Diagnostic Pathology

CASE REPORT

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An extralingual Ectomesenchymal chondromyxoid tumor with *RREB1::MRTFB* fusion: a rare case report of plantar fascia involvement

Yu Deng[1], Wei Liu[2] and Ke Sun[1*]

Abstract

Background Ectomesenchymal chondromyxoid tumor (ECT) is a rare benign intraoral tumor that almost exclusively presents as a small mass on the anterior dorsal tongue. Recently, the ras-responsive element-binding protein 1::myocardin-related transcription factor B *(RREB1::MRTFB*; previously known as *MKL2*) fusion gene has been identified in 90% of ECTs, all localized to the tongue, highlighting its genetic distinctiveness. Herein, we report a mesenchymal tumor involving the plantar fascia of the left foot in a young woman, harboring the *RREB1::MRTFB* fusion gene.

Case presentation The tumor presented as a well-circumscribed mass. Following complete excision, no recurrence was observed at the six-month follow-up. Histological examination revealed tumor cells exhibiting mild nuclear atypia and very low mitotic activity. Immunohistochemical analysis showed diffuse positive staining for S100, glial fibrillary acidic protein (GFAP), and CD56, variable expression of smooth muscle actin, and negative staining for SOX10 and P63. Targeted RNA sequencing identified *RREB1* (exon 8)–*MRTFB* (exon 11) fusion transcripts. Collectively, these findings suggest the possibility of a previously unreported extralingual ECT involving the plantar fascia. However, its atypical morphology and uncommon anatomical location posed significant diagnostic challenges.

Conclusions We report, for the first time, a mesenchymal chondromyxoid tumor with an *RREB1::MRTFB* fusion gene occurring in the foot. This case expands the known distribution of ECT beyond the tongue. Accurate differential diagnosis should rely on thorough histological assessment, combined with immunohistochemical and molecular analyses.

Keywords Extra-glossal Ectomesenchymal chondromyxoid tumor, ECT, *RREB1*, *MRTFB*, *MKL2*

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Background

Ectomesenchymal chondromyxoid tumor (ECT) is an extremely rare neoplasm almost exclusively occurring in the tongue, first described by Smith et al. in 1995 [1]. A recent study reported that the average patient age is 40 years, ranging from 2.3 to 78 years, with no significant sex predilection [2]. Most ECTs are localized to the anterior dorsal surface of the tongue [2, 3]. However, rare cases involving the lateral tongue, gingiva [4], hard palate [5, 6], tonsillar bed [7], and the right mandibular body [8] have also been documented. These tumors are typically slow-growing, painless, well-circumscribed, firm submucosal nodules without ulceration. Their sizes generally range from 0.3 to 4.0 cm in diameter, with rare exceptions exceeding 4.0 cm. Microscopically, ECTs are composed of small, round-to-fusiform cells arranged in sheets within a myxoid or hyalinized stroma. Hemorrhage and cyst formation are occasionally observed.

The term "ectomesenchymal chondromyxoid tumor," introduced in the initial publication, reflects the presumed origin of the tumor from migrated ectomesenchymal cells of the neural crest. This hypothesis is supported by the observation that the anterior tongue derives from neural crest mesenchyme of the first branchial arch, explaining the tumor's predilection for this location. The neural crest origin theory is further corroborated by immunohistochemical markers such as GFAP, S-100, and CD56, which are commonly expressed in neural tissue. Recently, Dickson et al. [9] identified a novel *RREB1::MRTFB* fusion gene in 90% of 21 ECTs, all of which were located in the tongue, underscoring the genetic distinctiveness of this neoplasm.

Herein, we report a mesenchymal tumor involving the plantar fascia of the left foot harboring the *RREB1::MRTFB* fusion gene. Although initially unclassifiable, histological reevaluation and additional immunohistochemical studies, informed by genetic insights, revealed phenotypic similarities to ECTs of the tongue, suggesting that this case represents an extralingual counterpart of ECT.

Case presentation

A 20-year-old woman presented with swelling and pain in the left plantar region persisting for 4 years. Outpatient examination revealed a left plantar mass measuring approximately 44 mm × 20 mm × 20 mm. She was admitted to the hospital with a diagnosis of "left lower limb tumor." The patient had no relevant personal or family medical history.

Magnetic resonance imaging (MRI) of the left foot revealed a block-shaped abnormal signal in the space between the left plantar flexor muscle tendon. The lesion demonstrated uneven, slightly low-intensity T1 signals and uneven high-intensity T2 signals, with well-defined borders and a size of approximately 44 mm × 20 mm × 20 mm (Fig. 1). Compression of adjacent flexor spaces was observed, but no significant bone destruction was detected in the calcaneus, talus, sphenoid bone, cuneiform bone, or metatarsal bones. The tarsal and metatarsophalangeal joints were free of stenosis, with smooth joint surfaces. No fluid accumulation was noted in the joints, or abnormal signals were observed in the surrounding soft tissues or tendons.

MRI findings suggested a left plantar mass with benign characteristics, potentially indicating focal bleeding or degeneration. A clinical diagnosis of a benign lesion was made, and local excision was performed. Histopathological examination confirmed tumor-free surgical margins, and the postoperative course was uneventful. The patient was referred to a local physician for follow-up care.

Pathological findings

The surgically resected specimen consisted of an elastic, soft tumor measuring 4.5 cm × 2.3 cm × 1.2 cm. The cut surface revealed a cystic and solid appearance, with bloody fluid in the cystic region and a grayish-white solid region exhibiting a focal mucinous texture. Histological examination showed that the tumor was well-circumscribed, with a thin fibrous capsule covering most areas and localized infiltration into the surrounding skeletal muscle tissue. The tumor predominantly comprised uniform, short spindle to ovoid cells with hyaline or eosinophilic cytoplasm. At high magnification, certain regions displayed cells secreting mucus, giving rise to a microcystic morphology. There was no evident nuclear atypia in the myxoid stroma. Mitotic figures were absent, and no necrosis was observed. The stroma was rich in blood-filled sinusoids, resembling vascular-related lesions, with some areas demonstrating collagenization suggestive of an osteoid matrix (Fig. 2).

Immunohistochemistry revealed diffuse positive staining for S100, GFAP, and CD56, with focal weak positivity for smooth muscle actin (SMA). The Ki-67 labeling index was 10% in hotspot areas. The tumor cells were negative for pan-cytokeratin, CD34, ERG, HMB45, Melan-A, P63, SOX10, and neuron-specific enolase (NSE) (Fig. 3). Notably, TFE3 showed nuclear weak positivity; however, fluorescence in situ hybridization (FISH) analysis for TFE3 did not detect signal separation.

Molecular analysis using targeted RNA-based next-generation sequencing (NGS) identified an in-frame fusion between exon 8 of *RREB1* (NM_001003698.4) and exon 11 of *MRTFB* (NM_014048.4), a genetic event previously reported as highly recurrent in ECTs [10] (Fig. 4).

Based on the histopathological, immunohistochemical, and molecular findings, a diagnosis of ECT was established. At the 3-month postoperative follow-up, the patient remained recurrence-free.

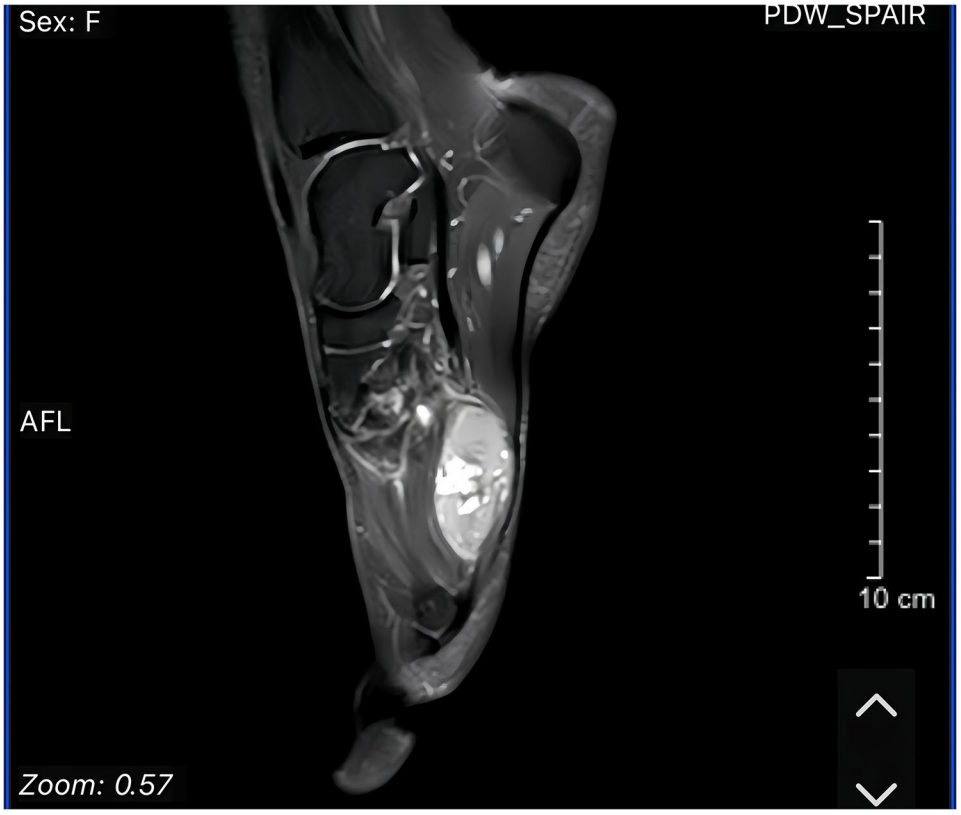

Fig. 1 Sagittal T2-weighted magnetic resonance imaging showing a well-defined, heterogeneous hyperintense lesion on the left foot

Discussion and conclusions

ECT is a rare intraoral mesenchymal tumor. In this report, we describe, for the first time, an extremely rare case of ECT arising from the plantar fascia of the left foot in a young woman, a location where it can be easily misdiagnosed in clinical practice.

Unlike the painless presentation commonly reported in the literature [2], this patient experienced swelling and pain, likely attributable to the tumor's location. Gross examination of the tumor revealed a firm-to-myxoid or gelatinous cut surface with variable coloration, including yellow and white. Cystic areas with or without hemorrhage, as described previously [11], were also observed in this case.

Morphologically, ECT typically presents as a well-defined, non-encapsulated mass composed of uniform round, ovoid, or fusiform cells embedded in a myxoid or chondromyxoid matrix, with no mitotic figures, nuclear pleomorphism, or vascular invasion [12]. In contrast, the present case showed a thin fibrous capsule and focal infiltration into the surrounding skeletal muscle. Sakurai et al. [13] previously reported involvement of striated muscle bundles and peripheral nerve fibers within ECTs, raising the question of whether these features suggest an invasive biological behavior that warrants further investigation.

Initially, we considered hemangioblastoma; however, this diagnosis was ruled out due to negative staining for α-inhibin and NSE. Microcystic/reticular schwannoma was also considered due to S100 positivity, but SOX10 negativity contradicted this diagnosis. Karamchandani et al. [14] noted that S100 and SOX10 exhibit similar sensitivity in neural crest-derived tumors, excluding malignant peripheral nerve sheath tumor. Other differential diagnoses, including monophasic spindle cell synovial sarcoma, solitary fibrous tumor, and myoepithelioma, were excluded due to negative staining for CD99, TLE1, CD34, and P63.

Non-ossifying fibromyxoid tumor, a subtype of ossifying fibromyxoid tumor (OFMT), was also considered because of its histological similarity to ECT, S100 positivity in 67% of cases, and lack of ossification. However, this diagnosis was ultimately excluded due to the absence of INI1 loss and the lack of TFE3 rearrangement on FISH analysis. Notably, OFMT is associated with recurrent gene rearrangements, predominantly involving the *PHF1* gene [15, 16].

Given these challenges, we explored mesenchymal tumors with *GLI1* alterations, but FISH analysis showed no changes in *GLI1*. NGS identified the presence of an ras-responsive element-binding protein 1::myocardin-related transcription factor B *(RREB1::MRTFB*;

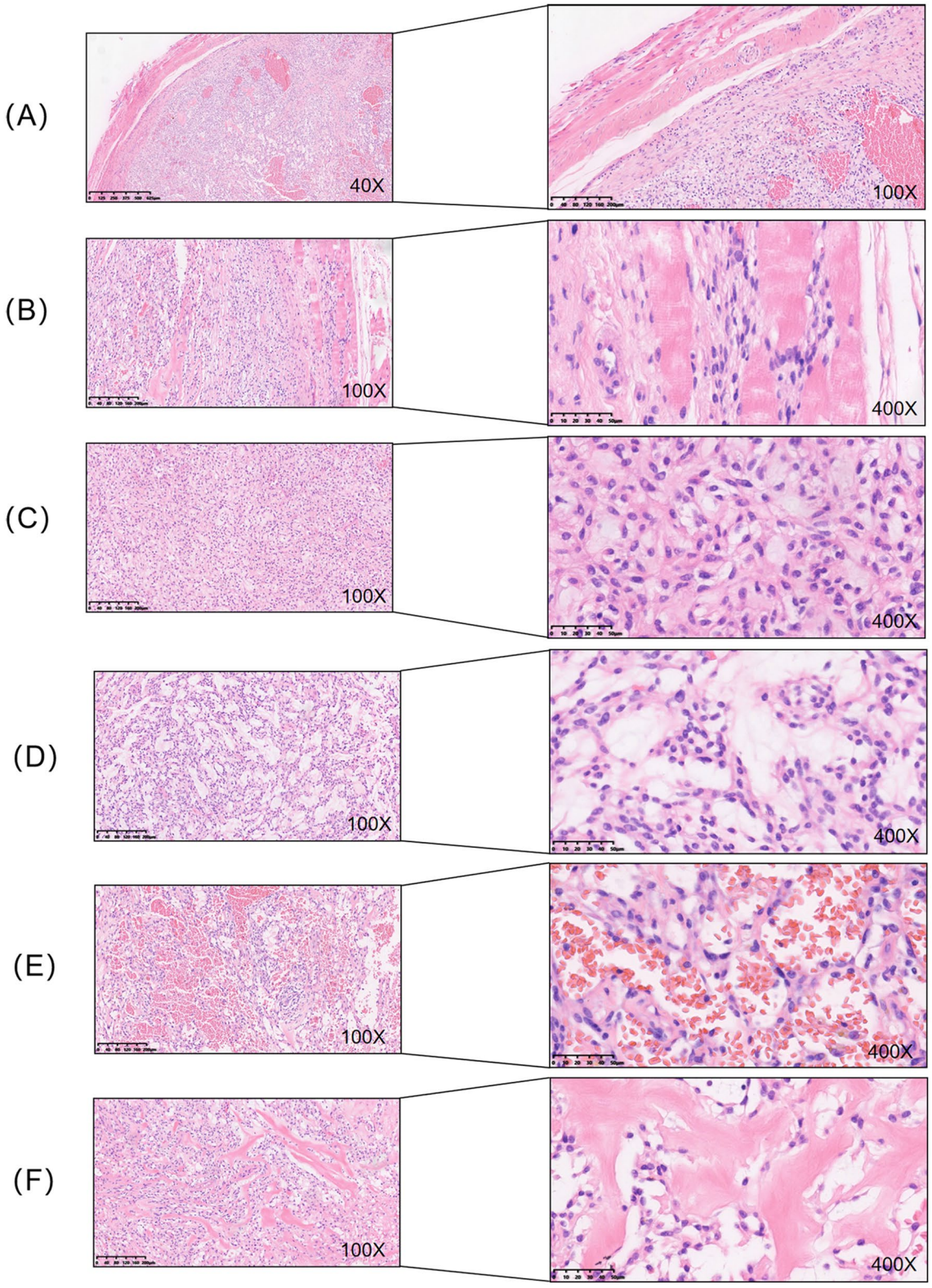

Fig. 2 Histopathological findings of the surgical specimen. (**A**) The tumor was well circumscribed, with a thin fibrous capsule encasing most areas. (**B**) Localized invasion into the surrounding skeletal muscle tissue. (**C**) The tumor predominantly comprised uniform short spindle to ovoid cells with hyaline or eosinophilic cytoplasm. (**D**) Some areas exhibited microcystic morphology, with an absence of cellular atypia. (**E**) Regions containing blood sinusoid-like structures. (**F**) Collagenized regions within the tumor

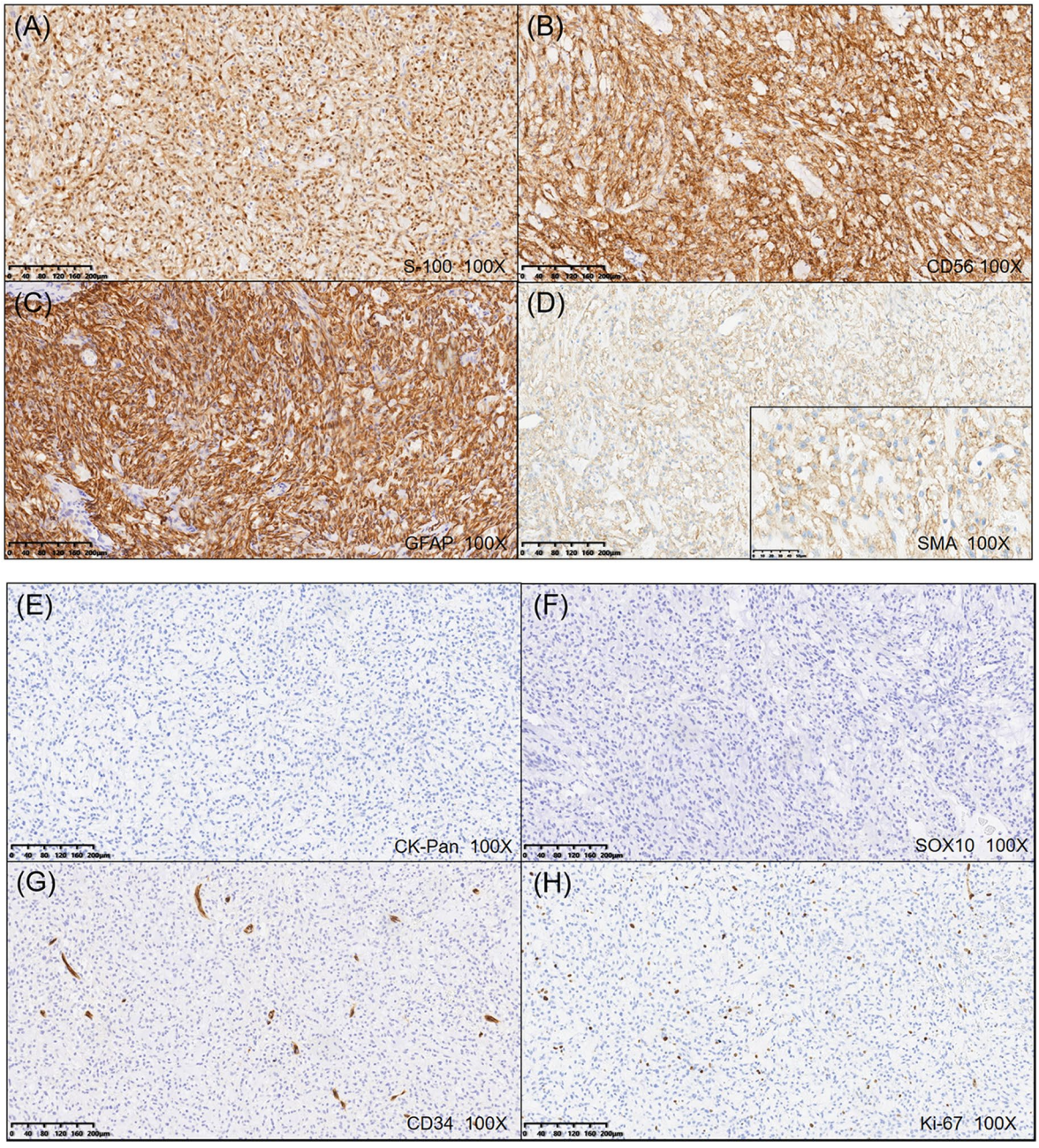

Fig. 3 Photomicrographs of immunohistochemical staining. (**A**) Positive staining for S100. (**B**) Positive staining for CD56. (**C**) Positive staining for GFAP. (**D**) Weakly positive staining for SMA. (**E**) Negative staining for CK-pan. (**F**) Negative staining for SOX10. (**G**) Negative staining for CD34. (All images, IHC ×100 magnification)

previously known as *MKL2*) fusion product, involving *RREB1* (located at 6p24.3 [17]) and *MRTFB* (located at 16p13.12 [18]). (The differential diagnosis has been summarized in Table 1.)

The *RREB1::MRTFB* fusion has been identified in various tumors, most commonly ECTs (Summaried in Tables 2 and 3). In a series of 21 cases, Dickson et al. [9] reported *RREB1::MRTFB* fusions in 19 cases (90%) of ETC, with one case showing no known genetic changes and another harboring an *EWSR1::CREM* fusion. Additional studies [10, 19–23], summarized in Table 3, have reported 11 sporadic cases with the *RREB1::MRTFB* fusion. Of these, two cases resembled biphenotypic sinonasal sarcoma [10, 22], two mimicked

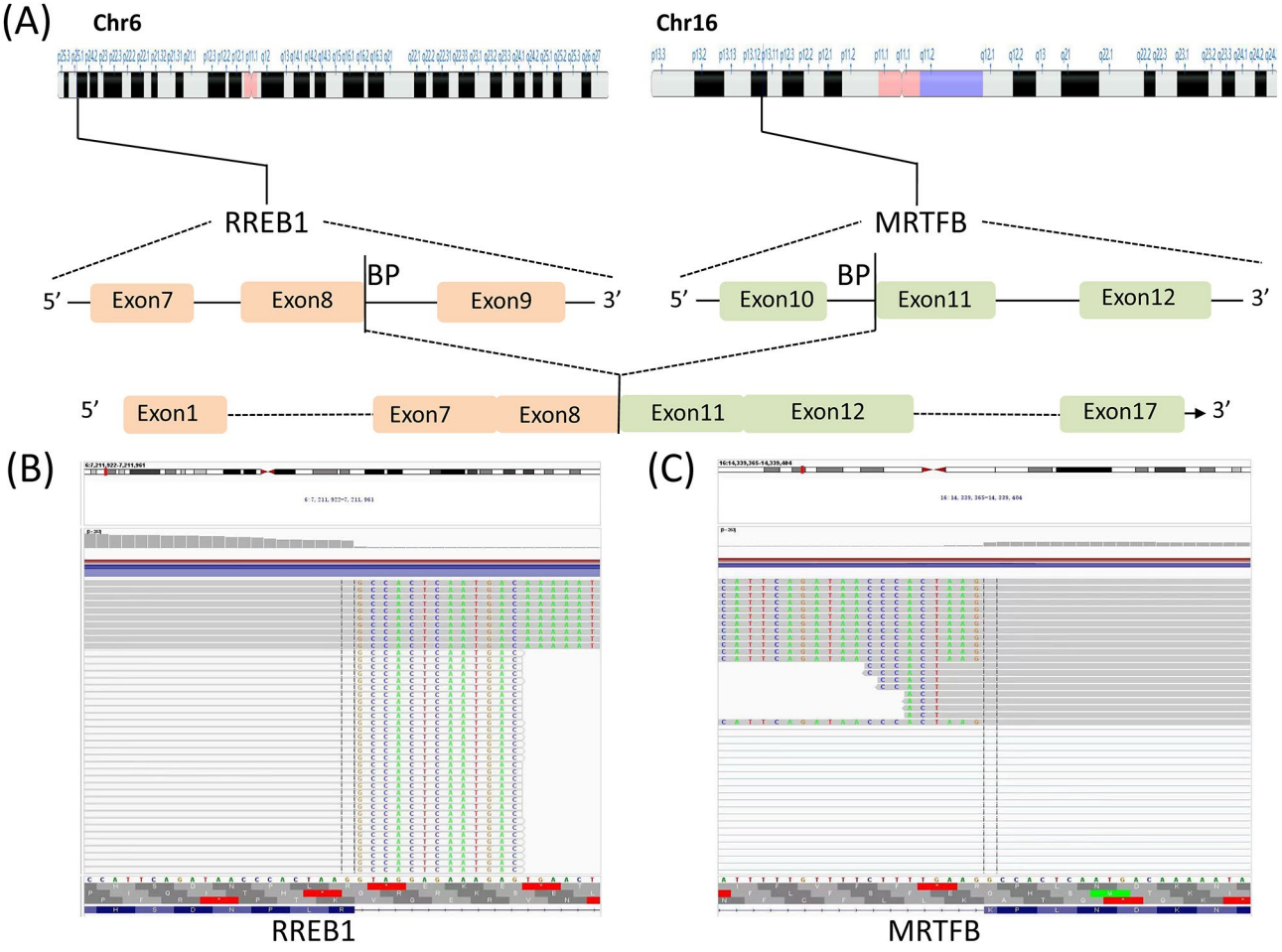

Fig. 4 *RREB1::MRTFB* fusion gene analysis. (**A**) Schematic representation of the fusion event between *RREB1* (orange) and *MRTFB* (green). The *RREB1* gene is located on chromosome 6p, and the *MRTFB* (formerly *MKL2*) gene is located on chromosome 16p. Lines and filled boxes represent intronic sequences and coding exons, respectively. BP, breakpoint. (**B**, **C**) Representative screenshots from the Integrative Genomics Viewer showing split reads mapping to the 3′ region of *RREB1* exon 8 and the 5′ region of *MRTFB* exon 11

rhabdomyosarcoma [21], and the remaining cases were reported as unclassified fibromyxoid neoplasms.

Siegfried et al. [22] proposed that the *RREB1::MRTFB* fusion promotes MRTFB translation, regulating both neural and myogenic differentiation, similar to the role of *PAX3* fusions in biphenotypic sinonasal sarcoma [24, 25]. This mechanism aligns with the immunophenotype of ECT, which shows neurogenic markers (S100 protein) and myogenic markers (SMA, desmin, and myosin). Further research is needed to elucidate the pathogenesis.

According to existing literature, ECT follows a benign course, with no reported metastases. However, five cases of recurrence have been documented, with follow-up durations ranging from 3 months to 10 years after initial resection [1, 2, 9, 26]. Adequate surgical excision is critical for favorable patient outcomes.

Table 1 Pathological differential diagnosis for ECT

Tumor	Clinical features	Histopathology	Immunohistochemistry	Molecular findings
ECT	Occurring in the tongue, no significant sex predilection	Small round-to-fusiform cells arranged in sheets, with a myxoid or hyalinized stroma. Occasionally, hemorrhage, and cyst formation can be seen.	Most show GFAP, S-100, and CD56 expression; Variable expressed for CD56, SMA; negative for CK, SOX10, P63	Most show RREB1-MRTFB fusion; subset show EWSR1 rearrangement
Hemangioblastoma	Occurring in the central nervous system	Rich in small blood vessels and large vacuolar stromal cells, with clear boundaries	Most positive for alpha-inhibin, variable positive for NSE, S-100, calretinin; negative for GFAP, CD10	VHL
Microcystic/reticular schwannoma	Predilection for the head, neck, and limbs; young patients	Bland ovoid, spindled cells; may demonstrate reticular or focal Microcapsules architectural patterns	Positive for S-100, SOX10, CD56; negative for CK, EMA, SMA, CD34	Not applicable
Monophasic spindle cell synovial sarcoma	The lower extremities; young adults with a male preponderance	Spindle cells arranged in fascicles with nuclear atypical	Expression TLE1, CD99, Bcl2, CD56; patchy positive for EMA, S-100	t(X;18)(p11;q11) translocation; SYT-SSX gene fusion
Solitary fibrous tumor	Intra-thoracic is the most common location; predilection for female	Oval to spindle-shaped nuclei cells; display a hemangiopericytic growth pattern, known as "staghorn" blood vessels, and perivascular sclerosis	Positive for CD34, BCL-2, CD99, STAT6; negative for CK, S-100, SOX10, SMA	NAB2-STAT6 gene fusion; GRIA2 highly expression
GLI1-Altered Mesenchymal Tumors	Predilection for the tongue; present in younger to middle-aged adults	Ovoid-to-round or vaguely epithelioid cells arranged in compact or ill-defined nests that are separated by a well-developed arborizing capillary network	Variable expression of SMA, CD56, S-100, NSE, P16, GFAP, calponin	GLI1-fusion-positive tumors (e.g. ACTB::GLI1); GLI1-amplified tumors
Non-ossifying fibromyxoid tumor	Often based within the subcutaneous tissues of proximal limbs and limb girdles	Well-circumscribed; absent ossifying; uniform, round, or ovoid epithelioid cells with defined cytoplasmic borders, within a myxohyaline or fibromyxoid stroma	Positive for vimentin, CD10, S-100, NSE, desmin, TFE3; negative for CK, SOX10; variable expression INI-1	PHF1 gene rearrangement (eg. PHF1-TFE3)
Soft tissue myoepithelioma	Most common in the limb girdles and extremities; in younger age group on average	Epithelioid cells with a chondromyxoid stroma; ductal differentiation or plasmacytoid morphology	Positive for AE1/AE3, Cam5.2, S-100, SOX10, GFAP, SMA, Calponin; Variable expression INI-1	EWSR1 gene rearrangement or FUS abnormalities

ECT, ectomesenchymal chondromyxoid tumor; GFAP, glial fibrillary acidic protein; SMA, smooth muscle actin; CK, keratin; RREB1: ras responsive element binding protein 1; MRTFB, myocardin related transcription factor B; EWSR1, EWS RNA binding protein 1; NSE, neuron-specific enolase; VHL, Von Hippel-Lindau; EMA, epithelial membrane antigen; TLE1, transducin-like enhancer protein 1; SYT: synaptotagmin; SSX, synovial sarcoma X breakpoint; NAB2, NGFI-A binding protein 2; STAT6, signal transducer and activator of transcription 6; GRIA2, glutamate receptor Ionotropic AMPA 2; GLI1, glioma-associated oncogene homolog 1; ACTB, beta-actin; INI-1, Integrase interactor-1; PHF1, PHD finger protein 1; FUS: fused in sarcoma

Table 2 Summary all ECT cases reported with the RREB1- MRTFB gene fusion

Authors	Age	Sex	Location	Size (cm)	Immunohistochemistry	Follow-up
Dickson et al. 2018 case1	31	F	Tongue, NOS	0.9	Positive for S-100; negative for GFAP, CK	NA
Dickson et al. 2018 case3	39	F	Tongue, Left dorsal	1.1	Positive for GFAP, S-100, SMA; negative for Myogenin	< 2ms
Dickson et al. 2018 case5	54	F	Tongue, dorsal	NA	NA	NA
Dickson et al. 2018 case6	23	M	Tongue, left anterior	3.0	Positive for GFAP, S-100; weakly positive for CK, SMA, Desmin	NER at 2ys
Dickson et al. 2018 case7	13	F	Tongue, right midline dorsal	2.0	Positive for GFAP; weakly positive for S-100	NA
Dickson et al. 2018 case8	45	F	Tongue, left dorsal	NA	Positive for GFAP, S-100; weakly positive for S-100	NA
Dickson et al. 2018 case9	54	M	Tongue, left anterior	1.2	Positive for S-100, SMA; negative for CK	NER at 1.4ys
Dickson et al. 2018 case10	35	M	Tongue, left anterior	0.7	Positive for S-100, SMA; weakly positive for GFAP; negative for CK, Desmin	Margins positive; Recurred at 41ms. NER after 4ys
Dickson et al. 2018 case11	14	F	Tongue, midline anterior	1.5	Positive for GFAP, S-100, Desmin; weakly positive for CK, SMA	Margins positive; NER after 8.5ys
Dickson et al. 2018 case12	49	F	Tongue, dorsal lateral	2.1	Positive for GFAP; weakly positive for S-100, CK, Desmin	NA
Dickson et al. 2018 case13	33	F	Tongue, NOS	NA	Positive for GFAP; weakly positive for S-100, CK, Desmin	NA
Dickson et al. 2018 case14	50	F	Tongue, dorsal tip	2.4	Positive for GFAP; weakly positive for S-100, Desmin; negative for CK	NA
Dickson et al. 2018 case15	53	F	Tongue, NOS	NA	Positive for GFAP	NA
Dickson et al. 2018 case16	59	M	Tongue, NOS	NA	Positive for GFAP; weakly positive for S-100	NA
Dickson et al. 2018 case17	31	F	Tongue, NOS	NA	Weakly positive for GFAP	NA
Dickson et al. 2018 case18	51	M	Tongue, midline dorsal, anterior	2.5	Positive for GFAP, S-100, SMA; weakly positive for CK, Desmin	NA
Dickson et al. 2018 case19	40	F	Tongue, right dorsal	1.0	Positive for GFAP; weakly positive for S-100, CK, Desmin	Margin focally positive, but no report of recurrence
Dickson et al. 2018 case20	51	M	Tongue, NOS	0.7	Positive for GFAP, Desmin, SMA; weakly positive for S-100, CK	Margin focally positive, but no report of recurrence
Dickson et al. 2018 case21	15	F	Tongue, right dorsal, posterior	0.7	Positive for GFAP; weakly positive for S-100, CK; negative for SMA, Desmin	NA
Bubola et al. 2021 case	37	F	Mandible	2.4	Positive for S-100, SMA, CD56; weakly positive for GFAP, Desmin; negative for CK, SOX10, calponin, myogenin	NA
Present case	20	F	The bottom of foot	4.4	Positive for GFAP, S-100, CD56; weakly positive for SMA; negative for CK, SOX10, CD34, P63	NER at 3ms

RREB1, ras responsive element binding protein 1; MRTFB, myocardin related transcription factor B; ECT, ectomesenchymal chondromyxoid tumor; F, female; M, male; NOS, no otherwise specified; NA, not available; NER, no evidence of recurrence; ms, months; ys, years

Table 3 Summary the other cases reported with the RREB1- MRTFB gene fusion

Authors	Age	Sex	Location	Size (cm)	Reported as	Pathological findings	Immunohistochemistry (Main marker)	Follow-up
Siegfried et al. 2018 case	53	M	Oropharyngeal	3.5	Biphenotypic "oropharyngeal" sarcoma	Uniform, elongate spindle cells; absent pleomorphism	Positive for S-100, SMA; weakly positive for Desmin; negative for SOX10, P63, CK	NER at 10 ms
Makise et al. 2020 case1	25	F	Paravertebral	5.6	Mesenchyme tumor with RREB1–MRTFB fusion	Round to oval small cells with rare mitoses; Lack myxoid or chondroid stroma	Positive for GFAP, S-100, SMA, CK; negative for SOX10, P63, CD34, Desmin	NER at 27 ms
Makise et al. 2020 case2	73	F	Superior mediastinum	4.3	Mesenchyme tumor with RREB1–MRTFB fusion	Spindle cells with a sclerotic background; absent mitoses and necrosis	Positive for GFAP, S-100, SMA, CK; negative for SOX10, P63, CD34, Desmin	NER at 27 ms
Mechtersheimer et al. 2021 case	73	F	Biphenotypic sinonasal	3.5	Biphenotypic sinonasal sarcoma	Monotonous spindle cells with a collagenous stroma; low mitotic activity	Positive for S-100, SMA; weakly positive for CD34, EMA; negative for CK, GFAP, Desmin	NA
Agaimy et al. 2023 case1	61	M	Inguinal	8	Unclassified fibromyxoid neoplasm	Spindle cells arrange with storiform pattern; myxoid to nonmyxoid stroma	Focally positive for CD34, EMA; negative for CK, SMA, Desmin, S-100, SOX10	NER at 17 ms
Agaimy et al. 2023 case2	36	F	Presacral region	20	Unclassified fibromyxoid neoplasm	Admixture of large epithelioid tumor cell; numerous nuclear pseudoinclusions	Positive for S-100, CD68; focally positive for EMA; negative for CK, GFAP, CD34, SOX10	NA
Agaimy et al. 2023 case3	28	F	Jaw	NA	Chondromyxoid sinonasal hamartoma	Rich in bone and immature chondromyxoid mesenchymal	Positive for S-100; focally positive for CD56; negative for CK, ERG, Desmin	NA
Agaimy et al. 2023 case4	28	M	Parapharyngeal space	6	Unclassified spindle and round cell neoplasm	Neoplasm with solid and cystic areas; prominent primitive reticular-myxoid stroma	Positive for Syn, GFAP, CD56; focally positive for CD99; negative for EMA, P63, S-100, SOX10, Desmin, CD34	NER at 5 ms
Agaimy et al. 2023 case5	18	M	Posterior nasopharyngeal wall	3.3	Low-grade mesenchymal neoplasm with rhabdomyoblastic differentiation	Round to ovoid bland cells; prominent pseudofollicular	Positive for MyoD1; focally positive for Desmin; negative for S-100, GFAP, CD34	NA
Sumransub et al. 2022 case	26	F	Oropharyngeal tongue	5.1	Variant Rhabdomyosarcoma or Aggressive Variant of ECT	Round, ovoid, or spindle-shaped tumor cell with focal mild atypia; absent mitoses	Positive for Syn, GFAP, CD56, S-100 and Desmin	NA
Midey et al. 2023 case	57	F	Intracardiac	9	Spindle-Cell Mesenchymal Tumor	The cells had pale or eosinophilic cytoplasm and bland ovoid nuclei without mitosis	Positive for Syn, S-100 and INSM1;negative for epithelial, muscular, vascular, and melanocytic markers	NER at 5 ms

RREB1, ras responsive element binding protein 1; MRTFB, myocardin related transcription factor B; ECT, ectomesenchymal chondromyxoid tumor; F, female; M, male; NA, not available; NER, no evidence of recurrence; ms, months

Conclusion

To the best of our knowledge, this is the first reported case of a tumor with an *RREB1::MRTFB* gene fusion occurring in the sole of the foot. Based on comprehensive histological, immunohistochemical, and molecular pathological analyses, the tumor was ultimately classified as an ECT. However, the atypical anatomical location and overlapping morphological features pose a significant risk of misdiagnosis. Our findings provide additional evidence that the *RREB1::MRTFB* fusion is not confined to tumors in the head region, thereby increasing awareness of this rare entity.

Abbreviations

ECT	Ectomesenchymal chondromyxoid tumor
RREB1	Ras-responsive element-binding protein1
MRTFB	Myocardin-related transcription factor B
MKL2	megakaryoblastic leukemia-2
GFAP	Glial fibrillary acidic protein
MRI	Magnetic resonance imaging
SMA	Smooth muscle actin
NSE	Neuron-specific enolase
S100	S100 Calcium Binding Protein
CD56	Cluster Of Differentiation 56
Ki-67	Nuclear-associatedantigenki-67
CD34	Cluster Of Differentiation 34
ERG	ETS transcription factor
HMB45	Premelanosome protein, also known as PMEL
Melan-A	Melanoma-A
SOX10	SRY-related HMG-box 10
TFE3	Transcription factor binding to IGHM enhancer 3
FISH	Fluorescence in situ hybridization
NGS	Next-generation sequencing
CD99	Cluster Of Differentiation 99
TLE1	Transducin like enhancer of split 1

OFMT	Ossifying fibromyxoid tumor
INI1	Integrase interactor-1
PHF1	PHD Finger Protein 1
GLI1	Glioma-associated oncogene homolog 1
EWSR1	Ewing sarcoma breakpoint region 1 gene
CREM	Cyclic AMP-Response-Element Modulating protein
PAX3	Paired box gene 3
INSM1	Insulinoma-associated protein 1

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Author contributions
Each author made critical contributions to the conception and design of this paper. Y.D. wrote the main original manuscript text and W. L. prepared Figs. K.S. reviewed and edited manuscript text. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study obtained ethical approval and consent for participation from the Clinical Research Ethics Board of the First Affiliated Hospital, Zhejiang University School of Medicine. The images used in this study do not include patient records or identifiable information.

Consent for publication
Written informed consent for publication was obtained from all participants.

Competing interests
The authors declare no competing interests.

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