
Bothra et al. [26]	34/M	Right tonsil	Not reported
Clement et al. [27]	27/F	Right tonsil	Not reported
Aydin et al. [28]	76/F	Left tonsil	Not reported
Shia et al. [29]	69/F	Tonsil	Not reported
Fan et al. [30]	33/F	Right tonsil	Not reported
McDuffie et al. [31]	59/F	Right tonsil	Not reported
Vaideeswar et al. [32]	50/F	Left tonsil	Not reported
Eun et al. [33]	65/M	Right tonsil	Not reported
Suhail et al. [34]	52/F	Right tonsil	Not reported
Duan et al. [7]	41/M	Tonsil	Not reported
Duan et al. [7]	39/F	Soft palate	Not reported
Li et al. [35]	60/M	Tonsil	Not reported
Li et al. [35]	55/M	Tonsil	Not reported
Suchitha et al. [36]	63/M	Left tonsil	Not reported
Mondal et al. [37]	27/M	Left tonsil	Not reported
Kara et al. [38]	72/M	Right tonsil	Not reported
Hu et al. [39]	36/F	Left tonsil and cervical lymph node	Not reported
Hu et al. [39]	59/F	Left tonsil	Not reported
Vorsprach et al. [40]	24/M	Left tonsil	Not reported
Horváth et al. [41]	55/not reported	Left tonsil	Not reported
Kulkarni et al. [42]	30/F	Left tonsil	Not reported
Lu et al. [43]	59/M	Right tonsil	Not reported
Wang et al. [8]	65/M	Tonsil	Not reported
Griffen et al. [6]	26/M	Tonsil/neck	Not reported
Amirtham et al. [44]	63/M	Left tonsil	Not reported
Amirtham et al. [44]	28/M	Right tonsil	Not reported
Amirtham et al. [44]	66/M	Right tonsil	Not reported
Amirtham et al. [44]	68/F	Left tonsil	Not reported
Amirtham et al. [44]	65/F	Left tonsil	Not reported
Amirtham et al. [44]	40/M	Left tonsil	Not reported
Amirtham et al. [44]	51/F	Left tonsil	Not reported
Amirtham et al. [44]	38/M	Right tonsil	Not reported

Table 1 (continued)

References	Patient age (years)/sex	Tumor location	p16 IHC
Agaimy et al. [45]	75/F	Posterior oropharynx + nasopharynx	Not reported
Pang et al. [46]	32/M	Left tonsil	Negative
Pecorella et al. [47]	60/F	Left tonsil	Not reported
Zhang et al. [10]	35/F	Right tonsil	Negative
Wu et al. [48]	52/M	Left tonsil	Not reported
Lopez-Hisijos et al. [49]	46/M	Base of tongue	Not reported
Current case	39/M	Right tonsil	Positive

AJCC criteria for p16 positivity [10], as in this case. To our knowledge, there are only two other reports of p16-positivity in FDCS in the current English language literature. Although there are over 40 reports of oropharyngeal FDCS, the majority do not document lesional p16 expression by immunohistochemistry (Table 1). Of the three cases of oropharyngeal FDCS for which p16 immunohistochemistry results were reported, only our oropharyngeal case demonstrates p16 positivity.

While p16 overexpression in HPV-related OPSCC is due to viral inactivation of Rb leading to removal of Rb's negative regulation of p16, the mechanism of p16 overexpression in FDCS seems to vary and may hint at the lesion's molecular drivers. Recent targeted molecular analyses of FDCSs indicate that a minority of cases show alterations in Rb, while a larger number of FDCSs harbor alterations in *CDKN2A*, the gene which encodes p16 [5, 6]. It is possible that *CDKN2A* alterations lead to paradoxical overexpression of p16 due to downstream inactivation of Rb, again resulting in removal of the negative regulation by Rb of p16 [10]. In our case, immunohistochemical stains revealed both retention of Rb and negativity for the *BRAF* V600E mutation (another mutation recently identified as a possible pathogenic driver in FDCS [4]), raising the possibility that an alteration in *CDKN2A* may be driving the observed p16 overexpression.

Such p16-positive cases of FDCS can prove treacherous for the pathologist, who risks misclassifying these lesions as non-keratinizing OPSCCs. Because of the rarity of FDCS, p16 positivity may falsely affirm the diagnosis of HPV-related OPSCC if FDCS is not considered in the differential diagnosis. For rare cases such as the one presented here, the lack of reactivity for cytokeratins or p40 should alert the pathologist to this potential diagnostic pitfall. Subtle histomorphologic clues such as nuclear pseudoinclusions, which are present in approximately 43% of FDCSs [2], a lack of cohesive nests of cells, a lymphocytic infiltrate with perivascular cuffing [50], or fibrillary pink cytoplasm in the lesional cells may also raise suspicion for FDCS. If considered in the differential diagnosis, FDCS is easily diagnosed by positive staining for CD21, CD23 and/or CD35.

Conclusions

Although rare, FDCS should be considered in the differential of spindle to ovoid-cell neoplasms in the oropharynx. In p16-positive oropharyngeal lesions showing the subtle but characteristic histologic features of FDCS (spindled to ovoid cells with pseudonuclear inclusions and fibrillary cytoplasm) as in the case presented here, additional immunostaining with cytokeratin and follicular dendritic cell markers should be used to confirm or exclude the diagnosis [1].

Compliance with Ethical Standards

Conflict of interest All authors declare that they have no conflict of interest.

Ethical Approval This article does not contain any studies with human participants or animals performed by any of the authors.

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