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(CASE REPORT)

Recurrent extra skeletal mesenchymal chondrosarcoma of the chest wall. Case report of a rare tumor and a brief review of the literature

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Abstract

Mesenchymal chondrosarcoma (MC) represents only 1% of all chondrosarcomas and, in approximately one-third of cases, is found in extra skeletal sites as extra skeletal mesenchymal chondrosarcoma (EMC). MC can be difficult to diagnose and treat because of its rare and aggressive presentation. We report the case of a 29-year-old female presenting with a gradually enlarging non-tender mass of 8 months duration in the upper part of her left chest wall. She was concerned about breast carcinoma. Imaging studies showed a loosely lobulated, calcified soft tissue mass without an osseous component and not attached to the bone. Core needle biopsy showed a biphasic histomorphology pattern with both undifferentiated small round blue cells and islands of well-differentiated hyaline cartilage. She was diagnosed with EMC, confirmed by molecular testing, which demonstrated a HEY1-NCOA2 gene fusion. The patient was treated by radical surgical excision followed by chemotherapy. Local relapse at the original site was observed 3 years after the remission. She had a repeat wide excision with chest wall reconstruction and subsequently received additional chemotherapy and remained disease-free for three more years until she was lost to follow-up.

This report highlights the value of multidisciplinary management of rare, high-grade sarcomas, such as EMC. Long-term survival of EMC is possible with curative-wide surgical excision and individualized adjuvant therapy.

Keywords: Mesenchymal Chondrosarcoma; Small Blue Round Cells; Immunohistochemistry; Molecular; Recurrence

1. Introduction

Mesenchymal chondrosarcoma (MC) is a rare chondrosarcoma with aggressive behavior characterized by the presence of primitive mesenchymal cells and mature cartilage. When this tumor occurs outside of bone in soft tissue, it is referred to as extra skeletal mesenchymal chondrosarcoma (EMC). [1] The EMC diagnosis is based on the recognition of this characteristic microscopic appearance, which consists of high-grade primitive blue cells as well as well-differentiated cartilaginous elements. Diagnosis of EMC as a soft tissue entity is, however, a challenging task due to its two-cell population, and when the sample is inadequate, it may only show one of the two components. [2] [1]

EMC has been described in various sites, including the meninges, orbit, retroperitoneum, uterus, kidney, prostate, thyroid, intravascular spaces, and soft tissues of the trunk and extremities. Its pathogenesis is largely unidentified, and it does not have a clear genetic or environmental risk factor. [3] [4] [5] [6] [7] [8] However, more and more molecular

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studies have shown that recurrent chromosomal translocations leading to EWSR1 gene fusing have been implicated in EMC tumorigenesis, especially EWSR1-NR4A3 fusion, suggesting a potential common pathogenesis with other Ewing family tumors. [9] EMC is difficult to diagnose because of its deep-seated site, varied manifestation, and long latency period toward recurrence or metastasis, which is even longer several years after the original treatment. These findings underscore the importance of long-term follow-up and a high index of suspicion when investigating deep soft tissue masses in young children. [10] [11]

The American Joint Committee on Cancer (AJCC) classification comprises eight different subtypes of chondrosarcoma. Central conventional chondrosarcomas are the most common and are subdivided into grade 1 tumors (low-grade) and grades 2-3 (high-grade), which exhibit higher-grade features. Likewise, peripheral chondrosarcomas conform to this grading scheme, with G1 classified as the conventional, low-grade variant and G2-3 as intermediate to high-grade disease. The others are periosteal chondrosarcoma, dedifferentiated chondrosarcoma, mesenchymal chondrosarcoma, and clear cell chondrosarcoma, which have distinct clinical and pathological characteristics. [12] Myxoid chondrosarcoma was not included in the cartilaginous tumor section in the latest edition of the WHO classification of cartilaginous tumors.

We report the case of a 29-year-old female with a mass in the left upper chest wall with the initial concern of breast cancer; the final diagnosis was EMC. We also provide a brief review of the medical literature on this infrequently occurring tumor.

2. Clinical presentation

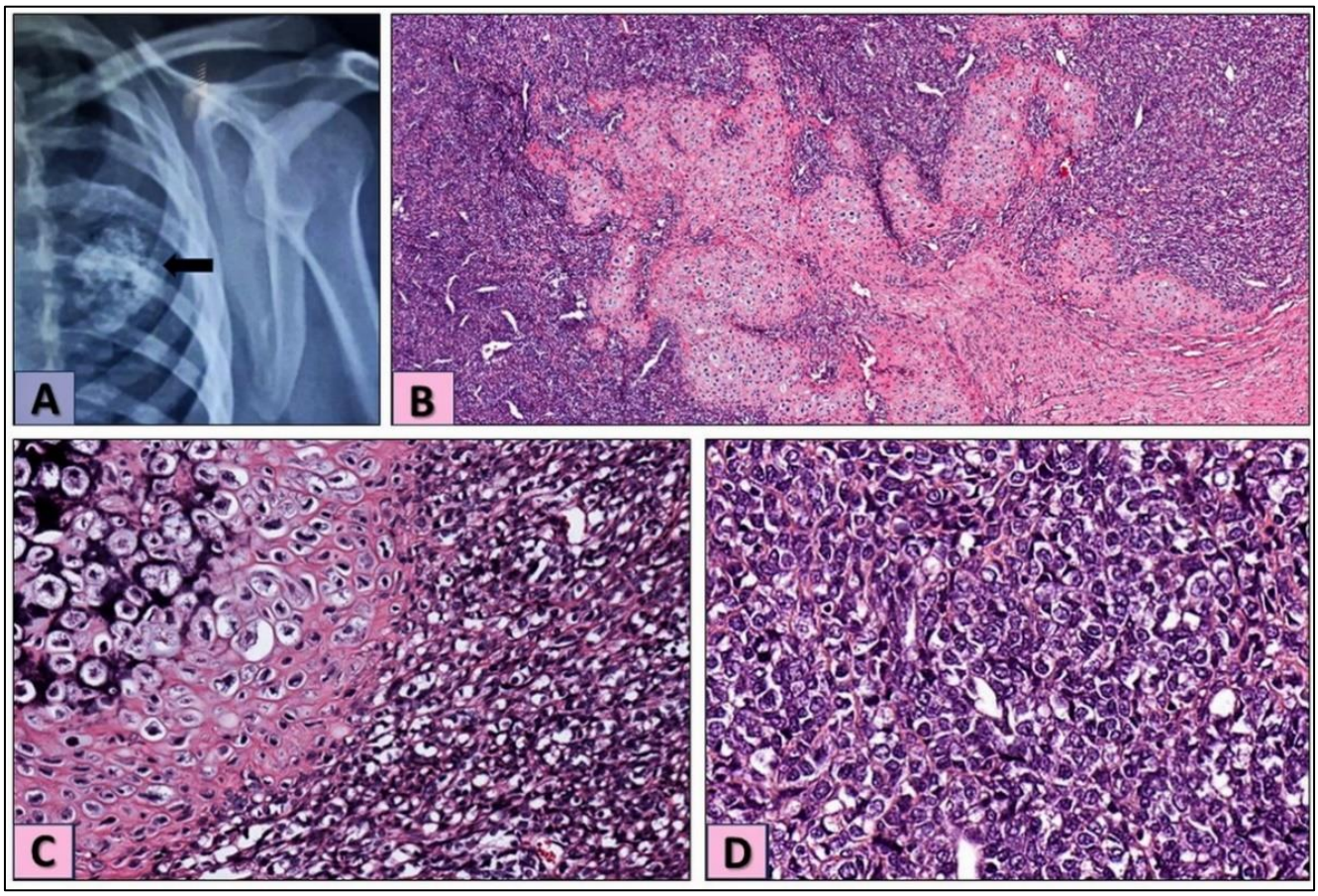
A healthy, active 29-year-old female was seen for an 8-month history of progressively enlarging, painless, palpable mass of the upper left chest wall. The mass was initially asymptomatic but had recently become enlarged, and she presented for medical attention, thinking it might be a breast tumor. She was known for a history of left shoulder dislocation, which occurred years before the presentation and was treated uneventfully. There was no personal or familial history of malignant disease or other notable medical conditions. All laboratory blood tests, including inflammatory markers and biochemical profiles, were within the normal limits.

Initial imaging tests included a chest X-ray, which revealed a calcified, round mass with a peripheral soft tissue rim. (Figure 1 A) Chest computed tomography (CT) scanning revealed a lobulated, destructive lesion with granular, ring, arc, and irregular calcifications. The lesion was found in the soft tissue behind the left pectoralis major muscle and was not connected to the surrounding bone or breast tissue. MRI studies revealed a lobulated and heterogeneous mass with mixed signal intensity in the soft tissue, measuring approximately 8 cm in maximum diameter. The lesion exhibited T2 hyperintense foci, likely corresponding to a chondroid matrix and T2 hypo intensity reflecting a solid component. The radiologic findings were nonspecific, and the differential diagnosis included both benign and malignant tumors, but a biphasic tumor with solid and cartilaginous components was suspected. As the mass was palpable, a core needle biopsy was performed for definitive tissue diagnosis.

Pathologic microscopic examination revealed a biphasic pattern of hypercellular sheets of solid, undifferentiated, small, round blue cells forming a hemangiopericytoma-like pattern sharply demarcated from islands of well-differentiated hyaline cartilage. The cellular solid component consisted of hyperchromatic small to intermediate-sized round and oval cells with a high nuclear-to-cytoplasmic ratio, similar in morphology to Ewing's sarcoma cells. Necrosis and a high mitotic rate (>10/10 high-power fields) were detected. Focal spindle cell pattern and foci of eosinophilic osteoid-like matrix were also seen occasionally. (Figure 1 B, C, D) The histomorphology features of a small blur round cell tumor led to consideration of several differential diagnoses, including synovial sarcoma, metastatic small cell carcinoma, Ewing's sarcoma, lymphoma, desmoplastic small round cell sarcoma, rhabdomyosarcoma, malignant solitary fibrous tumor, and EMC, which was the most likely diagnosis

Immunohistochemistry (IHC) played a critical role in the final diagnosis. The tumor displayed diffuse membranous CD99 positivity in the small cell component, strong SOX9 positivity in both components, and focal desmin reactivity. Cartilaginous zones were demonstrated using S100. Negative IHC staining for CD20, EMA, cytokeratin, myogenin, CD34, FLI1, and neuroendocrine markers excluded other potential mimickers. This immune profile, along with the characteristic biphasic histomorphology, supported a diagnosis of EMC in the soft tissue of the left chest wall. Molecular testing, performed at the family's request, revealed a HEY1-NCOA2 fusion gene, confirming the diagnosis. The high-grade nature of the tumor led to a recommendation by a multidisciplinary tumor board for complete surgical excision with wide margins followed by adjuvant chemotherapy. The mass was resected completely with a 2-cm margin all around. Grossly, the tumor measured 8 × 5 cm and was well-defined, lobulated, solid, firm, and gray-tan, with focal gritty white foci. Pathological examination of the excised tumor confirmed the initial biopsy diagnosis of EMC.

Postoperative adjuvant therapy included doxorubicin, ifosfamide, and etoposide chemotherapy. The patient responded well to treatment and was regularly followed up with imaging and clinical assessment. Three years after the treatment, a recurrence was identified at the site of prior surgery. The recurrent mass was widely excised with chest wall reconstruction by muscle flaps followed by more aggressive chemotherapy, including doxorubicin, ifosfamide, and vincristine. A successful postoperative recovery and treatment response was achieved, with no record of recurrence or distant metastasis in the 3 years of further follow-up until the patient was lost to follow-up.



1A chest X-ray showing calcified, round mass with a peripheral soft tissue rim (black arrow)

1B: Low power view showing biphasic pattern of hypercellular sheets of small, round blue cells forming a hemangiopericytoma-like pattern sharply demarcated from islands of well-differentiated hyaline cartilage (H&E stain X20)

1C: High power view showing well-differentiated hyaline cartilage (left side) and hypercellular sheets of solid, undifferentiated, small, round blue cells (right side) (H&E stain X40)

1D: High power view showing hyperchromatic undifferentiated, small, round blue cells with high mitotic activity (H&E stain X40)

Figure 1 X-ray and histomorphology features of the extra skeletal mesenchymal chondrosarcoma (EMC)

3. Discussion

3.1. History and classification

MC was first described by Lichtenstein and Bernstein in 1959 as a distinct variant of chondrosarcoma characterized by its biphasic histological pattern and aggressive clinical course. [13] The median age for MC is reported as 30 years in one large European study, which aligns with the second to third decade of life, while conventional chondrosarcomas predominantly affect middle-aged to older adults. MC accounts for less than 1% of all chondrosarcomas and exhibits no strong gender predilection. [2] Clinically, the overwhelming majority of chondrosarcomas seen in practice are low-grade primary central conventional tumors, which usually have a better prognosis than higher-grade tumors. The less common histologic subtypes (clear cell, mesenchymal, and dedifferentiated chondrosarcoma) are usually associated with less favorable clinical behavior and have more therapeutic implications for the treating physician. [14] Although

imaging and microscopic evaluation techniques have improved, chondrosarcomas remain challenging for clinicians to diagnose. Even experienced radiologists and pathologists often struggle to differentiate low-grade chondrosarcomas from benign enchondromas, as their imaging and histopathological findings can be very similar.

Similarly, segregating low-grade and high-grade chondrosarcomas can be quite challenging because the dissimilarities in cellularity are often slight and not immediately apparent. Although a systematic review of imaging features may be useful in differentiating between the diverse types of chondrosarcomas, the diagnostic dilemma in these cases highlights the challenges of caring for patients with cartilaginous bone tumors. [15] Table 1 summarizes the comparison of major clinically encountered chondrosarcoma subtypes.

Table 1 Comparison of Major Clinically Encountered Chondrosarcoma Subtypes

Type	**Typical Age Group**	**Location**	**Histology**	**Immunohistochemistry / Molecular**	**Behavior**	**Notes**
Conventional Chondrosarcoma	40–70 years	Long bones, pelvis, ribs	Lobulated hyaline cartilage; low-to-high grade	S100+; IDH1/2 mutations common	Locally aggressive; metastasizes if high-grade	Most common type
Clear Cell Chondrosarcoma	20–50 years	Epiphysis of long bones (femur, humerus)	Clear cytoplasm, central nuclei, bony trabeculae	S100+; SOX9+	Low-grade, slow-growing	Rare; often confused with chondroblastoma
Dedifferentiated Chondrosarcoma	50–70 years	Pelvis, femur	The juxtaposition of low-grade cartilage tumor with high-grade sarcomatous area	S100+ (cartilaginous part); IHC varies in dedifferentiated area	Highly aggressive; poor prognosis	The biphasic pattern is diagnostic; very poor survival
Mesenchymal Chondrosarcoma	Teens–30s	Axial skeleton, soft tissues (extraskeletal)	Small round blue cells + islands of cartilage (biphasic)	CD99+, vimentin+, S100 (cartilage only); EWSR1-NR4A3 fusion	High-grade; high recurrence/metastasis	Rare but distinct; commonly extraskeletal
Myxoid Chondrosarcoma	30–60 years	Soft tissues (deep extremities)	Myxoid stroma with cords of uniform cells	S100+, vimentin+; NR4A3 gene fusions (EWSR1/TAF15 etc.)	Intermediate-grade	Now considered a separate entity (Extraskeletal Myxoid Chondrosarcoma)
Periosteal Chondrosarcoma	20–40 years	The surface of long bones (femur, humerus)	Cartilage tumor on cortical bone with no medullary involvement	S100+; IDH mutations less common	Low to intermediate-grade	Arises from the bone surface may resemble periosteal chondroma

3.2. Imaging Features

The clinical presentations of EMC are variable and atypical and may even be similar to those of other types of soft tissue neoplasms. Nevertheless, some patterns may be suspicious in favor of EMC alone when combined with pertinent clinical and pathological information. [15] On radiographs and CT scans, EMC may be visualized as a soft tissue mass with focal or multifocal calcifications. The calcifications are usually arciform, stippled, or punctate and correspond to regions of cartilaginous formation. Nevertheless, in certain cases, particularly in undifferentiated tumors, there may be little or no calcification. [14]

MRI has more advantages for assessing the local extent of EMC and its relationship with peripheral structure. Signal intensity varied on the T1- and T2-weighted images. On T2-weighted images, cartilaginous foci generally demonstrate high signals and other more cellular or mesenchymal components exhibit intermediate or low signals. Following contrast administration, there is typically heterogeneous enhancement due to the biphasic histology and varying vascularity. [15] Larger and more aggressive tumors may exhibit areas of necrosis or hemorrhage. Functional imaging with PET-CT or bone scans can be used for staging and identifying distant metastases, with the lungs being the most common sites of metastasis, followed by the bones. EMC is a high FDG-avid tumor usually correlating with its aggressive behavior. [16]

Radiological signs are insufficient for diagnosis alone, and these features can be indistinguishable from other soft tissue sarcomas or cancers. Imaging should be considered in the context of the clinical presentation and verified with biopsy and ancillary studies, such as IHC and molecular testing. [15]

3.3. Differential Diagnosis

Differential diagnosis of EMC encompasses a wide range of tumors. Histomorphologically, EMC typically exhibits a biphasic distribution, consisting of unidentified small, round to spindle-shaped mesenchymal cells with densely packed nuclei and scattered, well-formed islands of cartilage that may become ossified. This biphasic structure is characteristic but can be overlooked in small biopsies, particularly if the cartilaginous component is scant. [17] When confronted with a small round cell tumor of soft tissue origin, the combination of Biphasic histology, Limited but targeted IHC and confirmatory molecular testing for HEY1:NCOA2 fusion is essential for distinguishing EMC from its histologic mimics. [9]

3.4. Immunohistochemistry (IHC)

EMC exhibits a characteristic immune profile with Positive CD99 (focal), vimentin in some areas, S100 (in the cartilage), and SOX9. Non-reactive usually includes Cytokeratin's, desmin, myogenin, and LCA (CD45). Ewing sarcoma is characterized by the expression of CD99 (diffuse), carcinoma (CK+), and lymphoma (LCA+). [18] The most confirmatory diagnostic molecular test for Ewing sarcoma is the identification of the EWSR1:FLI1 fusion gene. This is evident in the great majority of EMCs. The translocation, t (8;8) (q13; q21), is highly specific and contributes to diagnosis, particularly when histological findings are inconclusive. It also distinguishes EMC from other EWSR1-rearranged sarcomas. [10] Table 2 summarizes the main differential diagnosis of EMC.

Table 2 Key Differentials of Extra skeletal Mesenchymal Chondrosarcoma (EMC)

Entity	Overlapping Features	Distinguishing Features
Ewing Sarcoma	Small round blue cells	Lacks cartilage; CD99 diffuse; EWSR1-FLI1 fusion
Small Cell Osteosarcoma	Small round cells + osteoid	Presence of osteoid, not cartilage; SATB2+
Poorly Differentiated Synovial Sarcoma	Monotonous small round cells	May have hemangiopericytoma-like vessels; SYT-SSX fusion
Lymphoma	Small round cells	CD45+, pan-B/T cell markers
Metastatic Neuroendocrine Tumors	Small cells, vascular pattern	Synaptophysin, chromogranin+, keratin+

3.5. Treatment and Prognosis

Surgical resection with clear margins is essential in EMC management, particularly in cases involving the chest wall, as positive margins are associated with an increased risk of recurrence and a worse prognosis [19]. One of the most debated issues in managing chondrosarcomas is determining the width of surgical margins. Some research suggests that margin width may not directly impact survival, although it does significantly affect the risk of local recurrence in EMC [20]. Recurrence is especially common in EMCs of the chest wall and pelvis [21]. For this reason, surgery remains the only potentially curative option, although chemotherapy or radiation may be used beforehand depending on the patient's specific case [19]. That said, chemotherapy and radiation alone tend to be less effective, primarily due to the tumor's poor blood supply, dense extracellular matrix, and low number of actively dividing cells. [21[

Furthermore, radiation only seems to benefit those patients that have positive margins [1], while chemotherapy is saved for younger patients displaying aggressive histology. [10] EMCs pose special problems during surgery due to their location near vital structures of the chest, such as the great vessels, the heart, and the lungs. Our patient had an initial excision of an 8 cm tumor with a 2 cm margin and received typical adjuvant chemotherapy (doxorubicin, ifosfamide, etoposide). Although the patient achieved remission, recurrence occurred three years later. This prompted a second wide resection and complex chest wall reconstruction with muscle flaps. The patient's chemotherapy regimen was adjusted to include vincristine. The inclusion of vincristine in the patient's chemotherapy regimen demonstrates the necessity for adaptive treatment in EMC.

This case demonstrates the strong recurrence risk of the tumor and the persistent challenge of attaining durable long-term success with such aggressive neoplasms. The European Musculoskeletal Oncology Society (EMSOS), in a multicenter analysis, reported that almost half of those with localized disease progressed, with a recurrence after five years in 27% and a recurrence after more than a decade in 13%. [2] This literature places strong emphasis on the tumor's late ability to relapse and the necessity for long-term follow-up. Furthermore, although chemotherapy was reported to have a statistically significant decrease in recurrence risk ($P = 0.046$) and death ($P = 0.004$), long-term survival remains unpredictable, with an overall 10-year survival rate of approximately 60%. [2] Additionally, survival rates vary by anatomical site: cranial tumors had a 5-year overall survival rate of 74%, appendicular tumors 50%, and axial tumors 37%. [22] Axial tumor site, as in our patient with chest wall tumor, did not independently lead to poor outcome, but complete surgical resection with negative margins and adjuvant chemotherapy continues as essential for a favorable prognosis.

3.6. What does the future hold

EMC is very rare, but a growing body of evidence suggests that it should serve as a new opportunity in the era of advanced technology, thanks to its aggressive course of disease and complicated biology. The present work reflects how diagnostic and therapeutic advances are beginning to change the way we approach such challenging soft tissue tumors. Our case highlights the importance of integrating clinical, radiological, pathological, and molecular data, serving as a reminder of how subsequent technologies can further enhance patient outcomes. [23]

The most notable progress is molecular profiling. The recurrent HEY1-NCOA2 fusion gene has become a highly significant diagnostic marker for EMC. Its identification would be beneficial for confirming the diagnosis as well as for allowing selective therapy in the future. As molecular testing, such as next-generation sequencing (NGS), becomes more widely available, it should become increasingly possible to diagnose EMCs sooner and more accurately, thereby minimizing diagnostic errors and stratifying risk. Moreover, non-invasive diagnostic techniques, such as liquid biopsies, may aid in the real-time monitoring of tumor growth and response to treatment. [24]

In the imaging domain, present radiologic findings are usually nonspecific and are shared with other sarcomas. However, new AI (artificial intelligence) and radiomics techniques may change that. Machine learning algorithms trained on large datasets can assist radiologists in identifying patterns that are undetectable to the human eye, facilitating early detection and differential diagnosis. Such tools may also improve the follow-up strategy by estimating the risk of recurrence based on imaging characteristics. [25] [26]

From a therapeutic perspective, the scientific community has not reached a consensus on systemic therapies for EMC. Wide surgical excision is the treatment of choice, occasionally supplemented by radiation. However, the way forward looks significantly better with the introduction of targeted therapies that focus on the tumor's genetics. Studies of molecular pathways targeted by HEY1-NCOA2 and other such fusions might lead to the discovery of therapeutic targets. Moreover, drug repositioning approaches and personalized in vitro drug sensitivity testing could provide tailored treatment alternatives, particularly for metastatic or locally advanced diseases. [23]

In addition, tailored treatment based on a multidisciplinary tumor board is becoming increasingly important. With further development of rare tumor registries and collaboration on rare tumor treatment in recent years, oncologists may be better able to obtain real-world data, treatment strategies, and new treatment methods in the future. Multidisciplinary tumor boards involving genomics, pathologists, radiologists, surgeons, and clinical oncologists are instrumental in arriving at thoughtful, one-decision in such rare entities. [23] [24]

The long-term follow-up is a significant challenge due to the high recurrence potential of EMC. Although current monitoring recommendations often mirror those for soft tissue sarcoma, future follow-up may increasingly be informed by predictive biomarkers and molecular residual disease testing, as well as digital surveillance approaches tailored to specific risk measures. [16]

EMC is still a challenging and rare tumor, but the future seems promising. We hope that the advent of molecular diagnostics, artificial intelligence, and targeted therapy will change the way we address this disease, from diagnosis to treatment to surveillance, making us even closer to finally delivering on the dream of ultimate personalized care for those patients with an unusually aggressive neoplasm. [14]

3.7. What did we learn from this case

Our experience with this example of EMC taught us about the diagnostic dilemma, the aggressive nature of this malignancy, and the lack of standardized management tools to combat it. There were also a few key takeaways that not only taught us new things about EMC but may also be helpful for other physicians finding themselves in a similar diagnostic conundrum. This case emphasizes the value of a multidisciplinary assessment for an undifferentiated soft tissue tumor, especially at atypical sites, such as the chest wall. The differential diagnosis of our patient was rather wide on initial imaging, including from benign lesions up to high-grade sarcomas. An accurate diagnosis can only be made through the coordinated efforts of radiology, pathology, surgical oncology, and molecular diagnostics.

Abbreviations

Mesenchymal chondrosarcoma (MC); Extra skeletal mesenchymal chondrosarcoma (EMC); Immunohistochemistry (IHC); Next-generation sequencing (NGS)

4. Conclusion

EMC is a rare and highly aggressive malignancy that is diagnostically and therapeutically challenging due to its soft tissue location and extensive overlap with small round blue cell tumors. Our case highlights the paramount importance of a multidisciplinary diagnostic workup that involves clinical suspicion, advanced imaging, detailed histopathology, immunohistochemical evaluation, and molecular validation. In the evolving diagnostic era, the inclusion of molecular studies, such as the EWSR1-NR4A3 gene fusion test, would enhance specificity, which can aid in the management of targeted therapy as well. This case contributes to the limited literature on EMC and highlights the importance of long-term follow-up due to the late recurrence and metastasis of EMC. More study and collaborative reporting will be needed to reinforce the knowledge and treatment of these challenging individuals with EMC.

Compliance with ethical standards

Disclosure of conflict of interest

All authors declare the following

Statement of ethical approval

Ethical review and approval were not required for the study on human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper

Other relationships

All authors have declared that no other relationships or activities could appear to have influenced the submitted work.

Patient's consent

The patient was lost to follow-up, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

Author contributions

All authors contributed equally to producing this manuscript

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