Diagnostic Pathology

CASE REPORT Open Access

Description of two cases of follicular dendritic cell sarcoma, including next-generation sequencing analysis

Yuchen Jing[1,2], Hua Ye[1], Shuai Luo[1] and Jinjing Wang[1*]

Abstract

Follicular dendritic cell sarcoma (FDCS), an infrequent malignancy, poses diagnostic challenges due to its nonspecific clinical presentations and propensity for recurrence and metastasis, particularly when assessed through imaging modalities. Accurate diagnosis relies heavily on pathological morphology and immunohistochemical analysis. This study examines two FDCS cases from the Affiliated Hospital of Zunyi Medical University. Next-generation sequencing (NGS) identified three gene rearrangements—*HFM1::BIRC3*, *ELF4::AIFM1*, and *DIP2B::WIF1*—in one case, while no genetic alterations were detected in the other. The report explores clinicopathological characteristics, molecular genetics, differential diagnosis, therapeutic approaches, and prognosis to enhance diagnostic and pathological understanding of FDCS in medical practice.

Keywords Follicular dendritic cell sarcoma, Pathology, Next-generation sequencing, Differential diagnosis, Treatment

Background

Follicular dendritic cell sarcoma (FDCS) represents a rare malignant neoplasm originating from follicular dendritic cells (FDCs) within germinal centers, frequently exhibiting lymphatic or extranodal dissemination. The World Health Organization (WHO) categorizes it as an infrequent tumor, defined by spindle-shaped or oval cells exhibiting morphological and immunophenotypic characteristics of dendritic cells [1]. Initially described by Monda L et al. in 1986 [2], FDCS was predominantly associated with lymph nodes and historically referred to as reticular cell sarcoma or dendritic reticular cell sarcoma. Chan et al. subsequently documented a case with extranodal involvement in 1994 [3]. The rarity of this entity, coupled with its considerable histopathological variability, often leads to nonspecific clinical manifestations that contribute to diagnostic challenges and potential delays.

Case presentation

Case One: A 31-year-old male with a history of a right chest wall mass since childhood presented on October 17, 2022, following its progressive enlargement over the past three months. The patient denied fever or significant weight loss and reported a 17-year smoking history. The mass, situated beneath the right chest wall, measured approximately 10 × 20 mm, was non-tender, mobile, and not adherent to surrounding structures. Although the mass had been previously neglected, the patient sought medical attention after noticing its recent enlargement, which was accompanied by tingling sensations. Ultrasound imaging revealed a heterogeneous mass,

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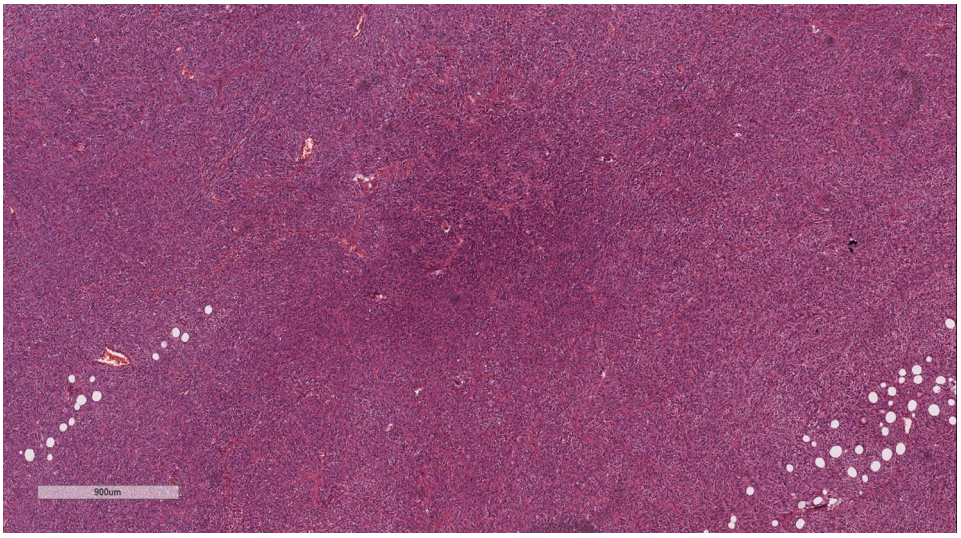

Fig. 1 Tumor cells display a sheet-like arrangement (HE×23)

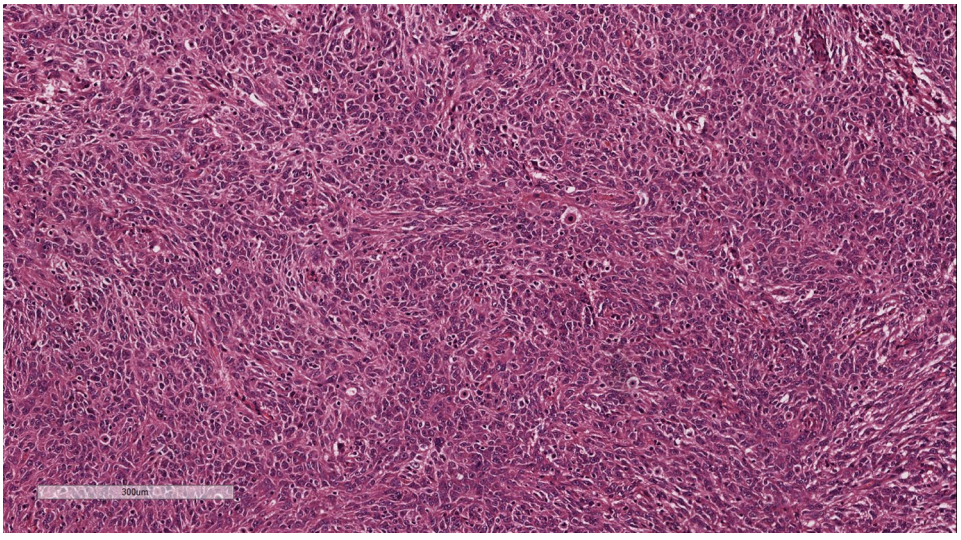

Fig. 2 Tumor cells exhibit a whirlpool pattern, resembling meningioma (HE×100)

measuring approximately 107 × 100 × 57 mm, located in the right axillary region. The mass exhibited an irregular shape with punctate blood flow signals and showed no signs of abnormal axillary lymphadenopathy.

The right axillary mass was surgically excised and subjected to comprehensive pathological analysis. Macroscopic examination revealed a lesion measuring 105 × 95 × 56 mm, exhibiting a gray-white to gray-yellow appearance with a solid, soft to moderately firm texture and gray-white cross-sections. Hemorrhagic and necrotic areas were observed, all enclosed by an intact capsule. Histological analysis identified tumor cells arranged in sheet-like patterns (Fig. 1) and whirlpool-like formations (Fig. 2). These cells were predominantly oval or polygonal, with significant size variability. Some cells displayed indistinct borders, forming syncytial aggregates. The cytoplasm was abundant and eosinophilic. The centrally located nuclei were round to oval, often enlarged, with marked atypia and vacuolation. The nuclear membrane appeared thin, and the nucleolus, though small, was prominent. Numerous mitotic figures were noted. The stroma exhibited varying degrees of infiltration by mature lymphocytes, plasma cells, and eosinophils. FDDCS is frequently associated with Castleman disease. HV-CD pathology is characterized by multiple germinal center structures, with small lymphocytes in the mantle zone displaying "onion skin" changes, and small vessels penetrating vertically into the atrophic germinal center, forming a "lollipop" shape. These features were absent in the current case, leading to the exclusion of HV-CD.

The immunohistochemical profile demonstrated the following expression patterns: Vimentin (+), CD21 (+++)

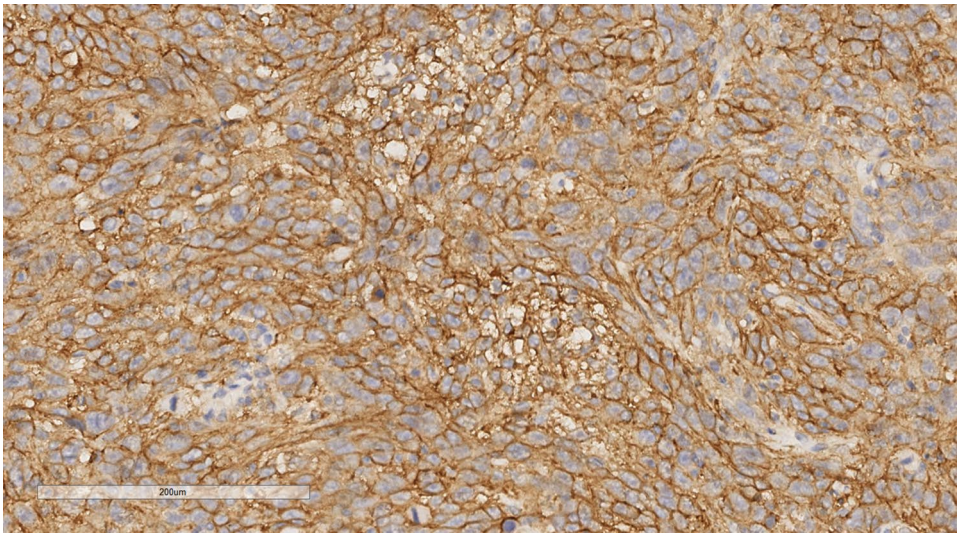

Fig. 3 CD21 is Highly expressed in tumor cells (EnViSion×200)

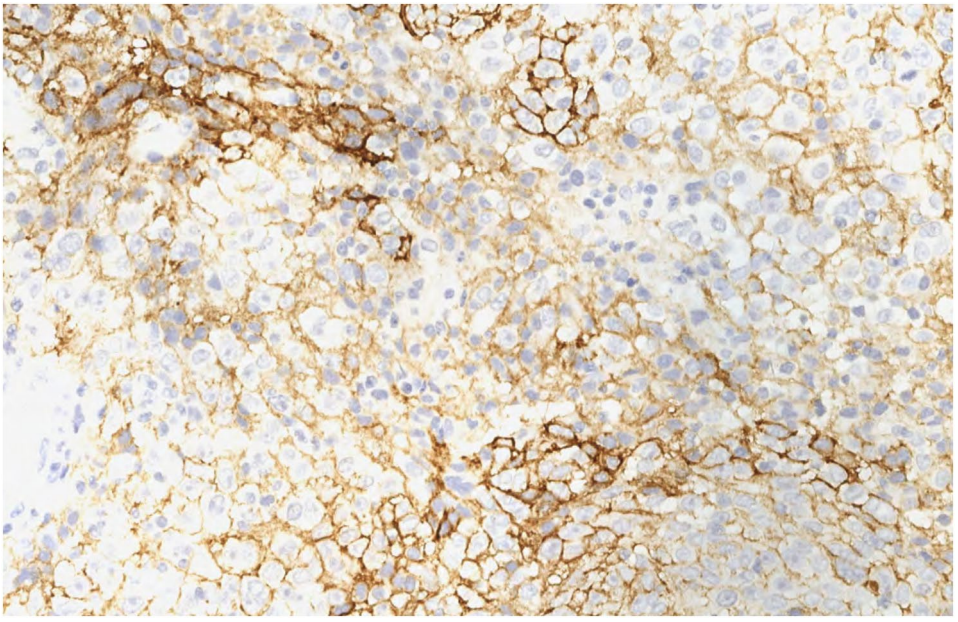

Fig. 4 Focal CD35 expression on tumor cells (EnViSion×200)

(Fig. 3), CD23 (focal +), CD123 (focal +), Catenin (membrane +), PGP9.5 (+), CK (-), CD34 (-), CK19 (-), Desmin (-), EMA (focal -), Melan-A (-), S-100 (-), SMA (-), SOX-10 (-), STAT6 (-), CD35 (focal +) (Fig. 4), Ki-67 (70%, +), CXCL13 (+) (Fig. 5), and D2-40 (+).

The next-generation sequencing (NGS) analysis (Figs. 6, 7 and 8), utilizing a targeted region capture technique to detect mutations across 1166 genes, revealed three gene rearrangements:

(1) *HFM1::BIRC3*(Fig. 6), comprising exon 5 of *HFM1* and exon 5 of *BIRC3*, with seven mutation reads identified in the sample (total read count: 32,868,708).
(2) *ELF4::AIFM1*(Fig. 7), involving exon 2 of *ELF4* and an intergenic region, displaying 13 mutation reads in the sample (total read count: 32,868,708).
(3) *DIP2B::WIF1*(Fig. 8), encompassing exon 1 of *DIP2B* and exon 3 of *WIF1*, similarly presenting 13 mutation reads (total read count: 32,868,708).

The lesion in the right axilla was diagnosed as FDCS based on the patient's clinical history, morphological features, immunohistochemical profile, and NGS data. The

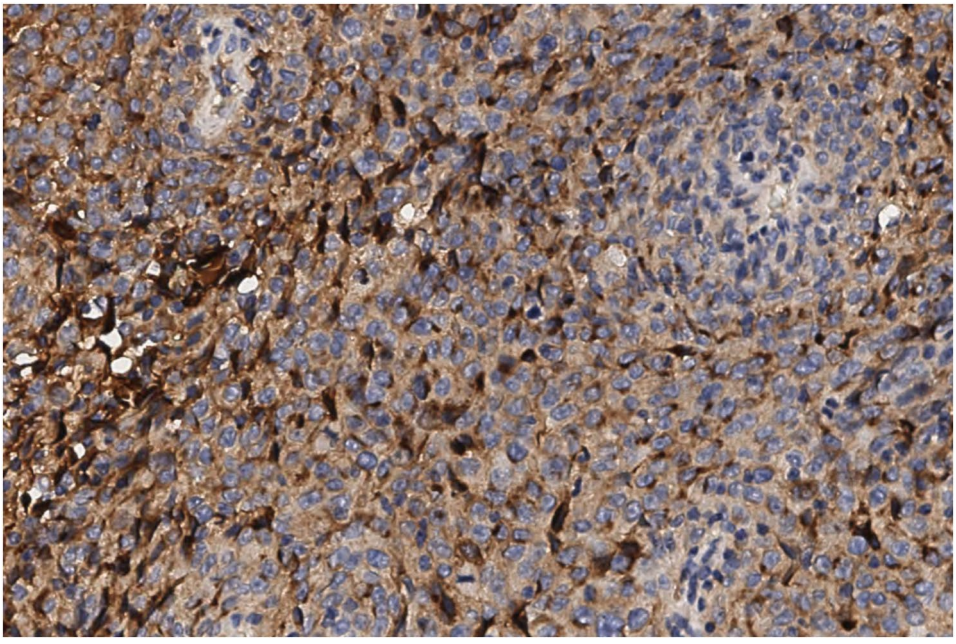

Fig. 5 Positive CXCL13 expression of tumor cells (EnViSion×200)

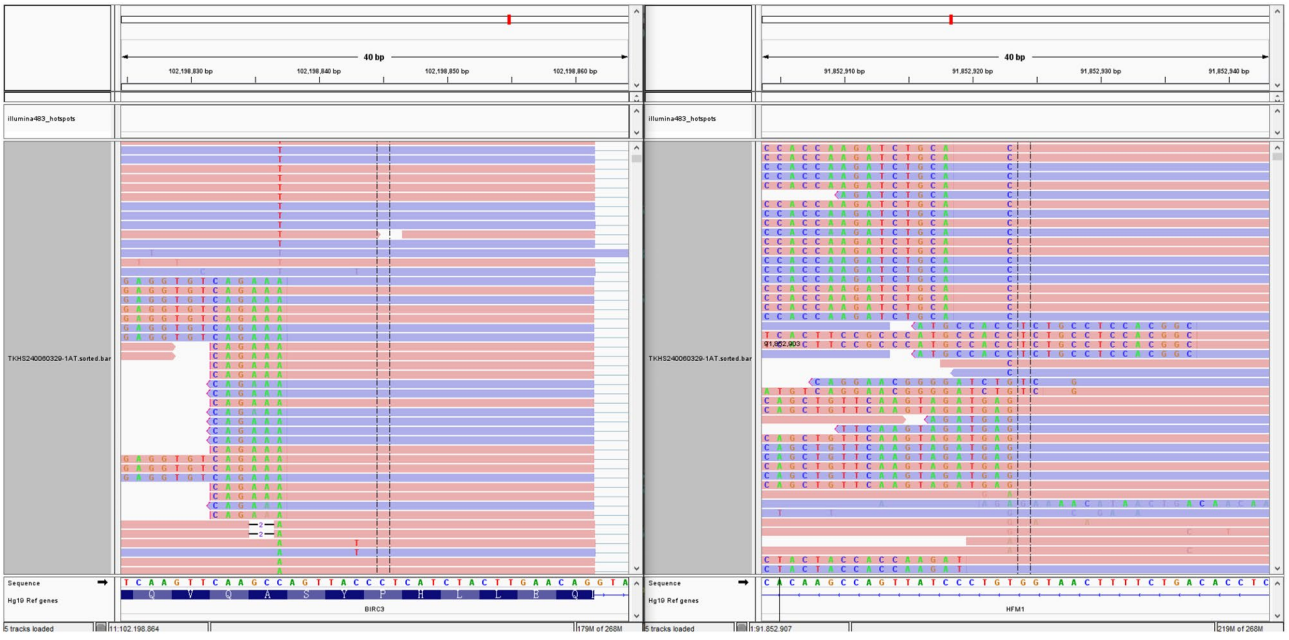

Fig. 6 Next-generation sequencing map (HFM1::BIRC3)

high mitotic activity and elevated Ki-67 index confirmed its classification as a high-grade tumor.

Follow-up at 21 months post-surgery revealed recurrence with lung metastasis. Systemic chemotherapy with ifosfamide and epirubicin was subsequently initiated.

Case Two: A 67-year-old female patient presented with left upper abdominal pain. Magnetic resonance imaging (MRI) revealed a cystic-solid mass located in the pancreatic tail, with indistinct borders extending into the adjacent gastric wall and intestines. The patient subsequently underwent open distal pancreatectomy, total splenectomy, and gastric repair, followed by pathological examination of tissue samples from the pancreas and the masses. Macroscopic analysis of the pancreas revealed dimensions of approximately 55 × 25 × 20 mm, with a grayish-white and grayish-yellow appearance and a moderately firm texture. Two masses were identified: the smaller mass, measuring 55 × 43 × 40 mm, was partially

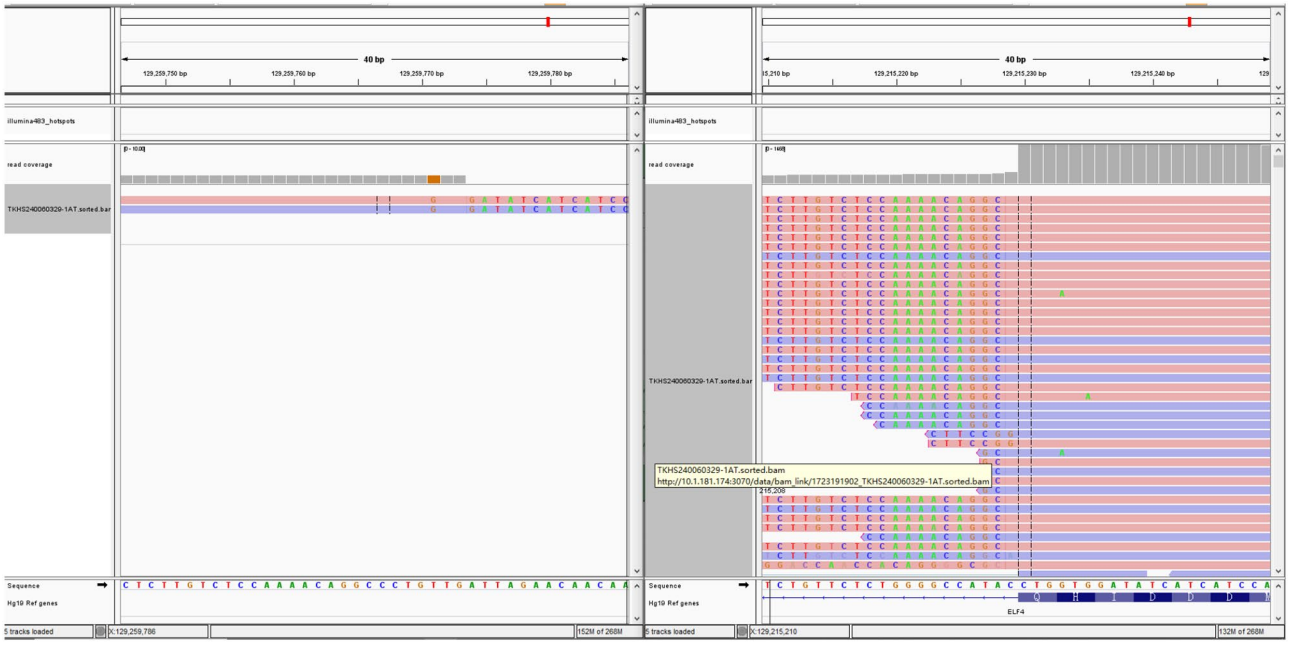

Fig. 7 Next-generation sequencing map(ELF4::AIFM1)

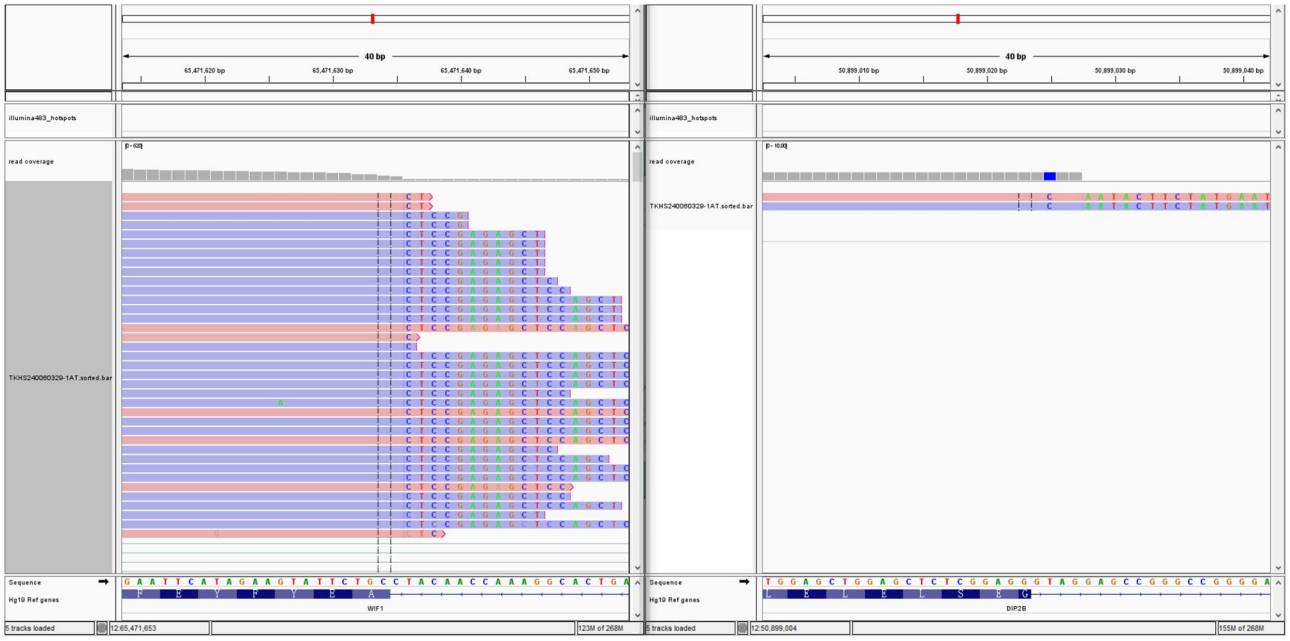

Fig. 8 Next-generation sequencing map(DIP2B::WIF1)

encapsulated, exhibiting a soft, solid, grayish-yellow consistency; the larger mass, measuring 95 × 78 × 70 mm, contained necrotic areas resembling fish flesh, with a similar grayish-white and grayish-yellow appearance and an intact capsule. Microscopic examination revealed tumor cells arranged in interwoven bundles and cords (Fig. 9). These cells were fusiform or oval in shape, with considerable size variation. The cytoplasm was abundant, lightly stained, eosinophilic, while the chromatin was fine, evenly distributed, and stippled. The nuclei were round or oval, often vacuolated, with thin nuclear membranes, prominent or multiple nucleoli, or a complete absence of nucleoli. Mitotic figures were evident. The stromal region exhibited varying degrees of infiltration by mature lymphocytes, plasma cells, and eosinophils.

Immunohistochemical analysis revealed the following tumor cell marker profile: CK (-), CD21 (+) (Fig. 10), CD23 (-), CD35 (+) (Fig. 11), CD45 (weakly +), CD20

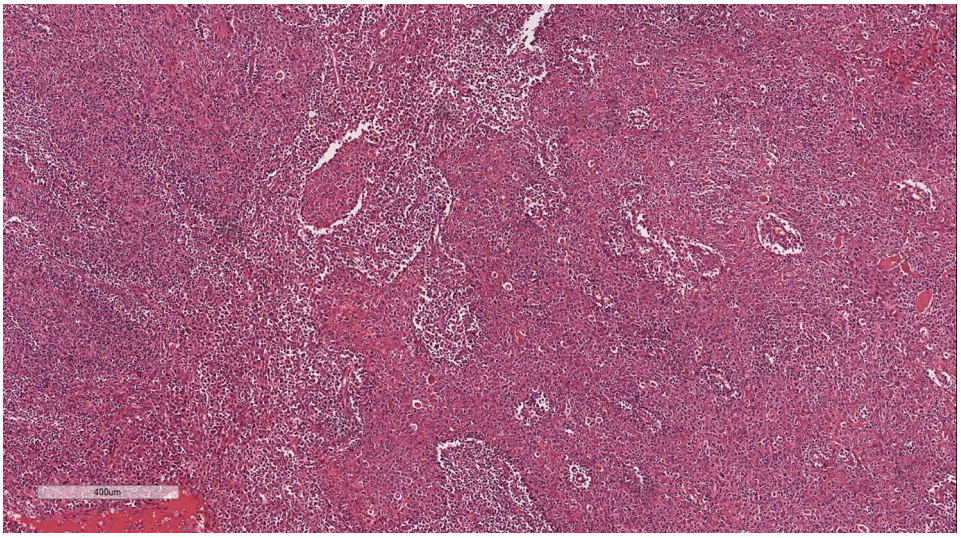

Fig. 9 Tumor cells arranged in interwoven bundles and cords (HE×100)

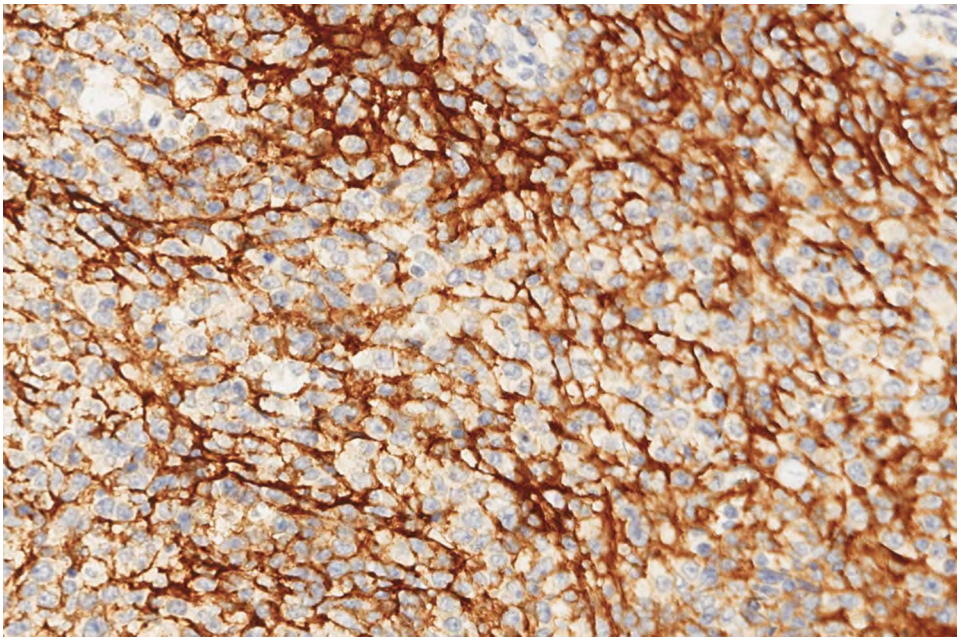

Fig. 10 Positive expression of CD21 on tumor cells (EnViSion×200)

(-), CD3 (-), CD5 (-), CD34 (partially +), CD43 (partially weakly +), CD117 (-), MPO (weakly +), E-cadherin (-), CD10 (-), CD15 (-), CD163 (-), CD235a (-), CD61 (-), CD138 (-), ALK (-), CD68 (-), Langerin (-), S-100 (-), SMA (-), Ki-67 (~ 50%+), MPO (-), CXCL13 (+), D2-40 (+) (Fig. 12), and EBER/ISH (-).

NGS analysis employing second-generation sequencing with targeted mutation capture across 1166 genes, revealed no genetic rearrangements applicable to the adjuvant sarcoma classification or aligned with current diagnostic guidelines and consensus.

The pathological diagnosis was established by integrating clinical history, morphological assessment, immunohistochemical findings, and NGS results, confirming FDCS localized to the pancreatic body and tail, with involvement of the spleen. Notably, no infiltration was observed in the splenic tissue, pancreatic parenchyma, or adjacent pancreatic resection margins.

However, recurrence and extensive metastases were identified 20 months following surgical resection.

Discussion

Clinical characteristics

FDCS occurs in both males and females across a wide age range (13–80 years) [4], with peak incidence observed between 41 and 55 years. It primarily originates in

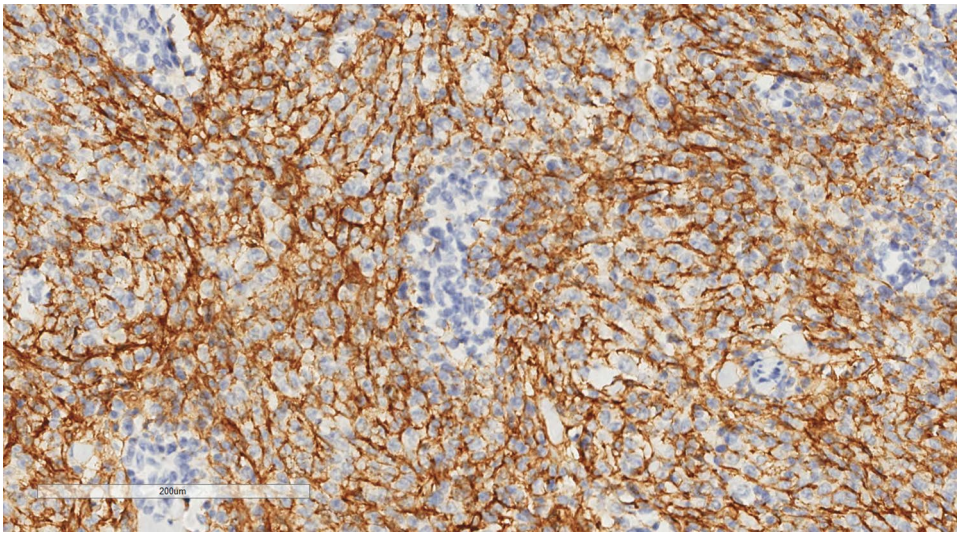

Fig. 11 Positive expression of CD35 on tumor cells (EnViSion×200)

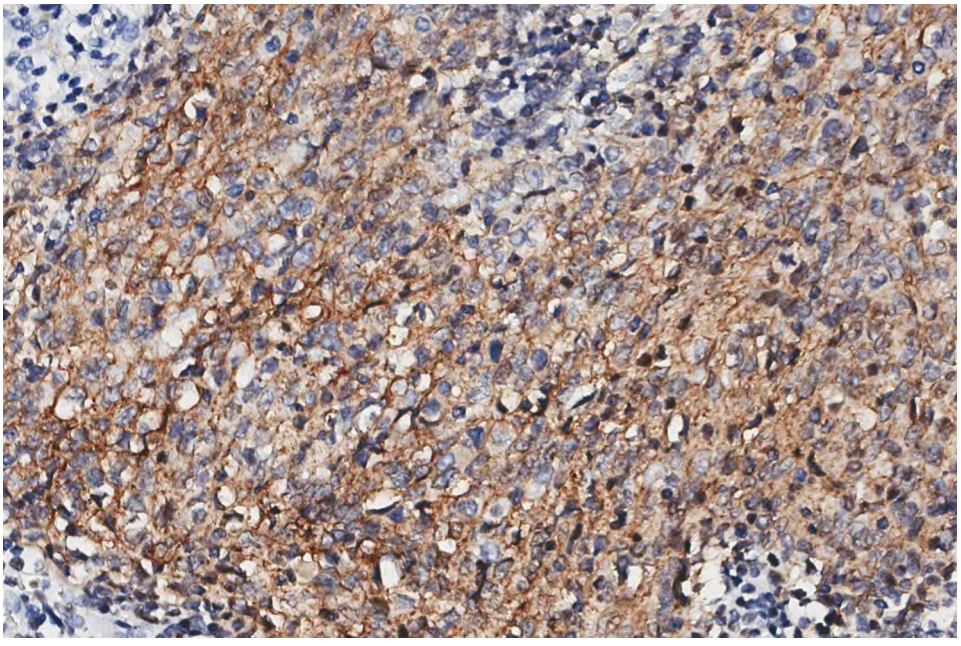

Fig. 12 Positive expression of D2-40 (EnViSion×200)

extranodal lymph nodes (79.4%) and intralymphatic regions (15.1%) [5]. Clinically, it most commonly presents as painless, progressively enlarging regional lymphadenopathy. While cervical lymph nodes are the predominant site of involvement [6], other regions, including the supraclavicular, axillary, mesenteric, and retroperitoneal lymph nodes, may also be affected. Systemic dissemination remains uncommon [7]. Extranodal FDCS has been reported in diverse locations, such as the head and neck, tonsils, nasopharynx, retroperitoneum, abdominal wall, spleen, parotid gland, palate, lung, pleura, digestive tract, breast, muscle, and testis [5]. Rare occurrences have been documented in the mediastinum, thyroid, liver, pancreas, orbit, thigh, and paravertebral tissues [8].

Notably, FDCS, generally asymptomatic, can present with gastrointestinal symptoms such as abdominal pain, bloating, or palpable masses when abdominal organs are involved. Early-stage lesions are often overlooked due to the absence of distinct clinical manifestations, with diagnosis typically relying on imaging or physical examination that identifies space-occupying lesions [9, 10]. The cases analyzed in this study display similar clinical characteristics.

Tumor sizes demonstrate significant variability, ranging from 1 to 20 cm, with an average diameter of

approximately 5 cm. Furthermore, 10–20% of cases are linked to Castleman disease, most commonly the hyaline vascular subtype [7].

The 2001 WHO classification defined this rare neoplasm based on dendritic cell morphology and immunophenotypic characteristics [11]. In the 2008 WHO Classification of Neoplasms of the Hematopoietic and Lymphoid Tissues, it was included under the broader designation of "FDCS." The 2022 WHO revision further differentiated inflammatory pseudotumor-like follicular dendritic cell sarcoma (IPT-like FDCS) from classical FDCS, highlighting its correlation with an inflammatory pseudotumor phenotype and Epstein-Barr virus-driven clonal proliferation [12, 13]. IPT-like FDCS primarily involves the liver and spleen, with occasional presentations in the gastrointestinal tract, mesentery, and tonsils [14]. The latest fifth edition of the WHO classification recognizes FDCS as a distinct pathological entity [15]. Although listed among hematopoietic and lymphoid neoplasms, its extranodal manifestations and biological features exhibit greater similarity to soft tissue tumors, warranting further investigation. FDCS typically follows an indolent course, which initially supported its classification as a low-grade malignancy. However, case series report local recurrence rates of 40-50% and metastasis in approximately 28% of cases, indicating a moderate malignant potential [16–18].

Pathological characteristics

Gross findings

The tumor exhibits encapsulation within a well-defined capsule and a solid gray-brown cut surface. Lesions affecting the tonsils, pharynx, larynx, or skin commonly present as small, polypoid, or expansile masses with sharply demarcated borders. In the abdominal cavity, the tumor often manifests as a larger mass, sarcomatous in appearance, frequently accompanied by hemorrhage and necrosis, with potential invasion into adjacent solid organs or surrounding soft tissues.

Microscopic findings

Lymph node lesions demonstrate varying extents of structural alteration, characterized by neoplastic cells arranged in bundles, radial spoking patterns, or whirlpool-like formations. The tumor cells, predominantly fusiform, oval, or polygonal, are organized into bundles, sheets, or storiform patterns, with localized whirlpool-like regions. In specific areas, indistinct cell boundaries suggest syncytial features. Nuclei exhibit enlargement with vacuolated or finely dispersed chromatin, and nucleoli, when present, are small, distinct, and typically number two or more, though they may occasionally be absent. Rare intranuclear pseudoinclusions and sporadic multinucleated giant tumor cells are observed. Mitotic activity is low, ranging from 1 to 10 figures per 10 high-power fields. The stroma frequently contains infiltrating mature lymphocytes, plasma cells, and eosinophils in varying proportions.

Electron microscopy revealed abundant elongated cytoplasmic processes and desmosomal junctions, with no detectable Birbeck bodies and minimal lysosomal presence.

Immunohistochemistry

FDCS tumor cells exhibit an immunophenotypic profile consistent with normal FDCs, characterized by the expression of FDC-associated antigens CD21, CD23, and CD35, with at least one antigen consistently detected. This pattern aligns with current literature, emphasizing the diagnostic value of combining these three antibodies to improve detection accuracy and minimize misdiagnosis [19]. Additional markers, including D2-40, CXCL-13, clusterin, and Ki-M4p, have been proposed for FDCS, although their specificity is variable [20–22]. Tumor cells frequently express vimentin, EMA, fascin, EGFR, HLA-DR, and clusterin, though none exhibit high specificity [23, 24]. S-100, CD18, CD68, and CD45 are also occasionally expressed. Of note, clusterin demonstrates strong and diffuse expression in FDCS, aiding differentiation from interdigitating dendritic cell tumors and Langerhans cell histiocytosis. In cases lacking CD21, CD23, and CD35 expression, clusterin remains a dependable marker [25]. Tumor cells consistently lack expression of myogenic markers (SMA, Desmin, and Myogenin), MPO, CD30, CD31, CD34, CD1α, CK, and HMB-45 [24]. Furthermore, clonal rearrangements of immunoglobulin and T-cell receptor genes are absent [21, 26].

Molecular genetics

Davila et al. [27] reported a case of thyroid FDCS involving mutations in *PTEN*, *RET*, and *TP53*, indicating potential molecular therapy targets. Go et al. [28] identified a *BRAF (V600E)* mutation rate of 18.5% in FDC sarcoma. Massoth et al. [29] analyzed 44 FDCS cases and identified *CDKN2A* as the most frequently mutated gene, followed by *TP53*, *BIRC3*, *NFKBIA*, *TRAF3*, *SOCS3*, *TNFAIP3*, *CCND2*, and *PTEN*. Additionally, gene fusions such as *TYK2::ATPAF2*, *MAP3K1::GCOM1*, and *NTRK1::PDIA3* were identified in individual FDCS cases.

Davila JI et al. [30] described *BPTF::WDR72* resulting in truncation of the *BPTF* functional domain critical for chromatin remodeling, while *HDGFRP3::SHC4* was associated with overexpression of the oncogene *SHC4*. The Src inhibitor dasatinib has demonstrated efficacy in soft tissue sarcoma and anaplastic thyroid carcinoma and may represent a viable therapeutic strategy for this tumor type.

Lorenzi et al. [31] reported in their FDCS cohort that mutations in common tumor suppressor genes, including *CDKN2A* deletion, as well as frequent alterations in *RB1*, *BRCA2*, *WRN*, and *TP53*, were associated with the accumulation of inactivating mutations in these genes. Such mutations correlate with multifocal disease and poor prognosis.

The study utilized RNA hybridization capture combined with NGS sequencing, targeting 1166 genes, and achieved ≥ 12 million reads at the RNA level. Bioinformatics analysis, alongside knowledge databases, facilitated the identification and interpretation of various rearrangements, including both known and novel fusion variants. This approach is particularly effective for classifying and supporting the diagnosis of Ewing sarcoma, synovial sarcoma, and other solid tumors. Previous research has highlighted the accessibility and advantages of this methodology [32–35].

This study identified three gene rearrangements: *HFM1::BIRC3*, *ELF4::AIFM1*, and *DIP2B::WIF1*. *HFM1::BIRC3* encodes an inhibitor of apoptosis protein (IAP) capable of suppressing cell apoptosis by interacting with *TRAF1* and *TRAF2*, key mediators in tumor necrosis factor receptor signaling, thereby potentially inhibiting ICE-like protease activation. *BIRC3* has been implicated in multiple hematologic malignancies, including lymphoma, mucosa-associated lymphoid tissue lymphoma, and marginal zone B-cell lymphoma. Furthermore, mutations in *BIRC3* have been documented in FDCS, as reported by Andersen EF [36] and Zheng Y [37].

The *ELF4::AIFM1* and *DIP2B::WIF1*, not previously described, may constitute novel molecular targets for FDCS, providing potential pathways for therapeutic innovation and improved prognostic strategies.

The *ELF4* gene encodes a transcriptional activator that regulates the promoters of *CSF2*, *IL3*, *IL8*, and *PRF1*, playing a role in natural killer cell development, innate immune responses, and inducing cell cycle arrest in naive CD8 + T cells. Evidence suggests that *ELF4* functions as a tumor suppressor [38].

WIF1, which encodes a protein that inhibits Wnt signaling—a pathway integral to embryonic development [39]—is recognized as a tumor suppressor often silenced through epigenetic mechanisms in various cancers, including colorectal cancer and squamous cell papilloma [40, 41]. This gene is implicated in key pathways such as GPCR and Wnt signaling.

The gene fusion discussed in this study has not been validated through alternative methods, highlighting inherent limitations in the conclusions presented.

Differential diagnosis

The diagnosis of FDCS requires pathological confirmation and is often hindered by a high rate of misdiagnosis, particularly in cases with extranodal involvement. Misdiagnosis rates have been reported between 30% and 58% [42–44]. The differential diagnosis of FDCS includes the following entities:

(1) Interdigitating dendritic cell sarcoma: Diagnostic challenges arise due to overlapping fusiform and pleomorphic morphologies. Immunohistochemistry plays a vital role in distinguishing these entities. Interdigitating dendritic cell sarcoma strongly expresses S100, Vimentin, and lysozyme, while FDCS partially expresses S100 and lacks markers such as CD21, CD23, CD35, CD1a, and Langerin [45, 46].

(2) Langerhans cell sarcoma: This tumor is characterized by classic Langerhans cell morphology, including marked atypia, abundant cytoplasm, and kidney- or horseshoe-shaped nuclei with coffee bean-like nuclear grooves. Birbeck granules can be identified by electron microscopy. Immunohistochemical markers include CD1α, S100, and Langerin, with negative results for CD21, CD23, and CD35.

(3) Malignant melanoma: Primarily occurring in the skin, its tumor cell morphology may resemble FDCS; however, the presence of brown cytoplasmic pigment aids in differentiation. Immunohistochemistry typically reveals positivity for HMB-45, Melan-A, and S-100.

(4) Inflammatory myofibroblastic tumor (IMT): Differentiation of IMT primarily aims to distinguish it from IPT-like FDCS in the liver and spleen. IMTs predominantly occur in children and adolescents, with myofibroblasts as the main cellular component. Immunohistochemical analysis typically demonstrates SMA and Desmin expression, with ALK positivity observed in some cases, while CD21, CD23, and CD35 are consistently absent. Conversely, IPT-like FDCS primarily affects middle-aged adults, characterized by immunoreactivity for CD21, CD23, and CD35 and an absence of SMA and Desmin expression [47]. (5) Differentiation of FDCS within the abdominal cavity, including the mesentery, stomach, and colon, often involves distinguishing it from gastrointestinal stromal tumors (GISTs). GISTs are predominantly composed of fusiform cells arranged in variable patterns, occasionally forming whorled or storiform structures in localized regions. The stroma contains sparse small lymphocytes. Immunohistochemical analysis reveals CD34 and CD117 positivity, with no staining for CD21, CD23, or CD35 [48]. The differential diagnosis further includes malignant fibrous histiocytoma, histiocytic

sarcoma, ectopic meningioma, malignant peripheral nerve sheath tumor, Langerhans cell histiocytosis, metastatic lymphoepithelial carcinoma, fusiform cell tumor, squamous cell carcinoma, undifferentiated carcinoma, sarcomatoid carcinoma, ectopic thymoma, and inflammatory pseudotumor, among others [49, 50]. When morphological evaluation is inconclusive, immunohistochemistry serves as a decisive diagnostic tool, as none of these tumors exhibit CD21, CD23, or CD35 expression.

Treatment and prognosis

The tumor generally displays indolent biological behavior, with recurrence occurring frequently, while metastasis to regional lymph nodes or distant sites remains rare. Prognostic factors associated with poor outcomes include tumor size exceeding 6 cm, abdominal cavity involvement, pronounced cellular atypia, high proliferation index, and extensive coagulative necrosis within the tumor [51]. Some studies, however, indicate that these prognostic factors are more relevant to abdominal cavity tumors exhibiting marked nuclear pleomorphism rather than being universally applicable [52]. The therapeutic efficacy of chemotherapy and radiotherapy in FDCS remains inconclusive [53]. For localized tumors, complete surgical excision is the treatment of choice, supplemented by routine postoperative surveillance. The benefit of adjuvant radiotherapy or chemotherapy after surgery remains controversial. In instances where complete resection is not achievable or recurrence occurs, adjuvant therapies may be considered [54].

Conclusion

FDCS, a rare tumor with diverse histological presentations and an absence of specific clinical markers, poses significant diagnostic and therapeutic challenges, often resulting in misdiagnosis or delayed detection due to limited clinical familiarity. This study examines the clinicopathological features, molecular genetics, differential diagnoses, treatment modalities, and prognosis of two FDCS cases to enhance diagnostic precision and advance understanding among clinicians and pathologists. Additionally, NGS is employed to identify potential molecular targets for developing future therapeutic strategies.

Abbreviations

FDCS	Follicular dendritic cell sarcoma
FDCs	Follicular dendritic cells
NGS	Next-generation sequencing
IPT-like FDCS	Pseudotumor-like follicular dendritic cell sarcoma

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13000-025-01614-5 .

Supplementary Material 1

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Author contributions

Writing–original draft: YC J and H Y, Writing & editing: YC J, JJ W. H Y and S L prepared all figures.All the authors have read & approved the final manuscript.

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None.

Data availability

All the data regarding the findings are available within the manuscript.

Declarations

Ethics approval and consent to participate

Ethics approval was granted by the Ethics Committee of the Affiliated Hospital of Zunyi Medical University. Written informed consent for the publication of this clinical case report was obtained from the patients and their families.

Consent for publication

Written informed consent was obtained from the patients and their families for the publication of this case report, including any associated images.

Competing interests

The authors declare no competing interests.

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