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Novel *ACTB::FER* Promoter Swap Fusion Characterizes Rare Superficial Myoid/Myofibroblastic Tumors

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ABSTRACT

Pediatric fibroblastic, myofibroblastic, and myoid tumors encompass several entities, many with characteristic gene fusions that are now emerging as molecularly defined tumor groups. Here, we present two cases of spindle cell neoplasms with novel *ACTB::FER* promoter swap fusions. Both tumors presented in the extremities of pediatric patients (9-year-old and 6-year-old females) as superficial skin nodules with slow growth. Histologically, both tumors showed monomorphic spindle cell proliferation in short fascicles, but without significantly increased mitotic activity, high-grade atypia, or necrosis. Both cases showed diffuse positivity for SMA with patchy desmin expression. RNA sequencing confirmed fusion breakpoints, revealing transcriptional upregulation of *FER*. Neither patient has had evidence of interval growth or recurrence to date. While the biological significance of *ACTB::FER* fusions remains unclear, their recurrence and the absence of other clear oncogenic drivers suggest a distinct molecular pathway that may define a novel entity. Fusions of *ACTB* and *FER* genes with different partners have been observed in rare aggressive mesenchymal tumors; however, the *ACTB::FER* promoter swap fusion is currently unrecognized in soft tissue tumors. We report the first two cases of soft tissue tumors harboring *ACTB::FER* fusions and expand the molecular spectrum of mesenchymal tumors with kinase gene alterations. Further, we highlight the importance of target-agnostic approaches for the detection of rare kinase fusions, which may not be included on targeted next-generation sequencing panels.

1 | Introduction

Pediatric and infantile fibroblastic and myofibroblastic lesions are rare and encompass a wide spectrum from benign to malignant tumor types [1]. Due to their rarity, diagnosing these tumors can be challenging. The 2020 update to the World Health Organization (WHO) classification system classifies bone and soft tissue tumors according to their localization, their morphologic and histologic characteristics, clinical features, molecular profiles, staging, and prognosis [2, 3]. Fibroblastic and myofibroblastic soft tissue tumors exist on a spectrum of biological potential, ranging from benign (e.g., fibrous hamartoma of infancy, nodular fasciitis [NF]) to intermediate (e.g., desmoid fibromatosis, dermatofibrosarcoma protuberans) to malignant entities (e.g., sclerosing epithelioid fibrosarcoma, low-grade fibromyxoid sarcoma, myxofibrosarcoma, fibrosarcoma) [2, 3]. While morphological and immunophenotypic characterization is still essential for histologic diagnosis and grading, molecular profiling is becoming increasingly important in clinical practice and can provide diagnostic support and guidance for further clinical management [1]. Recurring molecular alterations in these tumors, particularly in tyrosine or serine/threonine kinases, may be amenable to targeted therapy [1]. While cost-effective, next-generation sequencing (NGS) panels may miss actionable alterations, suggesting the need for comprehensive and target-agnostic sequencing approaches.

Here, we report two cases of pediatric spindle cell tumors with novel *ACTB::FER* fusions. Case 1 was a low-grade/benign fibroblastic/myofibroblastic tumor involving the deep dermis of the right upper thigh identified in a 9-year-old patient with multiply relapsed B-acute lymphoblastic leukemia (B-ALL); Case 2 was a dermal-based, mildly nodular lesion identified on the right anterior shoulder of a previously healthy 6-year-old female. Comprehensive whole transcriptome RNA-sequencing analysis in Case 1 identified a novel promoter swap fusion between the beta-actin gene (*ACTB*, at 7p22.1) and the feline encephalitis virus related (*FER*, at 5q21.3) tyrosine kinase gene, leading to *FER* transcriptional upregulation. Similarly, the TruSight RNA Pan-Cancer sequencing panel performed in Case 2 identified an *ACTB::FER* fusion with a nearly identical structure. FER, a non-receptor tyrosine kinase, has been infrequently reported in tumors to date and, for this reason, is not included in many targeted sequencing panels. Herein, we describe the histomorphologic features of these two lesions and provide a brief literature review on FER kinase fusions in neoplasia.

2 | Materials and Methods

2.1 | Whole Transcriptome Sequencing and Analysis (Case 1)

The punch biopsy was evaluated histopathologically to assess specimen adequacy (≥40% tumor cells). Total RNA was extracted from FFPE tumor tissue using the RNeasy kit (Qiagen Inc., Germantown, MD). RNA was quantitated on a Qubit 2.0 Fluorometer by using an RNA HS assay kit (Thermo Fisher Scientific, Waltham, MA), and RNA quality was assessed via an Agilent 2200/4200 Tapestation (Agilent Technologies, Santa Clara, CA). The Illumina TruSeq Stranded Total RNA LT Kit (Illumina Inc., San Diego, CA) was used to generate libraries that were sequenced by using a paired-end 2×125 bp cycle protocol on a NovaSeq 6000 platform (Illumina Inc.). Samples were sequenced to obtain a minimum of 15% of exonic bases at 45× or greater depth of coverage. The reads were mapped to the human genome reference sequence (hg19) and fusion detection was carried out using the CICERO algorithm, as previously described [4, 5].

2.2 | TruSight RNA Pan-Cancer Sequencing Panel (Case 2)

The TruSight RNA Pan-Cancer panel (Illumina, San Diego, CA) was used to detect gene fusions, single-nucleotide variants (SNVs), and indels in the targeted 1385 genes. The punch biopsy was histopathologically assessed for tumor content (> 50% tumor cells). RNA was extracted with the RNAstorm FFPE RNA Isolation Kit (Biotium Inc., Fremont, CA). A total of 200 ng was used for library preparation according to the manufacturer's recommendation before sequencing on a NextSeq-500/550 (Illumina). The sequence was aligned to the hg19 human genomic scaffold using the Illumina STAR aligner (v2.6.1a). Analysis was performed using the Illumina BaseSpace platform (Strelka & DRAGEN), as well as the Metafusion fusion caller [6]. The fusion breakpoints and frame of the fusion were determined manually by using the Integrated Genomics Viewer from the Broad Institute (https://software.broadinstitute.org/software/igv/).

3 | Results

3.1 | Case Presentations

3.1.1 | Case 1

A 9-year-old female with a history of relapsed hyperdiploid B-ALL presented with a superficial nodule at the medial right upper thigh. Ultrasound imaging showed a small hypoechoic, somewhat ill-defined focus in the superficial soft tissue measuring approximately 0.8×0.6×0.2 cm. There was no significant surrounding edema/hyperemia or additional lesions identified. The clinical differential diagnosis included extramedullary leukemia relapse or a reactive process. A punch biopsy was performed for diagnosis. The patient is under follow-up of her leukemia and has not shown any interval growth or recurrence of her cutaneous lesion at 10 months.

Histologically, the biopsy demonstrated a dermal spindle cell proliferation arranged in haphazard fascicles within loose collagenous stroma. Focal extravasated red blood cells and some interspersed lymphocytes were present. The spindle cells were monomorphic, without significant nuclear atypia or mitotic activity. The tumor was limited to the dermis without subcutis involvement. The epidermis showed elongated rete ridges with mildly increased basal layer pigmentation. The spindled tumor cells showed diffuse strong positivity for smooth muscle actin (SMA) and patchy desmin expression (Figure 1A–E). Additional immunostains were performed, including CD34 and ALK-1, which were negative in the tumor cells. Moreover, there was no evidence of leukemia in this sample, and stains for CD20, Pax5, CD3, TdT, and CD19 were all negative.

Overall, the histological features were felt to be consistent with a low-grade/benign fibroblastic/myofibroblastic proliferation. The possibility of NF was considered in the differential diagnosis, among other low-grade/benign fibroblastic lesions and reactive processes. Previous germline testing did not reveal any pathogenic or likely pathogenic variants in this individual [7]. Given the diagnostically challenging nature of this tumor, molecular profiling using whole transcriptome RNA-sequencing analysis was pursued.

3.1.2 | Case 2

A 6-year-old female presented with a hyperpigmented, mildly nodular lesion measuring 1.0 cm in greatest dimension on her right anterior shoulder. The lesion had been present for approximately 1 year, with slight growth noted over several months before presentation. The lesion was reported to be tender when manipulated or bumped. She underwent a punch biopsy initially, followed by an excisional resection. No ultrasound or other imaging was performed during her workup.

Microscopic evaluation revealed a bland, monomorphic spindle cell proliferation confined to the dermis, organized in short

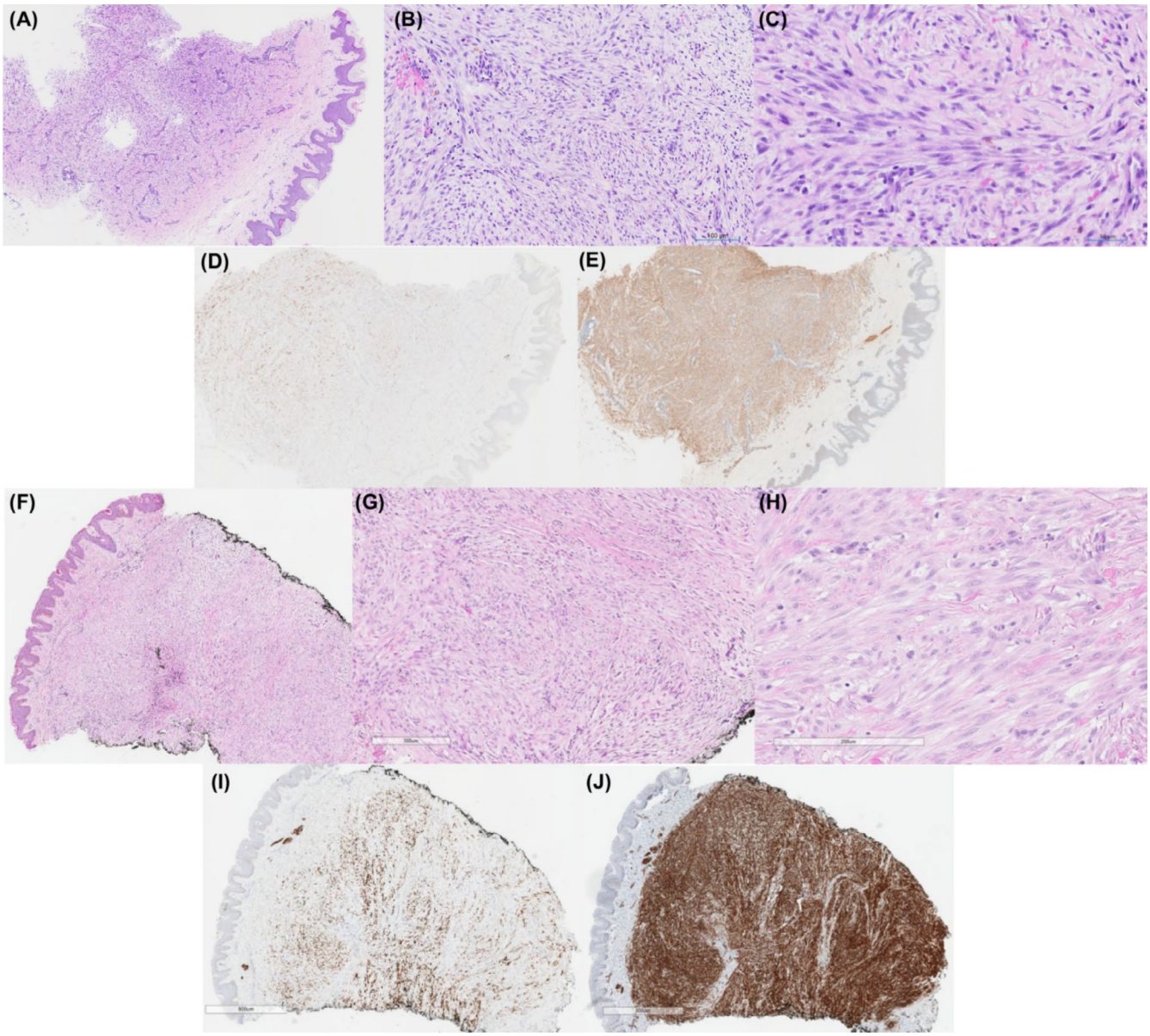

FIGURE 1 | Representative images of histopathology and immunohistochemistry (IHC) in the fibroblastic/myofibroblastic lesions. (A–C) In Case 1, H&E demonstrates the deep dermis involved by a fibroblastic/myofibroblastic proliferation with bland spindle cells admixed with inflammatory cells. Immunohistochemical analyses revealed patchy positivity of the neoplastic cells for desmin (D) and strong positivity for SMA (E). Case 2 shows similar findings with H&E (F–H) showing a bland, monomorphic spindle cell proliferation confined to the dermis. Immunohistochemistry demonstrated patchy positivity for desmin (I) and diffuse, strong positivity for SMA (J).

fascicles with focal storiform and haphazard arrangements. No necrosis was present, and the mitotic count is low (up to 1 mitosis per 2 mm^2). The proliferation did not extend into the subcutis. The overlying epidermis shows elongation of the rete ridges and increased pigmentation of the basal layer, with a clear Grenz zone separating the lesion from the epidermis. The lesion exhibited perivascular inflammation in the overlying dermis and periphery close to the papillary dermis (post-resection and not the on the punch) with only rare inflammatory cells in the lesion, and no red blood cell extravasation within the lesion. Immunohistochemistry demonstrated diffuse, strong positivity for SMA and patchy positivity for desmin (Figure 1F–J), while markers such as caldesmon, CD34, Factor XIII, S100, CD68, EMA, and Keratin AE1/AE3, were negative. The MIB1 proliferative index was less than 3%.

Overall, the histological features were consistent with a bland-appearing spindle cell neoplasm of fibroblastic/myofibroblastic origin that is not morphologically typical for a well-defined WHO entity. Therefore, molecular profiling using a TruSight RNA Pan-Cancer sequencing panel analysis was pursued. Of note, the patient has not had any evidence of recurrence in the 4 months post-resection.

3.2 | NGS Sequencing Findings

In Case 1, unstained slides from the punch biopsy were sent for whole transcriptome (RNA-seq) analysis, which detected an *ACTB::FER* in-frame promoter swap fusion [chr7:5570155(–)::chr5:108133825(+); *ACTB* (NM_001101.5) exon 1 (5′UTR)

is fused to *FER* (NM_005246.4) exon 3 (5′UTR)] likely via a t(5;7)(q21.3; p22.1) (Figure 2A). The fusion results in loss of the first two non-coding exons of *FER* and places the strong beta-actin gene (*ACTB*) promoter upstream of the coding exons of *FER*. Elevated *FER* expression was confirmed in the RNA-sequencing data (Figure 2B).

In Case 2, TruSight RNA Pan-Cancer sequencing panel analysis detected a similar *ACTB::FER* in-frame promoter swap fusion *ACTB* (NM_001101.5) exon 1 (5′UTR) is fused to *FER* (NM_005246.4) exon 3 (5′UTR) (Figure 2C). No oncogenic SNVs were detected by panel testing in this sample.

4 | Discussion

To our knowledge, the *ACTB::FER* promoter swap fusion identified in both cases has not been previously described in the literature. The *ACTB* gene (located at 7p22.1) encodes beta-actin, a ubiquitously expressed structural protein with a number of essential cytoplasmic and nuclear functions, including regulation of cell shape, migration, gene expression, and proliferation [9]. *ACTB::GLI1* promoter swap fusions have been described in a group of pericytic tumors characterized by the translocation t(7;12)(p22; q13). In this fusion, the *GLI1* oncogene is fused to *ACTB*, resulting in the upregulation of *GLI1*, which is driven by the strong beta-actin promoter. Similar fusions have also been found in a group of spindled and/or epithelioid neoplasms with variable S100 expression that exhibit both benign and malignant potential [10–13]. The *FER* gene (at 5q21.3) encodes a member of a subfamily of non-receptor protein tyrosine kinases. In a rat fibroblast model, overexpression of human FER resulted in reduced cell adhesion, suggesting it may play a role in the regulation of adhesion and migration through modulation of the adherens junctions and focal adhesions via upstream signaling through epidermal growth factor (EGF) or platelet-derived growth factor (PDGF) receptors [14, 15]. In-frame fusions involving the *FER* kinase gene include *SSBP2::FER* in T-cell acute

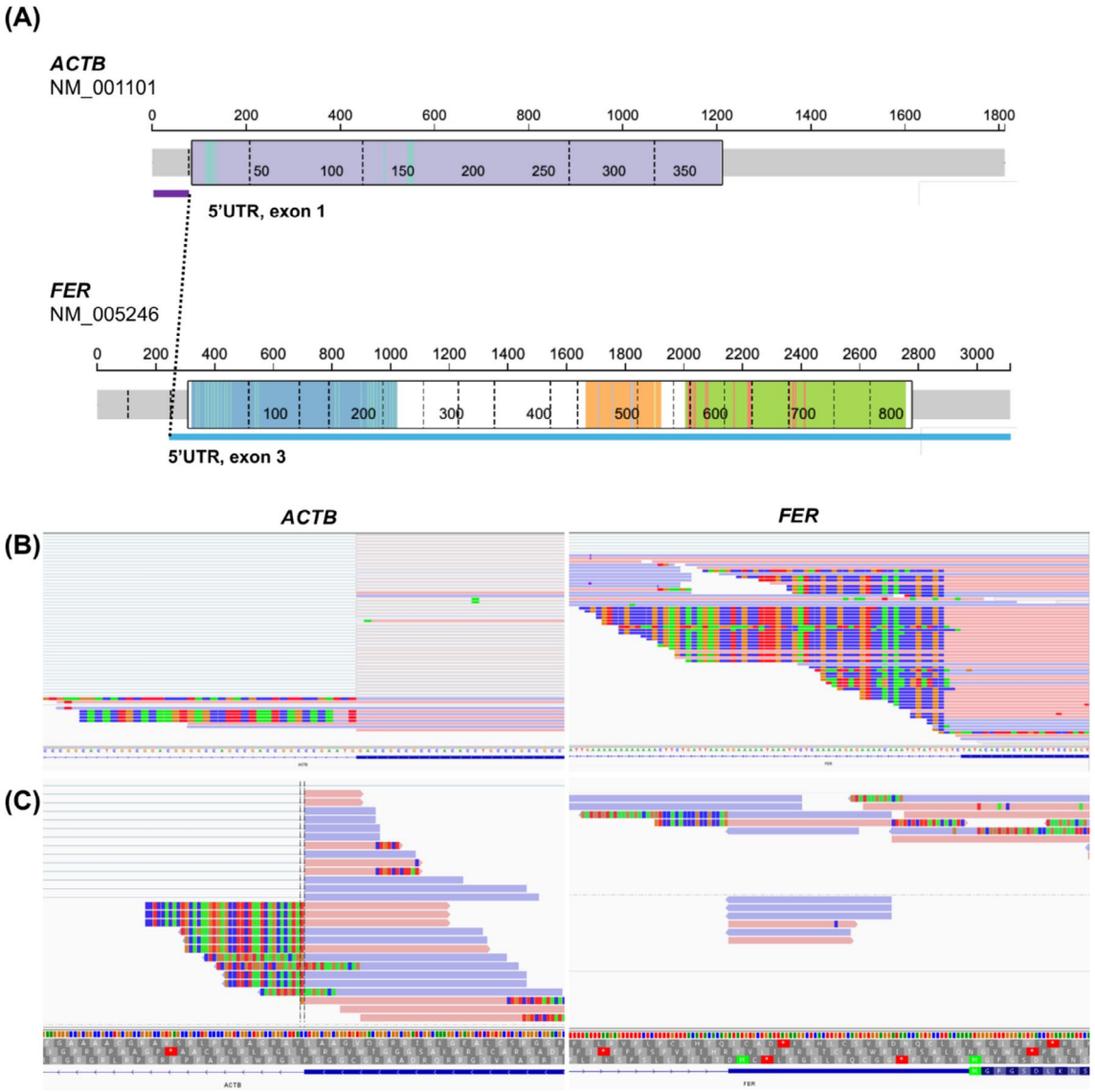

FIGURE 2 | Schematic representation of the *ACTB::FER* promoter swap fusion. (A) Schematic diagram showing the fusion between *ACTB* (NM_001101.5) promoter/5′UTR and *FER* (NM_005246.4) 5′UTR/exon 3 generated using ProteinPaint [8]. RNA-sequencing support for the fusions (shown by soft-clipped reads) detected in Case 1 (B) and Case 2 (C) visualized in IGV.

lymphoblastic leukemia [16], *MAN2A1::FER* found in hepatocellular carcinoma and other tumor types [17], *ITK::FER* detected in peripheral and follicular T-cell lymphomas [18, 19], and *MYO5A::FER* fusion described in a case of glioneuronal tumor [20].

Of note, two recent studies identified FER kinase alterations in myofibroblastic tumors, including an *ARHGAP23::FER* fusion [21] and a *SH3PXD2B::FER* fusion [22]. These chimeric fusions were predicted to result in aberrant constitutive tyrosine kinase activity of FER, primarily through fusions upstream of the tyrosine kinase domain (*ARHGAP23* [NM_001199417.2] exon 7 in-frame to *FER* [NM_005246.4] exon 11; *SH3PXD2B* [NM_001017995.3] exon 11 in-frame to *FER* exon 11 [NM_005246.4]). The overall fusion structure suggests a different mechanism of constitutive activation compared with the current cases, which are predicted to result in overexpression of the full-length FER tyrosine kinase. Although the plexiform myofibroblastic tumor with *SH3PXD2B::FER* fusion had a relatively benign general appearance and disease course, the *ARHGAP23::FER*-fused tumor was a highly infiltrative intramuscular lesion that metastasized to the lymph nodes and lungs. Interestingly, this tumor did not harbor any additional molecular alterations, suggesting that *ARHGAP23::FER* fusion was the sole oncogenic driver. This patient had a sustained and durable response to treatment with single agent lorlatinib, suggesting that *FER* kinase fusions may be amenable to targeted therapy [21].

Both lesions described here showed myoid or myofibroblastic differentiation, raising the possibility of superficial benign or low-grade tumors with this phenotype, including superficial NF [23], cellular dermatofibroma, myopericytoma/myofibroma, or the entity recently proposed under the term pediatric-type myoid neoplasm [24]. The morphology of our two cases somewhat overlaps with superficial *USP6*-rearranged NF but without the classic features (tissue culture-like appearance, microcystic spaces). Whether these *ACTB::FER*-associated lesions could represent a rare variant of NF is yet to be determined. Myopericytoma/myofibroma classically displays a distinctive biphasic growth pattern or characteristic perivascular arrangement, which was absent in our cases. Additionally, alterations in the *PDGFRB* gene appear to represent a common pathogenesis for conventional myofibroma. Pediatric-type myoid neoplasms, another diagnostic consideration, display morphologic features of conventional smooth muscle—particularly the ovoid to spindled cells with elongated, cigar-shaped nuclei and abundant eosinophilic cytoplasm that were not observed in either lesion [24]. In contrast, a subset of myoid tumors or tumors with a smooth muscle-like immunophenotype, including cellular/atypical myofibroma, have been reported to harbor *SRF* fusions, further distinguishing our cases at the molecular level [25, 26]. Whether these lesions correspond to pediatric myofibroblastic or myoid neoplasms with undescribed fusions or represent a novel entity remains unclear and requires further study of many more cases. The cytoarchitectural features also resemble those of a cellular dermatofibroma; however, we did not see significant hyaline dermal collagen entrapment or entrapment of superficial subcutaneous tissue, which are common findings in this lesion. Nevertheless, while the biological significance of *ACTB::FER* fusions remains unclear, identifying two cases with similar morphological, phenotypical, and molecular characteristics, without other clear driver alterations, suggests a distinct molecular pathway that may define a novel entity or a new variant. Neither patient has had evidence of interval growth or recurrence to date, though follow-up is limited (with Case 1 at 10 months post-biopsy and Case 2 at just 4 months post-resection).

The tumors described in this report are unlike the two previously reported *FER*-rearranged myofibroblastic neoplasms. Both of our cases lacked the plexiform architecture described by Vallese et al. [22], and did not show the epithelioid, ganglion-like, and multinucleated tumor cells with nuclear atypia reported by Sadaf et al. [21] In addition, the fusion structure in our cases is unique and places the strong beta-actin gene (*ACTB*) promoter upstream of the coding exons of *FER* tyrosine kinase, leading to elevated *FER* expression. While this upregulation suggests a potential role in tumorigenesis, its precise oncogenic function remains to be determined. Further studies are needed to elucidate the role of FER kinase activation in soft tissue tumorigenesis, including functional analyses and clinical follow-up to assess long-term outcomes. Additionally, the inclusion of *FER* in targeted sequencing panels or gene target-agnostic approaches may reveal similar fusions in morphologically ambiguous soft tissue neoplasms. Given reports of *FER* fusion-positive tumors responding to kinase inhibitors, identifying similar cases may have important therapeutic implications, particularly for patients with advanced or recurrent disease. The development of a standardized diagnostic approach, incorporating histomorphology, immunohistochemical markers, and molecular testing algorithms, could aid in the recognition and appropriate classification of these and similar lesions.

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Ethics Statement

This study is approved by the St. Jude Children's Research Hospital Institutional Review Board (IRB Number: 23-1515; approved October 16, 2023), which determined that this project did not meet the criteria for human subjects research and, therefore, did not require full institutional review board approval. This project was also approved by the Research Ethics Board of The Hospital for Sick Children.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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