

BRIEF REPORT

LDB1::KMT2A Fusion in a Spindle-Cell Sarcoma: A Case Report

Zhigan Wang¹ | Ying Zhang^{2,3} | Mingxing Zhu^{2,3} | Jing Zhou¹ | Jingjing Feng^{2,3} | Dongbing Li^{2,3} | Rongjun Mao⁴ | Sheng Xiao^{2,3} 

¹Department of Pathology, The Affiliated Changsha Central Hospital, Hengyang Medical School, University of South China, Changsha, Hunan, China | ²Advanced Molecular Pathology Institute of Soochow University and SANO, Suzhou, Jiangsu, China | ³Suzhou SANO Precision Medicine Ltd., SANO Medical Laboratories, Suzhou, Jiangsu, China | ⁴Department of Pathology, The Eighth Clinical Medical College of Guangzhou University of Chinese Medicine, Foshan Hospital of Traditional Chinese Medicine, Foshan, Guangdong, China

Correspondence: Rongjun Mao (304089107@qq.com) | Sheng Xiao (sxiao2025@126.com)

Received: 16 July 2025 | **Revised:** 2 September 2025 | **Accepted:** 5 September 2025

Funding: This study was supported by Foshan 14th Five-Year Medical Key Specialty Construction Project (FSZD145010).

Keywords: BCOR mutation | epigenetic dysregulation | *KMT2A* fusion sarcoma | *LDB1::KMT2A* fusion | molecular pathology | spindle-cell sarcoma

ABSTRACT

KMT2A-rearranged sarcomas represent a heterogeneous group of tumors with clinical behaviors ranging from surgical cure to local recurrence and metastasis. Previously reported fusion partners include *YAPI* and *VIM*: *YAPI::KMT2A::YAPI* is associated with sclerosing epithelioid fibrosarcoma (SEF)-like histology, whereas *VIM::KMT2A* tumors exhibit a small round-to-spindle cell morphology. A third fusion, *CBX6::KMT2A::PYGO1*, was reported with a spindle-cell morphology somewhat different from the *YAPI::KMT2A::YAPI* pattern. Here, we describe a novel *LDB1::KMT2A* fusion in a spindle-cell sarcoma. The case involves a 19-year-old male who presented with an 8 cm mass situated in the left erector spinae muscle. Histopathological examination revealed a biphasic pattern comprising hypercellular fascicular/matted regions and hypocellular fibroma-like areas. Immunohistochemistry revealed diffuse positivity for CD99, SATB2, cyclin D1, BCL2, TLE1, pan-TRK, and NKX2.2, with focal BCOR expression and a Ki-67 proliferation index of approximately 10%. The tumor was negative for MUC4, SS18-SSX, WT1, cytokeratin (CKpan), vimentin, CD34, S-100, SOX10, SMA, STAT6, desmin, and MyoD1. Comprehensive genomic profiling via next-generation sequencing (NGS) identified a novel *LDB1::KMT2A* fusion, involving exons 1–10 of *LDB1* and exons 4–36 of *KMT2A*. The rearrangement was verified using fluorescence in situ hybridization (FISH) and reverse transcription PCR (RT-PCR) techniques. Additionally, a pathogenic BCOR frameshift mutation (c.3203dup, p.E1069Gfs*10) was identified. The patient underwent wide surgical excision and remains disease-free at a 5-month follow-up. This report presents the first known case of an *LDB1::KMT2A* fusion in a spindle-cell sarcoma, expanding the molecular spectrum of the emerging entity of *KMT2A*-rearranged sarcomas.

1 | Introduction

The *KMT2A* gene (or *MLL*, for mixed lineage leukemia) encodes a histone lysine methyltransferase that plays a crucial role in transcriptional regulation during development and

hematopoiesis [1]. *KMT2A* is among the genes that are most commonly rearranged in leukemia and is frequently linked to a poor prognosis [2, 3]. The *KMT2A* fusion proteins interact with menin, a nuclear scaffold protein, to regulate the expression of *HOX* genes, which are crucial drivers of

Zhigan Wang and Ying Zhang contributed equally to this work.

leukemogenesis [4]. The menin inhibitor revumenib, which blocks this interaction, has been developed due to recent advances and has received FDA approval for treating *KMT2A*-rearranged leukemia [5].

Although *KMT2A* rearrangements are well characterized in hematologic malignancies, they also occur in solid tumors, albeit rarely [1]. For example, the *YAP1::KMT2A::YAP1* rearrangement, resulting from a *KMT2A* insertion, has been identified in tumors with a morphology resembling sclerosing epithelioid fibrosarcoma (SEF) [6]. These tumors typically express cyclin D1 but lack MUC4 expression, and their clinical behavior is variable, ranging from cure after surgical resection to local recurrence and metastasis. Another fusion, *VIM::KMT2A*, resulting from a classic translocation, has been reported in five tumors of small round to spindle-cell morphology, with three of them metastatic. A recent unique case of a *CBX6::KMT2A::PYGO1* fusion, likely resulting from a three-way translocation, is described in a spindle-cell tumor, which was cured by surgical resection [7].

Interestingly, the functional domains retained in *KMT2A* fusion proteins differ between hematologic and solid tumors, suggesting distinct oncogenic mechanisms. In leukemia, the N-terminal region of *KMT2A* is preserved, whereas in sarcomas, the central or C-terminal region is retained. In hematologic malignancies, the N-terminal domains of *KMT2A* target the fusion protein to *HOX* gene clusters, and the fusion partner contributes transactivation domains. In contrast, the *YAP1::KMT2A::YAP1* and *CBX6::KMT2A::PYGO1* fusions retain only the CXXC domain, which binds unmethylated CpG islands in gene promoters and likely contributes to tumorigenesis via aberrant transcriptional activation [6, 7]. The *VIM::KMT2A* fusion retains almost the entire *KMT2A* protein, with the breakpoint at exon 2.

In this report, we describe a novel *LDB1::KMT2A* fusion in a spindle-cell sarcoma. As observed in other *KMT2A*-rearranged solid tumors, this fusion lacks the N-terminal region of *KMT2A*. We also outline the tumor's distinctive clinicopathologic, immunohistochemical, and molecular features, and propose a potential oncogenic mechanism.

2 | Case Presentation

2.1 | Clinical Findings

The patient, a 19-year-old male, had a 2–3 cm protruding mass on his left back that was painless. The mass has a firm texture, clear boundaries, and limited mobility. Ultrasound revealed a 7.9 × 5.3 × 3.2 cm solid, hypoechoic mass within the paraspinal musculature, suggesting the possibility of an intramuscular myxoma (Figure 1A). Computed tomography (CT) scan revealed an 8.1 × 4.6 × 3.6 cm oval-shaped, well-defined, low-density lesion with a density of 33 Hounsfield Units (HU) within the left erector spinae muscle, suggestive of a benign lesion (Figure 1B). The patient subsequently underwent wide local excision.

2.2 | Histopathology and Immunohistochemical Study

The dimensions of the resected specimen were 8.0 cm × 5.5 cm × 4.0 cm, and it had a smooth, encapsulated surface that ranged in color from gray-white to gray-red. The cut surface exhibited a gray-white to gray-red hue, was solid and firm in texture, had a slightly woven and lobulated appearance, and the capsule had a thickness of about 0.1–0.3 cm.

As shown in Figure 1C–H, low-power microscopy reveals a tumor with distinct borders surrounded by a thick fibrous capsule. The tumor tissue is divided into dark blue and light eosinophilic staining areas. The blue areas display numerous tumor cells arranged in interlacing bundles, mat-like configurations, parallel wavy patterns, or palisading arrangements. The cells are spindle to oval, with mild to moderate atypia, fine pale chromatin, and small or inconspicuous nucleoli, similar to monophasic fibrous synovial sarcoma. The mitotic rate is about 5 mitoses per 10 high-power fields. Some areas show an onion-skin-like arrangement around small vessels, and occasionally a radial distribution around thick-walled vessels, resembling perivascular epithelioid cell tumor (PEComa) or pericytic tumor. Hemorrhagic foci form pseudovascular spaces. In the lightly stained areas, tumor cells are sparsely distributed, fibroblast-like, with pale chromatin and inconspicuous nucleoli, interspersed with fine, parallel wavy collagen fibers, and varying degrees of lymphocytic infiltration. The stroma contains abundant thin-walled dilated small vessels resembling hemangiopericytoma, spiral artery-like vessels similar to those in the uterus, and thick-walled vessels. No necrosis is observed.

Immunohistochemical analysis of tumor cells is diffuse positivity for CD99, SATB2, cyclin D1, TLE1, BCL2, NKX2.2, and pan-TRK, with weak to moderate positivity for BCOR (Figure 2). CD99 shows membranous staining, BCL-2 and pan-TRK show cytoplasmic staining, and the remaining markers demonstrate nuclear staining. In hotspot areas, the Ki-67 proliferation index is about 10%. The tumor cells show negativity for MUC4, SS18-SSX (E9X9V Rabbit mAb, #72364, CST), WT1, CKpan, Vimentin, CD34, S-100, SOX10, SMA, STAT6, Desmin, and MyoD1.

2.3 | Molecular Analysis

Comprehensive genomic profiling was performed due to the inability to establish a definitive diagnosis based on morphology and immunophenotyping. RNA sequencing was performed on an MGISEQ-2000 instrument (2 × 150 bp paired-end reads) using a Nanodigm NEXome Core Panel supplemented with intronic probes for fusion detection. Fusions were called with STAR-2.7.10, STAR-Fusion v1.10.1, Arriba v2.2.1, and FusionCatcher v1.33 integrated in the SANO Fusion Pipeline; only in-frame events supported by ≥ 3 types of unique-length fusion reads and > 10 total reads were retained. For DNA analysis, libraries prepared with the same NEXome Core Panel kit were sequenced on the MGISEQ-2000. Reads were aligned with BWA-MEM v0.7.17; SNVs and indels were identified with

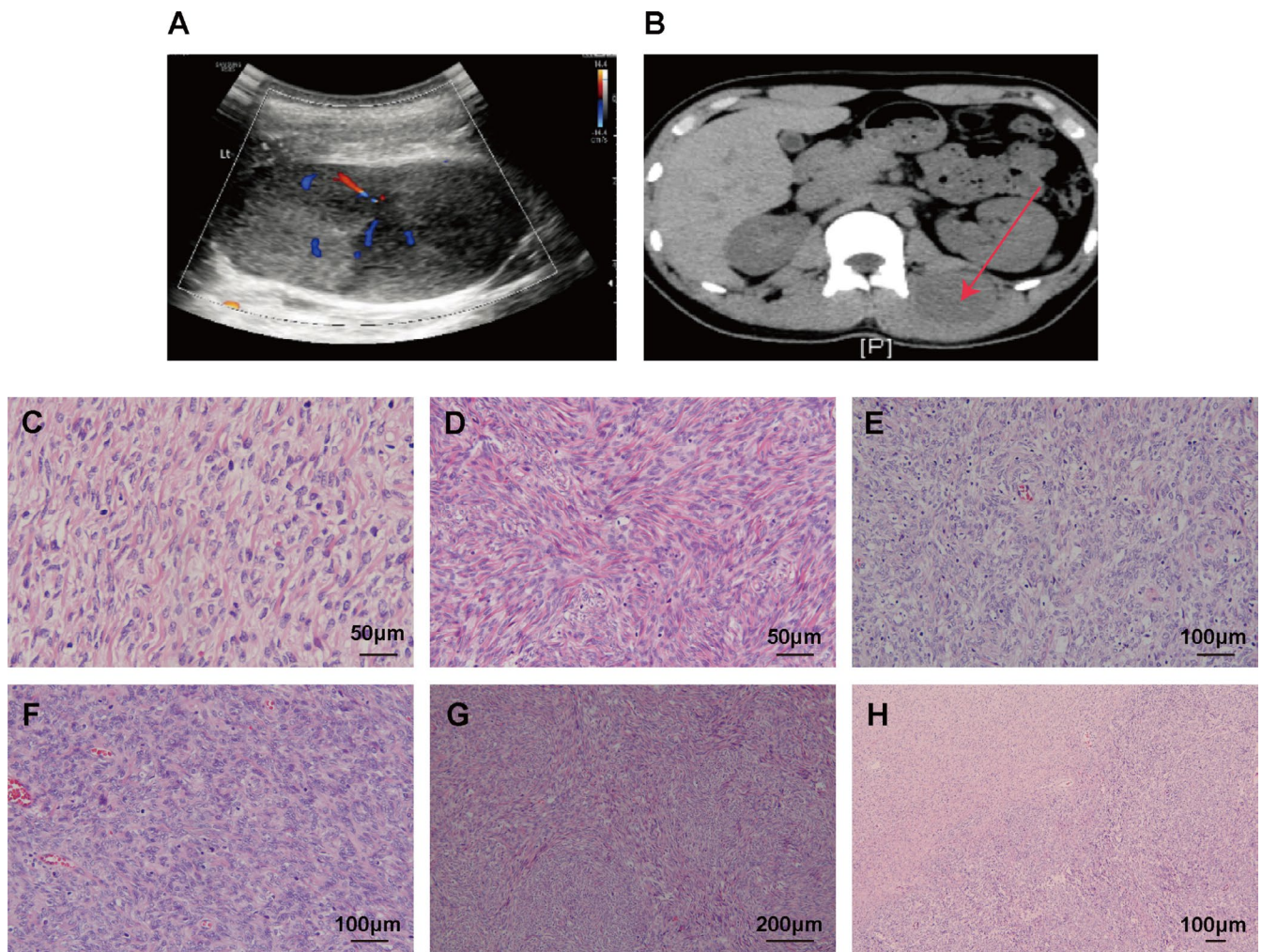


FIGURE 1 | Ultrasound, CT imaging of the patient's left back and his histological findings. (A) Ultrasound shows a well-defined, solid hypoechoic mass in the left posterior muscle layer, measuring $79 \times 53 \times 32$ mm. The mass has heterogeneous internal echotexture with patchy low echogenicity. Color Doppler flow imaging (CDFI) reveals punctate and linear blood flow signals (PS: 82.5 cm/s, ED: 27.5 cm/s, RI: 0.67). (B) Noncontrast CT shows an oval-shaped, well-defined hypodense lesion in the left erector spinae muscle, measuring $81 \times 46 \times 36$ mm, with a CT attenuation value of 33 HU. The lesion is initially considered benign. (C, D) Spindle-shaped tumor cells are arranged with variable cellularity in a stroma containing cord-like collagen bundles, mimicking a solitary fibrous tumor. (E) Focally oval-shaped tumor cells demonstrate perivascular growth. (F, G) The tumor consists of densely packed spindle cells arranged in fascicular or storiform patterns, with finely granular chromatin, bearing a resemblance to synovial sarcoma. (H) At low magnification, a well-demarcated interface is observed between superficial eosinophilic tumor regions and deeper zones; the upper left area is hypocellular with abundant hyalinized collagen bundles, while the lower right is hypercellular with sparse fibrous stroma.

Vardict v1.8.3-0, then annotated with ANNOVAR v2020Jun8. Variants were filtered for depth $\geq 5\times$, allele fraction $\geq 5\%$, and absence or frequency $<0.1\%$ in gnomAD v2.1.1. Whole transcriptome analysis identified an in-frame *LDB1::KMT2A* fusion consisting of the first 10 exons of *LDB1* and the last 33 exons of *KMT2A*, preserving the majority of functional domains of *KMT2A*, including its catalytic SET domain (Figure 3A). Additional DNA sequencing detected a pathogenic frameshift mutation in the *BCOR* gene (c.3203dup, p.E1069Gfs*10, 12.7%) as the sole alteration, predicting a truncated, nonfunctional protein (Figure 3D). No copy number variations (CNVs) across the genome or copy-neutral loss of heterozygosity (CN-LOH) were detected.

KMT2A rearrangement was confirmed by fluorescence in situ hybridization using the *KMT2A* (11q23) Break Apart FISH Probe (F.01045-01, AccuPa+h, Guangzhou, China) according

to the manufacturer's protocol. FISH using a *KMT2A* split-apart probe confirmed the *KMT2A* gene rearrangement in 70% of cells (Figure 3E). The *LDB1::KMT2A* fusion transcript was amplified by an RT-PCR assay using primers specific to *LDB1* (F_ *LDB1*: 5'-GCGGAAACGGAAGATGTCAG-3'; F_ *LDB1* nest: 5'-AAGAAGAGCCCAGCTAGCAC-3') and *KMT2A* (R_ *KMT2A*: 5'-TCATCATTCCTCCATGGAAGACA-3'; R_ *KMT2A* nest: 5'-TGGGCATGTCATCAGGAAACA-3'), which produced a PCR product at the expected size. Sanger sequencing of the PCR product confirmed *LDB1::KMT2A* (Figure 3B,C).

2.4 | Treatment and Follow-Up

The patient underwent wide excision with negative margins. No adjuvant radiotherapy or chemotherapy was administered. At

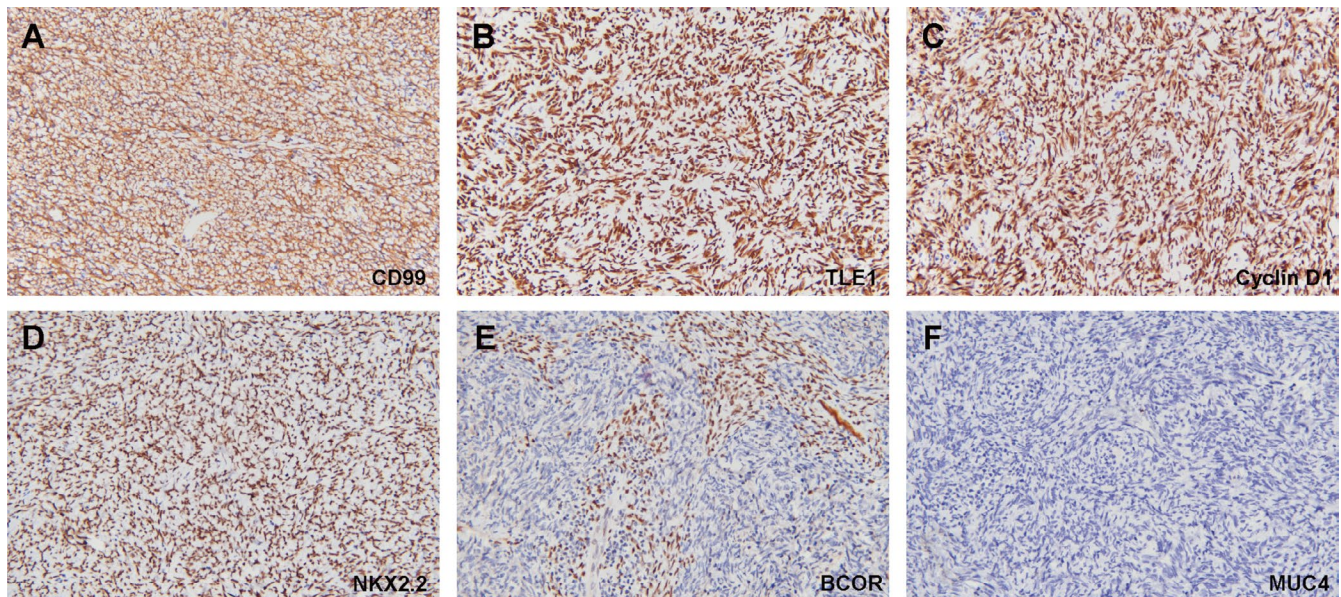


FIGURE 2 | Immunohistochemical findings. (A–D) Immunohistochemistry: CD99 (A), TLE1 (B), cyclin D1 (C), and NKX2.2 (D) are strongly positive; BCOR shows weak-to-moderate nuclear positivity (E); MUC4 is negative (F).

5 months postoperatively, the patient remains disease-free with no evidence of recurrence or metastasis.

3 | Discussion

We report a *KMT2A*-rearranged soft tissue tumor located in the paraspinal musculature of a 19-year-old male. The tumor was completely resected, and no recurrence was observed at 5 months of follow-up. The majority of *KMT2A*-rearranged sarcoma cases occur in young adults, with a median age at diagnosis of about 35 years and an equal male-to-female patient ratio [8]. These tumors predominantly arise in soft tissue, with only a single reported case involving bone [8]. Clinical outcomes are variable, ranging from complete surgical cure to local recurrence or distant metastasis. The basis for this clinical heterogeneity, despite the presence of a shared fusion driver, remains unclear. However, most of these tumors harbor secondary genomic alterations, including *CDKN2A/B* deletions, *MDM2* amplification, and others [9]. These secondary changes may contribute significantly to tumor behavior. For example, homozygous *CDKN2A/B* loss is associated with poor prognosis in multiple sarcoma types, while *MDM2* amplification can lead to TP53 degradation and increased genomic instability, both of which are negative prognostic indicators. In our case, a *BCOR* mutation was identified as the lone secondary change. Although *BCOR* is involved in chromatin remodeling and epigenetic regulation, its isolated presence may not significantly contribute to tumor aggressiveness in this patient. Of note, a *BCOR* frameshift mutation has been documented in one *VIM::KMT2A* sarcoma [6], and *BCOR* mutations are also reported in myeloid tumors, although not specifically associated with *KMT2A*-rearranged cases. Whether *BCOR* mutations represent a recurrent event in these *KMT2A*-rearranged sarcomas requires further investigation.

In hematologic malignancies, patients with *KMT2A* rearrangements typically are resistant to standard chemotherapy and have a poor prognosis [10]. *KMT2A* fusion proteins interact

with menin to initiate leukemogenesis. By specifically disrupting the binding of menin to the N-terminal AT-hook domain of *KMT2A* that is retained in leukemic fusions, menin inhibitors like revumenib have demonstrated efficacy [11]. This targeted approach has significantly improved outcomes in these otherwise lethal leukemias. However, in sarcomas, the *KMT2A* fusion involves the C-terminal portion of the gene, lacking the AT-hook domain, and thus is not expected to respond to menin inhibitors [6]. However, expression profiling of tumors with *YAP1::KMT2A::YAP1* fusion has revealed overexpression of multiple pathways, including endogenous cannabinoid signaling, hedgehog signaling, and Wnt signaling [6]. These findings suggest that targeting dysregulated signaling pathways may provide novel therapeutic approaches for these tumors. Alternatively, secondary alterations such as *CDKN2A/B* loss and *MDM2* amplification could also be targeted.

KMT2A-rearranged sarcomas had marked heterogeneity in both morphology and genomic architecture [12]. Both *YAP1::KMT2A::YAP1* and *CBX6::KMT2A::PYGO1* retain only the CXXC domain of *KMT2A*, while *VIM::KMT2A* and *LDB1::KMT2A* fusions retain most *KMT2A* functional domains, excluding the N-terminal AT-hooks [13]. This genomic variability may underlie differences in histologic appearance and immunophenotype: *YAP1::KMT2A::YAP1* shows SEF-like morphology, expressing diffuse cyclin D1, patchy EMA, S100, and BCOR, but lacking MUC4; while *VIM::KMT2A* sarcomas show round blue cell morphology with strong expression of CD99, BCL2, BCOR, NKX2.2, EMA, S100, and are also negative for MUC4. Our patient showed spindle-cell morphology with an immunophenotype overlapping that of *VIM::KMT2A* sarcomas, positive for CD99, BCL2, TLE1, BCOR, NKX2.2, EMA, and S100. Further accumulation of cases is essential to determine whether *KMT2A*-rearranged sarcomas can be subclassified based on the specific *KMT2A* functional domains retained, that is, those retaining only the CXXC domain versus those preserving most of the protein's functional domains.

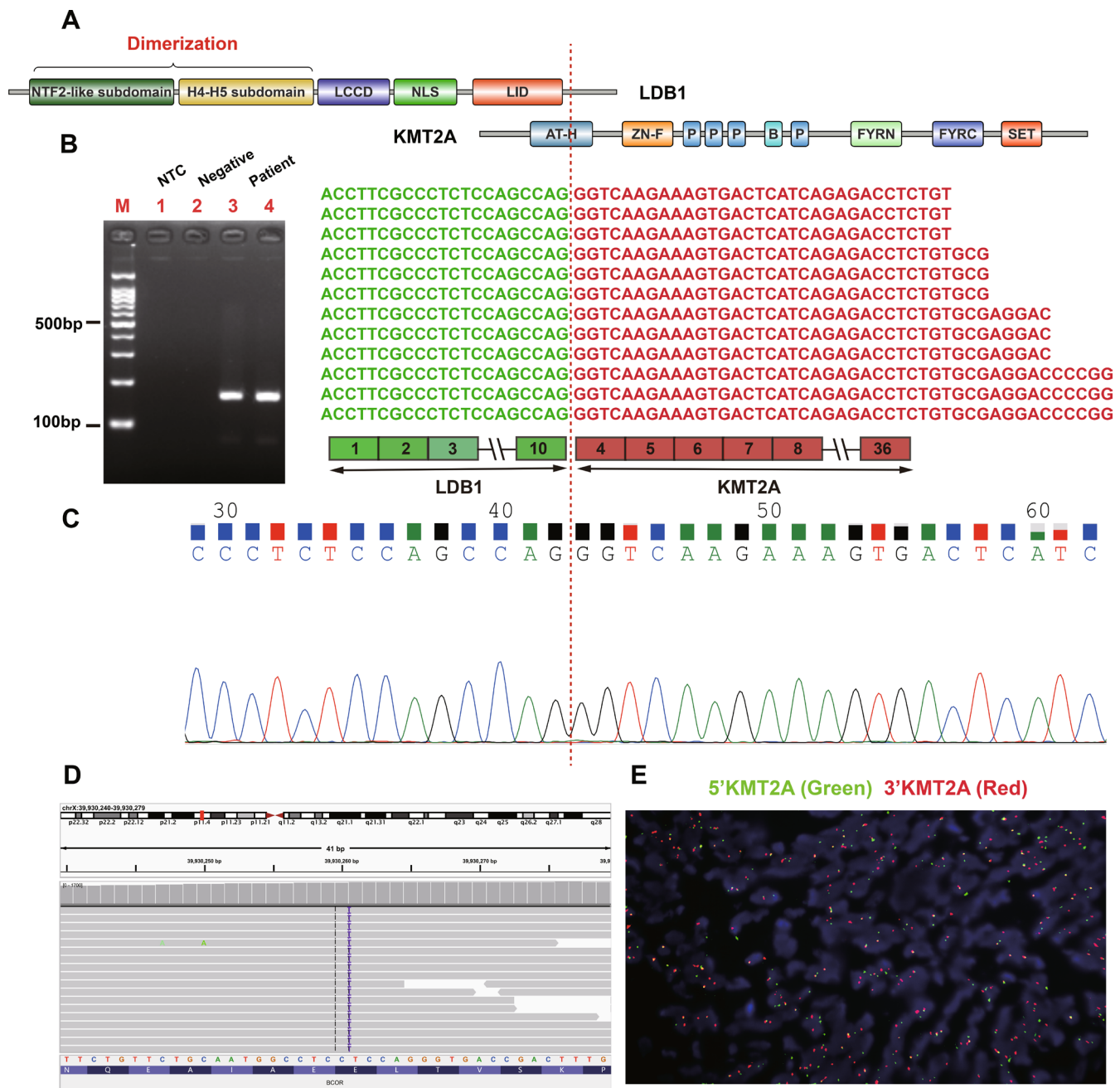


FIGURE 3 | Molecular characterization of the *LDB1::KMT2A* fusion and *BCOR* mutation. (A) RNA NGS identifies an *LDB1::KMT2A* fusion transcript, retaining key domains from both genes (indicated by vertical dotted line). LID (LIM-interaction domain, exons 9 of *LDB1*) is retained in the fusion. (B) RT-PCR using *LDB1* and *KMT2A* primers amplifies a product of the expected size. Lanes 3 and 4 are technical replicates from the same RNA specimen. (C) Sanger sequencing confirms the *LDB1::KMT2A* fusion. (D) IGV image shows a pathogenic frameshift mutation in the *BCOR* gene (c.3203dup, p.E1069Gfs*10). (E) FISH confirms *KMT2A* gene rearrangement, with separated 5' (green) and 3' (red) signals.

The oncogenic role of LDB1 in the *LDB1::KMT2A* fusion remains unclear. LDB1 is a nuclear adaptor protein that functions as a scaffold for transcriptional complexes involved in cell fate determination and oncogenesis [14]. It is part of the oncogenic LMO2/LDB1 complex in T-ALL and AML. Three critical functional domains of LDB1 are preserved in the *LDB1::KMT2A* fusion: the LIM-Interaction Domain (LID), the Dimerization Domain, and the Nuclear Localization Signal [15]. Combined with the histone methyltransferase

activity of KMT2A, these LDB1 domains may interact with different transcription machineries to activate a distinct set of tumor-promoting genes.

In summary, we report a novel *LDB1::KMT2A* fusion in a spindle-cell sarcoma, expanding the molecular spectrum of *KMT2A*-rearranged soft tissue tumors. Our findings highlight the genomic diversity, diagnostic challenges, and therapeutic implications within this emerging sarcoma category.

Author Contributions

Sheng Xiao and **Rongjun Mao**: conceptualization. **Rongjun Mao**: funding acquisition. **Zhigan Wang** and **Jing Zhou**: data curation. **Mingxing Zhu** and **Jingjing Feng**: validation. **Ying Zhang** and **Dongbing Li**: writing – original draft. All authors did writing – review and editing.

Acknowledgments

The authors have nothing to report.

Ethics Statement

The authors have nothing to report.

Consent

Informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data and materials of this study will be available from the corresponding author [Sheng Xiao] upon reasonable request.

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