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ORIGINAL PAPER

Primary Tonsillar Epithelioid Follicular Dendritic Cell Sarcoma: Report of a Rare Case Mimicking Undifferentiated Carcinoma and a Brief Review of the Literature

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Abstract
We present a 52 years old male with a left tonsillar follicular dendritic cell sarcoma with prominent epithelioid features that on light microscopical examination bore a striking resemblance to a lymphoepithelial or undifferentiated carcinoma. The tumor was immunohistochemically positive for CD21 and CD35 and negative for cytokeratins. Two distinct histopathological features (both present in our case) that may serve as clues to the correct diagnosis on light microscopical examination were formation of ectatic pseudovascular spaces lined by malignant cells and the presence of non-neoplastic multinucleated giant cells. Familiarity with the above-mentioned morphological clues, and awareness that this tumour may occur in anatomical sites outside the lymph node, are essential for accurate diagnosis.

Keywords Dendritic cell sarcoma · Follicular · Palatine tonsil · Carcinoma · Oropharynx

Introduction

Follicular dendritic cell sarcoma (FDCS) was first described in 1986 by Monda et al. in a series of four cases with initial presentation of unilateral cervical lymphadenopathy [1]. The age range is wide, but FDCS commonly occurs in young to middle-aged adults. There is no gender predilection, except for a higher incidence among women for the inflammatory pseudotumor-like variant, which is consistently associated with Epstein–Barr virus (EBV), which selectively involves the liver and spleen and is clinically often associated with systemic symptoms. FDCS may be more prevalent in East Asia [2]. Approximately 10 to 20% of FDCS is associated with Castleman's Disease, predominantly the hyaline vascular variant [3].

Follicular dendritic cell sarcomas (FDCS) may occur in both nodal and extra-nodal sites and the range of extranodal distruibution is very wide [2]. Herein we report a case of a primary extranodal, tonsillar FDC which displayed a significant component of epithelioid neoplastic cells which gave an initial impression of a lymphoepithelial/undifferentiated carcinoma.

Case Report

The patient is a 52 years old male who presented with an ulcerated left tonsillar mass. He had a past medical history of scleroderma, for which he was treated with methotrexate for many years. A computed tomography (CT) scan of the neck, performed at another institution, revealed a lobulated left tonsillar mass, which measured $4.1 \times 2.8 \times 2.6$ cm in size. The tumor bulged laterally into the left parapharyngeal space. No overt evidence of tumor invasion into the surrounding structures was discerned on imaging. A positron emission tomography (PET) scan, performed at another institution, revealed only intense uptake in the left tonsillar region, corresponding to the tumor site. Hence, there was no other evidence of tumor (primary or metastatic). After biopsy, the left tonsillar tumor was resected.

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Results

Gross Features

The left oropharyngectomy resection specimen showed a partially ulcerated, circumscribed, firm white tumor measuring 3.3 × 2.7 × 1.5 cm in size. The tumor was very close (< 0.1 cm) from the deep resection margin.

Histopathology

The pre-operative biopsy and the post-operative resection specimen showed similar histologic features. The tumor was predominantly composed of syncytial sheets of atypical epithelioid-appearing cells with enlarged nuclei, marked nuclear membrane irregularity, vesicular chromatin, and occasional discrete single nucleolus, amidst a lymphocyte-rich background (Fig. 1a, b). In some areas, the tumor cells displayed a spindled morphology and were arranged in vague fascicles (Fig. 1c). On close inspection several foci contained scattered multinucleated giant cells (Fig. 1d). In addition, several dilated pseudo-vascular spaces lined by tumor cells were also present (Fig. 1e). These dilated spaces were predominantly located at the periphery of the tumor. No tumor necrosis was present. The mitotic activity was variable; up to 5–8 mitotic figures per 10 high power fields. There was an accompanying histiocyte-rich, vague granulomatous infiltrate without necrosis. Ziehl-Neelsen, Periodic Acid-Schiff, and Warthin-Starry stains were negative for acid-fast bacilli, fungal organisms, and spirochetes respectively.

Immunohistochemistry and In-Situ Hybridization

The immunohistochemical study showed that the tumor cells diffusely expressed CD21 and CD35 (Fig. 2a, b). The Ki-67 proliferation index was approximately 40%. The neoplastic cells did not express cytokeratins (AE1/AE3, CAM5.2), S100-protein, Melan A, Desmin, CD68, CD163, or CD45. The tumor cells were negative for EBV (EBER) on in-situ hybridization.

The multinucleated giant cells were negative for CD163, CD21 and CD35.

Discussion

Follicular dendritic cell sarcomas (FDCS) are rare neoplasms that may occur in nodal and extra-nodal sites [3]. Extra-nodal sites include tonsils, nasopharynx, pancreas, peripancreatic and peritoneal tissue, but a very wide range of anatomical locations are on record [2, 4]. FDCs arise from antigen-presenting cells within germinal centers of lymphoid follicles and retain many of the immunophenotypical features of these cells [4]. The fact that FDCSs retain protein expression akin to the non-neoplastic cellular counterpart is of great value in establishing the diagnosis. However, the main difficulty with correctly diagnosing this tumor is the rarity with which they occur and this is especially true at extra-nodal sites. The difficulty is further compounded by the wide spectrum of cyto- and histopathological patterns that FDCS may exhibit [4].

On histological examination, FDCSs are most often composed of oval to short spindle neoplastic cells with storiform, whorled, fascicular, and/or sheet-like architectural patterns. A combination of different architectural patterns may be encountered within the same tumor [4]. These tumors contain populations of reactive T and B cells. The neoplastic cells generally exhibit ovoid vesicular nuclei with small nucleoli, and syncytial eosinophilic cytoplasm. The degree of nuclear atypia/pleomorphism is variable; most cases demonstrate mild nuclear atypia, but marked nuclear pleomorphism can also be present and is associated with/ predicts aggressive behaviour [5, 6]. Similarly, mitotic activity can be variable and increased mitotic activity (more than 5 mitoses per 10 high power fields) is associated with more aggressive behaviour [5, 6]. Useful histologic features associated with FDCS that may serve as clues to the correct diagnosis include multinucleated giant cells and dilated ectatic pseudo-vascular spaces lined by tumor cells [3–6].

The differential diagnoses for FDCS depend on the site and histological appearance. The main key to the diagnosis is the awareness that FDCS may occur in a variety of anatomical subsites and may exhibit a wide range of histopathological features. Once the diagnostic hypothesis has been formulated an adequate immunohistochemical study that includes specific follicular dendritic cell (FDC) markers such as CD21, CD23 and CD35 will clinch the diagnosis [7]. Recent suggested novel markers for FDCS are claudin4, clusterin, Follicular Dendritic Cell Secretory Product (FDCSP) and Serglycin (SRGN) [8]. While these novel markers have been described to be highly sensitive and specific for FDCS, it remains to be seen what role they will play in a clinical diagnostic setting. It is important to remember that FDCS can also be positive for epithelial membrane antigen (EMA), S100-protein, and podoplanin [3]. Common diagnostic pitfalls reported in the literature include ectopic meningioma (whorled, syncytial appearance as well as EMA and podoplanin expression), melanoma (S100-protein), and undifferentiated carcinoma (syncytial appearance and EMA expression) [3, 4].

In our case, the histologic features bear marked resemblance to lymphoepithelial/undifferentiated oropharyngeal (OP) carcinoma. These tumors may (in the OP) be associated

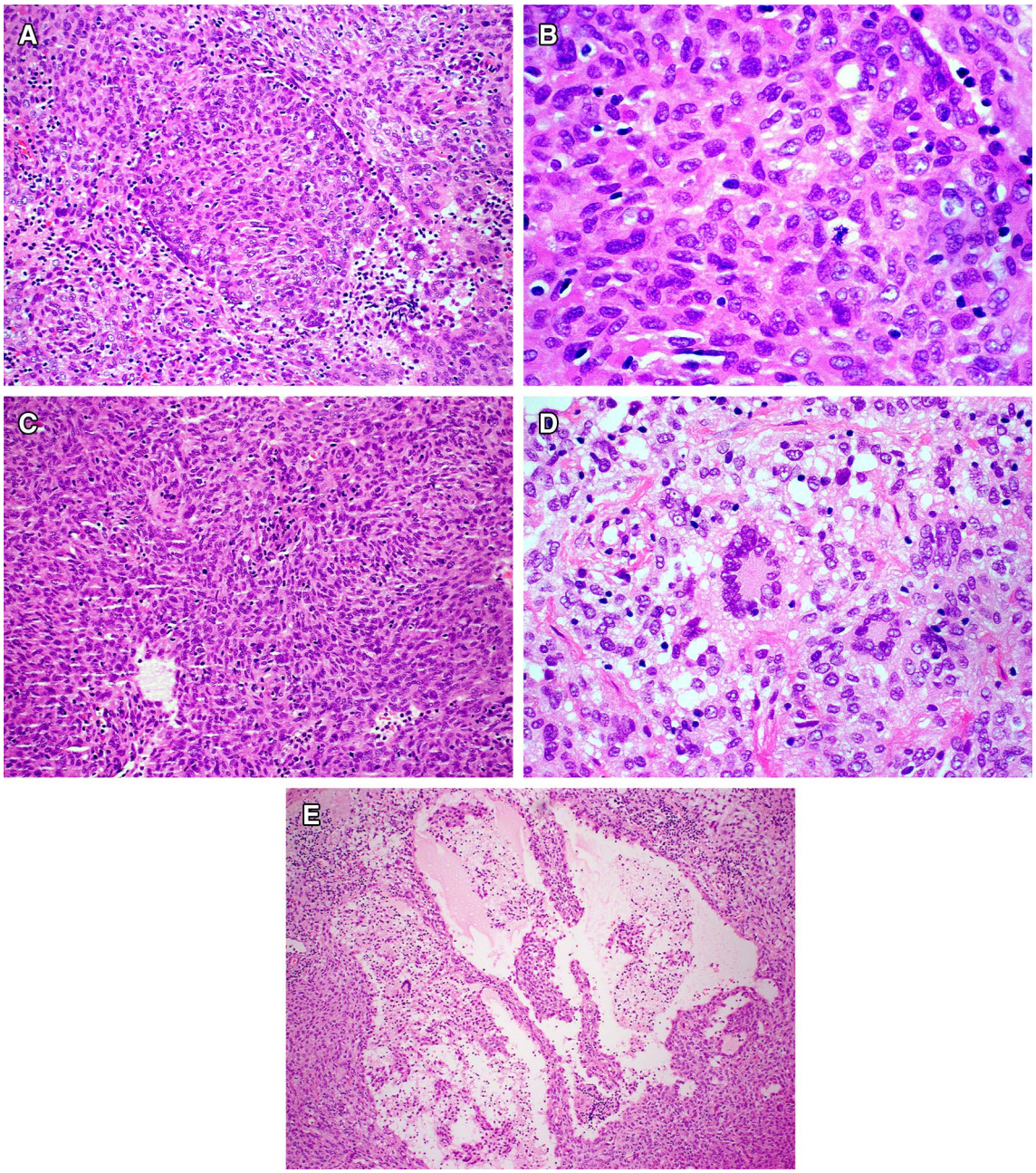

Fig. 1 **a** The tumor was predominantly composed of syncytial sheets of atypical epithelioid-appearing cells amidst a lymphocyte-rich background. Hematoxylin and Eosin stain, ×100 magnification. **b** The tumor cells exhibited enlarged nuclei, marked nuclear membrane irregularity, vesicular chromatin, and occasional discrete single nucleolus, Hematoxylin and Eosin stain, ×400 magnification. **c** In some areas, the tumor cells displayed a spindled morphology and were arranged in vague fascicles. Hematoxylin and Eosin stain, ×100 magnification. **d** On close inspection several foci contained scattered multinucleated giant cells. Hematoxylin and Eosin stain, ×400 magnification. **e** Occasional significantly dilated ectatic pseudo-vascular spaces lined by tumor cells were also present. Hematoxylin and Eosin stain, ×40 magnification

with either human papilloma virus (HPV) or Epstein Barr virus (EBV) [9–11]. Notably, the present tumor was in large areas composed of syncytial sheets of mitotically active atypical epithelioid-appearing cells with enlarged nuclei, marked nuclear membrane irregularity, vesicular chromatin pattern, and occasional discrete single nucleolus, amidst a lymphocyte-rich background. There was a component of tumor cells with a spindle cells in the case presented herein. This may, however, also be encountered in undifferentiated and lymphoepithelial carcinomas. In contrast, the presence of multinucleated giant cells and dilated ectatic pseudo-vascular spaces lined by neoplastic cells, which has been

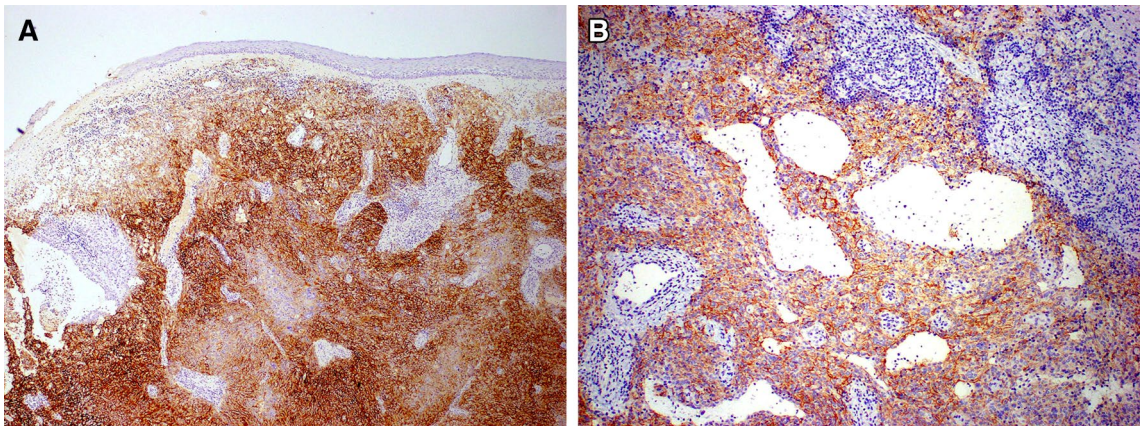

Fig. 2 **a** The tumour cells diffusely expressed CD21. Immunohistochemistry, CD21 antibody, ×20 magnification. **b** The tumour cells diffusely expressed CD35. Immunohistochemistry, CD35 antibody, ×40 magnification

previously described in FDCS [5, 6], has to the best of our knowledge not been described in OP lymphoepithelial/undifferentiated carcinoma.

Based on the light microscopic features, one differential diagnosis that also could be considered is an angiomatoid fibrous histiocytoma (AFH). These tumors most often contain lymphoid cells and display a spectrum of morphological features that includes sheets to large nodules and vague fascicular patterns and often contain spindled and ovoid-epithelioid neoplastic cells featuring mitotic activity, some degree (up to moderate) of nuclear atypia and "angiomatoid" spaces lined by neoplastic cells. These spaces may or may not contain blood. In some AFHs there is a component of non-neoplastic histiocytic-type foam cells. However, although 5–10% of AFH occur in the head and neck region, to the best of our knowledge, no case of AFH has been described in the palatine tonsils.

While multinucleated giant cells have been described in FDCS [3], the nature of these giant cells remain indeterminate. Our immunohistochemical work up revealed these giant cells to be negative for follicular dendritic cell markers CD21 and CD35. Hence, these giant cells are not tumour cells, or of follicular dendritic cell lineage. These giant cells are also negative for CD163. Hence, these giant cells are unlikely to be of monocyte/macrophage lineage either.

Head and neck FDCS, and in particular primary FDCS of the tonsils, are uncommon [4, 12]. To the best of our knowledge, to date only 35 cases of primary FDCS of the tonsils, including this case, have been reported in the English language literature (Table 1). The rarity of this tumor in the tonsil renders the diagnosis challenging and awareness of this possibility is critical in initiating the process, e.g. immunohistochemistry for arriving at the correct diagnosis.

Owing to the overall rarity of FDCS, optimal treatment recommendations have not been well defined. Localized disease is primarily treated with surgery [2, 39]. A role for adjuvant radiation therapy and chemotherapy has not been well established [2, 40, 41]. However, radiation therapy and chemotherapy may be used in cases of recurrent or refractory disease [31, 42].

FDCS were initially thought to behave like low grade sarcomas, but subsequent studies have indicated a propensity for loco-regional recurrence and distant metastases. Metastatic sites have included lung, liver and lymph nodes. This implies that FDCS behaves more like an intermediate grade sarcoma [3, 5]. Histologic predictors of aggressive behavior include nuclear pleomorphism, high mitotic activity (more than 5 mitoses per 10 high power fields), tumor necrosis and large tumor size [3, 5, 7]. Based on our literature review on FDCSs of the tonsil (Table 1), it is apparent that the majority of patients did not suffer local recurrence or metastatic disease progression irrespective of whether they were treated with surgery (with or without neck dissection) alone or a combination of surgery radio- and/or chemotherapy. However, approximately 25% of patients suffered disease progression; local recurrence and or metastatic disease.

In the case presented herein, the mitotic activity was focally high. The light microscopical impression was supported immunohistochemically which showed a Ki-67 proliferation index of up to 40%. Following surgical resection with very close margins, the patient was offered adjuvant chemotherapy and radiation therapy, but he declined both. The present case was recently diagnosed which thus precludes meaningful follow-up. The patient is currently on close follow up.

Conclusion

In conclusion, we herein describe the clinicopathological features of a patient with a primary tonsillar follicular dendritic cell sarcoma which on light microscopy bore a

Table 1 Previously reported cases of primary tonsillar follicular dendritic cell sarcoma

Case no.	Age (years)	Gender	Tumor size	Initial treatment	Outcome (duration of follow up)
1 [13]	76	Female	3.5 cm	Resection and adjuvant radiation therapy	No evidence of disease after (48 months)
2 [4]	48	Female	3.5 cm	Resection and neck dissection	No evidence of disease (6 months)
3 [4]	48	Male	3.5 cm	Resection only	No evidence of disease (8 months)
4 [14]	40	Male	Not available	Resection only	No evidence of disease (12 months)
5 [14]	45	Male	Not available	Resection only	No evidence of disease (12 months)
6 [14]	34	Male	Not available	Resection only	No evidence of disease (120 months)
7 [15]	44	Male	1.5 cm	Resection only	No evidence of disease (36 months)
8 [16]	18	Female	2 cm	Resection and adjuvant chemotherapy (CHOP)	Not available
9 [17]	27	Female	4 cm	Resection, neck dissection and adjuvant radiation therapy	No evidence of disease (6 months)
10 [18]	48	Male	1.5 cm	Resection, neck dissection and radiation therapy	No evidence of disease (36 months)
11 [19]	41	Male	3 cm	Resection only	No evidence of disease (9 months)
12 [20]	65	Male	3 cm	Resection and adjuvant radiation therapy	No evidence of disease (24 months)
13 [21]	48	Female	Not available	Resection, adjuvant chemotherapy and radiation therapy	Disease recurrence (180 months)
14 [22]	57	Female	Not available	Resection only	Alive with disease (8 months)
15 [23]	36	Female	3 cm	Resection only	Disease recurrence (6 months)
16 [23]	59	Female	4.5 cm	Resection only	Disease recurrence (17 months)
17 [24]	77	Female	Not available	Pre-operative radiation therapy, resection and radical neck dissection	Lung and lymph node metastases (96 months)
18 [25]	72	Male	5 cm	Resection and adjuvant chemotherapy	Dead; unknown cause of death (12 months)
19 [26]	30	Female	2.2 cm	Resection	No evidence of disease (6 months)
20 [27]	60	Male	5 cm	Resection and adjuvant radiation therapy	No evidence of disease (86 months)
21 [27]	55	Male	2 cm	Resection and adjuvant radiation therapy	Recurrent disease (18 months), alive with disease (21 months)
22 [28]	59	Male	4.6 cm	Resection and adjuvant radiation therapy	No evidence of disease (44 months)
23 [29]	59	Female	4 cm	Resection and adjuvant radiation therapy	No evidence of disease (18 months)
24 [30]	27	Male	2.8 cm	Resection and adjuvant radiation therapy	No evidence of disease (6 months)
25 [31]	32	Male	3.3 cm	Resection, neck dissection, adjuvant chemotherapy and adjuvant radiation therapy	Lung and liver metastases (48 months)
26 [6]	62	Female	Not available	Resection	No evidence of disease (36 months)
27 [32]	69	Female	Not available	Pre-operation radiation therapy, resection and radical neck dissection	Lung and lymph node metastases (96 months)
28 [33]	63	Male	4.2 cm	No treatment given	Alive with disease (8 months)
29 [34]	52	Female	2.5 cm	Resection and adjuvant chemotherapy	No evidence of disease (12 months)
30 [35]	51	Male	Not available	Resection and adjuvant radiation therapy	No evidence of disease (60 months)
31 [36]	50	Male	2.5 cm	Resection	No evidence of disease (48 months)
32 [37]	54	Female	3 cm	Resection and radical neck dissection	No evidence of disease (8 months)
33 [38]	24	Male	2.5 cm	Resection	No evidence of disease (duration of follow up not available)
34 [12]	60	Female	6 cm	Resection	Lost to follow up
35 (current case)	52	Male	3.3 cm	Resection	No meaningful follow up at the time of publication

striking resemblance to a lymphoepithelial or undifferentiated carcinoma. The awareness that this tumor type may occur in many anatomical sites outside the lymph nodes and exhibits a wide range of histopathological appearances, is critical in establishing the correct diagnosis. Pertinent morphological clues that should trigger the consideration of a FDCS diagnosis include pseudo-vascular spaces and multinucleated giant cells. Subsequent CD21 and CD35

immunohistochemistry would then readily confirm the morphological impression of FDCS.

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Compliance with Ethical Standards

Conflict of interest The author has no conflict of interest.

Ethical Approval It is our institution's policy not to require formal ethical approval for reports on up to two patients.

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