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Solid Aneurysmal Bone Cyst in the Rib: A Case Report with Review of Literature

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Abstract

Solid aneurysmal bone cysts (ABCs) are rare tumors first identified by Sanerkin et al in 1983. While they commonly occur in long bones and the vertebral column, their presence in the ribs is unusual and poses distinct diagnostic and treatment challenges. This report discusses a case of a giant solid ABC in an 11-year-old girl who presented with a mass on the left side of her chest. A CT scan revealed a large expansile lytic lesion on the left third rib, measuring $18 \times 17 \times 13.5$ cm, initially raising suspicion of Ewing sarcoma. A tru-cut biopsy identified a spindle cell neoplasm with multinucleated giant cells, indicative of an ABC, though giant cell-rich osteosarcoma needed to be ruled out. The patient underwent rib resection, and histopathological analysis revealed a neoplasm with solid fibroblastic areas and blood-filled cystic spaces separated by fibrous septa, along with scattered osteoclast-type giant cells. This case represents the largest solid ABC reported to date and underscores the crucial role of histopathology in diagnosing small biopsy samples.

Keywords
- giant aneurysmal bone cyst
- solid ABC
- pediatric thoracic tumor
- tumor of the rib

Introduction

Aneurysmal bone cysts (ABCs) are benign bone tumors that commonly affect long bones and the vertebral column. Their presence in the rib is rare, presenting distinct diagnostic and treatment challenges. The term "solid variant" of ABC was first introduced by Sanerkin et al in 1983 to describe cases with predominantly solid areas on histology,[1] although the fifth edition of the WHO classification does not recognize it as a distinct subtype. This article presents a rare case of a solid ABC in the left third rib of an 11-year-old girl, highlighting the clinical significance of distinguishing it from other pediatric bone tumors, particularly Ewing sarcoma, due to their differing treatment approaches.[2,3] To the best of our knowledge, this is the largest reported case of solid ABC in the published literature. This case highlights the importance of obtaining an adequate pretreatment biopsy that accurately represents the tumor, recognizing the subtle histomorphological differences between the giant cells in ABC and those in other tumors, particularly osteosarcomas, and conducting a robust multidisciplinary discussion to ensure the appropriate management of this rare tumor.

Case Report

An 11-year-old girl presented with a mass on the left side of her chest, first noticed a week earlier. An initial chest X-ray revealed a mass in the left mediastinum. Further evaluation at our hospital with a CT scan showed a large, heterogeneously enhancing, expansile lytic lesion in the left

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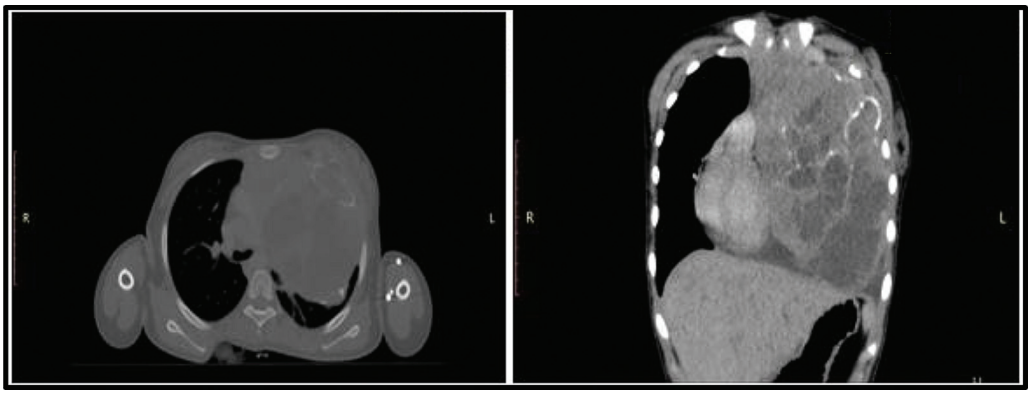

Fig. 1 CT chest: Large heterogeneously enhancing expansile lytic lesion occupying the entire left hemithorax and arising from the left third rib, measuring 13.7 × 18 × 17 cm in size.

hemithorax, originating from the left third rib and measuring 13.5 × 18 × 17 cm. The lesion compressed the adjacent lung and was near the main pulmonary trunk and cardiac border, with indistinct fat planes, raising suspicion of Ewing sarcoma (►**Fig. 1**).

A tru-cut biopsy of the mass revealed a spindle cell neoplasm arranged in fascicles, interspersed with ropy collagen and scattered osteoclast-type giant cells (►**Fig. 2A**), along with prominent blood vessels. The spindle cells exhibited moderate anisonucleosis, vesicular nuclei, inconspicuous nucleoli, and scant-to-moderate pale eosinophilic cytoplasm, with a low mitotic rate. No necrosis was observed. Immunohistochemistry demonstrated variable staining for smooth muscle actin (SMA) and S100, while pan cytokeratin (PanCK), Desmin, CD34, signal transducer and activator of transcription 6 (STAT 6), epithelial membrane antigen (EMA), and transducin-like enhancer of split 1 (TLE1) were negative. The Ki-67 proliferation index was 20% (►**Fig. 2B–D**). These findings were consistent with a solid variant of an ABC, although giant cell-rich osteosarcoma remained a consideration in the differential diagnosis.

Due to clinical suspicion of malignancy, a repeat biopsy was conducted, which confirmed the previous findings. Following a multidisciplinary tumor board discussion, treatment options included targeted therapy with Denosumab

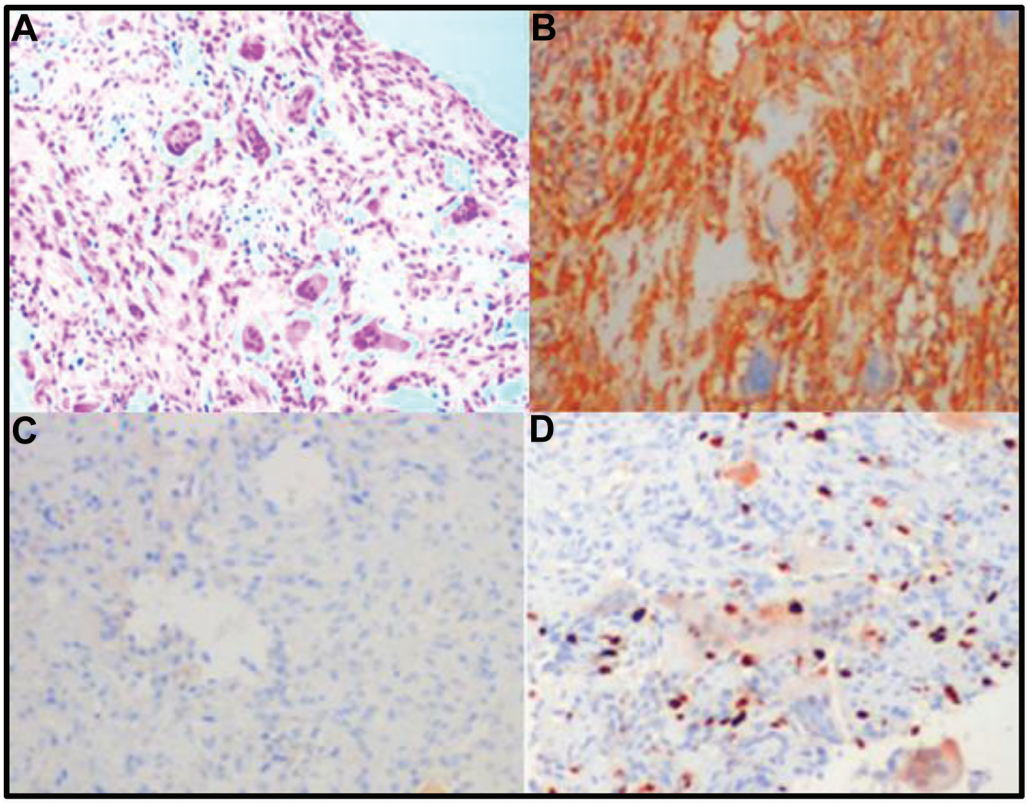

Fig. 2 (**A**) Spindle cells arranged in fascicles with intervening ropy collagen and scattered osteoclast-type giant cells (H&E 40 ×, 100 ×). The neoplastic cells, exhibiting strong membranous staining for SMA (**B**), no immunostaining with PanCK (**C**), and a Ki-67 proliferation index of 20% (**D**).

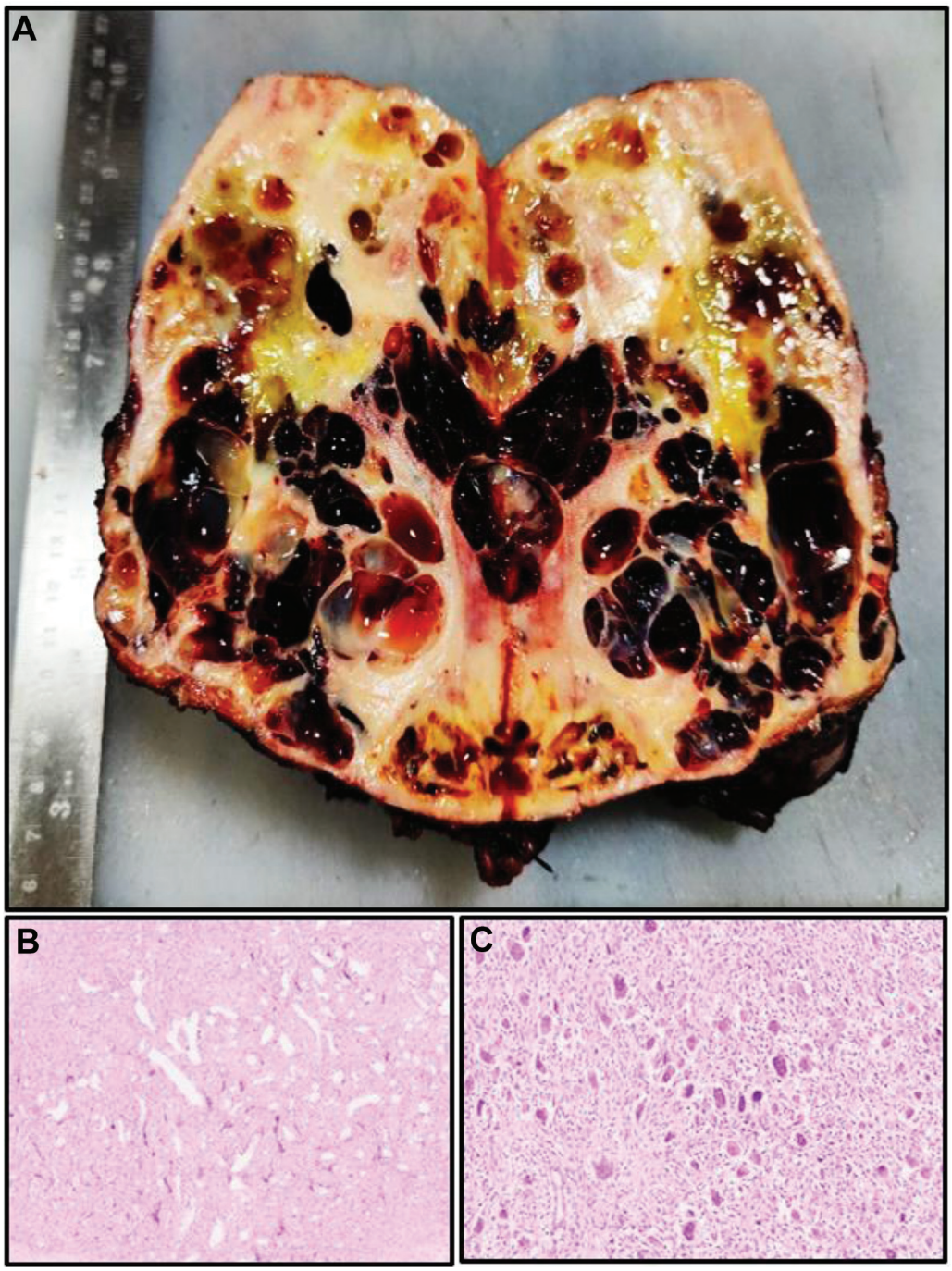

Fig. 3 (**A**) Gross examination of resected rib mass: Multiloculated blood-filled cystic spaces with solid areas. (**B**) Multiple blood-filled cystic spaces, separated by fibrous septa. (**C**) Cellular proliferation of bland fibroblasts with multinucleated osteoclast-type giant cells.

followed by surgical resection. After 3 months of Denosumab therapy, the patient underwent a left anterolateral thoracotomy, segmental resection of the third and fourth ribs, partial resection of the adherent pericardium, and reconstruction using the latissimus dorsi muscle.

The resected specimen comprised a large, globular, gray-brown mass measuring $17.5 \times 15 \times 10$ cm, with an attached rib segment of 4.5 cm. Serial sectioning revealed both solid and cystic areas, with mucoid and hemorrhagic regions (►**Fig. 3A**). No necrosis was observed. Microscopic examination showed trabeculae of lamellar bone along with a neoplasm characterized by multiple blood-filled cystic spaces separated by fibrous septa. The fibrous septa contained moderately dense fibroblasts, scattered multinucleated osteoclast-type giant cells, and reactive woven bone rimmed by osteoblasts (►**Fig. 3B, C**). Some areas of woven bone appeared basophilic. The mitotic rate was low, approximately 1 to 2 per 10 high-power fields, with no atypical mitoses. The pericardium, adjacent soft tissue, and skeletal muscle were uninvolved by the tumor.

The final diagnosis was a solid ABC. A moderate response to Denosumab was observed, indicated by a reduction in

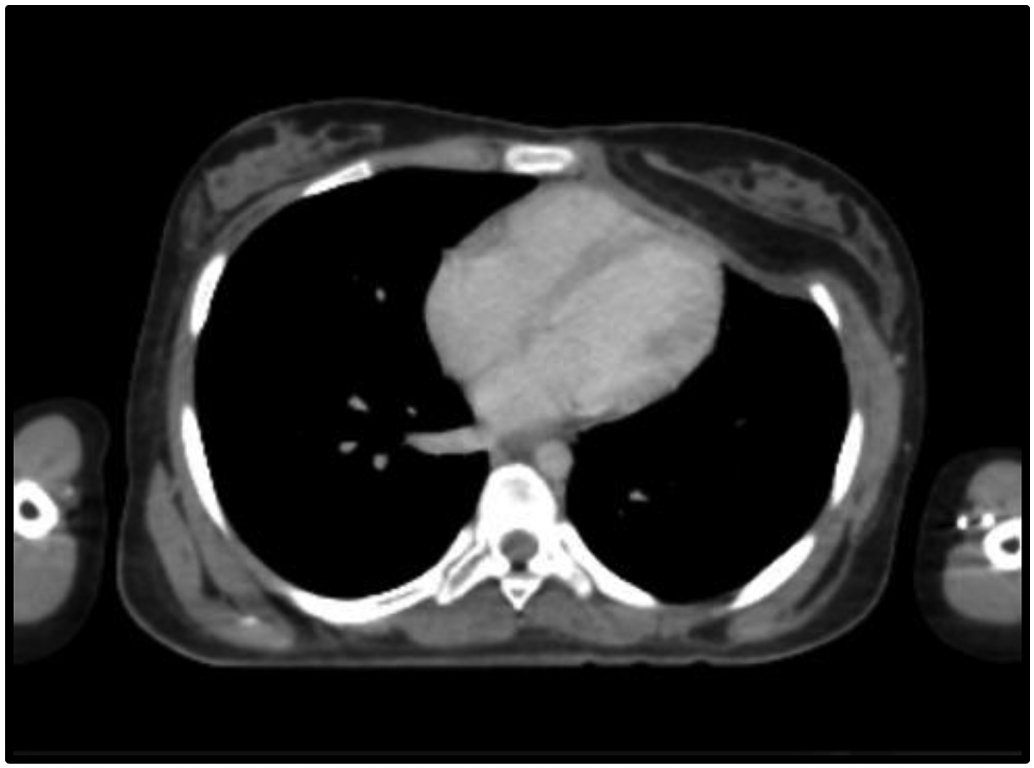

Fig. 4 Postoperative CT thorax showed postoperative changes of partial resection of the left third and fourth ribs with graft reconstruction. No abnormal enhancing lesion is seen at the site of surgery.

multinucleated giant cells and new bone formation. The patient tolerated the surgery well and recovered without complications. The child is under regular follow-up and is doing well 16 months postsurgery (►**Fig. 4**).

Discussion

Primary tumors of the rib are uncommon, comprising only 5% to 7% of all primary bone tumors, with ABCs in the rib being particularly rare.[4] First described by Jaffe and Lichtenstein in 1942 as benign, tumor-like lesions, classical ABCs are characterized by blood-filled cavities separated by thin septa. Lichtenstein attributed their development to local hemodynamic changes leading to increased venous pressure, whereas Jaffe suggested they arise secondarily in preexisting bone lesions. Recent research challenges the reactive theory of ABCs, instead identifying chromosomal translocations, particularly t(16;17)(q22;p13), as a recurrent cytogenetic abnormality. The *USP6* gene, which disrupts normal bone maturation, is now recognized as a key driver of ABCs.[5–7]

A neoplastic origin for primary ABCs is further supported by the identification of clonal chromosome 17p13 translocations that place the *USP6* oncogene under the regulation of the *CDH11* promoter. In a study of 52 primary ABC cases, 69% exhibited *CDH11* and/or *USP6* rearrangements—some with *CDH11–USP6* fusions, others with variant *USP6* rearrangements, and a few with variant *CDH11* rearrangements. Notably, these genetic alterations were exclusive to spindle cells in ABCs and absent from multinucleated giant cells.[8]

ABCs can be either congenital or acquired. Congenital ABCs, typically seen in children and young adults without prior trauma, differ from acquired ABCs, which are more common in adults with a history of trauma. Additionally, ABC-like areas can develop in other bone tumors that exhibit hemorrhagic cystic changes, historically referred to as "secondary ABCs." Common bone lesions associated with ABC include fibrous dysplasia, non-ossifying fibroma, chondromyxoid fibroma, hemangioendothelioma, and osteoblastoma. ABCs account for approximately 9.1% of all bone tumors and usually exhibit slow growth. Involvement of the ribs is uncommon, with around 29% of cases detected incidentally on routine chest X-rays. When symptomatic, ABCs present with pain (46%), a palpable lump (21%), dyspnea (7%), paraplegia (7%), or pathological fractures (7%).[9–14] In our case, the primary symptom was swelling, without respiratory distress—a notable finding given the tumor's significant size and extent. Measuring $18 \times 17 \times 13.5$ cm, this tumor represents the largest solid ABC reported in the literature.[4,15]

Radiologically, ABCs typically appear as lytic, expansile lesions with a thin bony rim, and aggressive cases may invade adjacent soft tissues. CT and MRI are valuable for diagnosis, with CT scans often revealing fluid-filled cavernous spaces, suggestive of ABC. On MRI, solid variant ABCs are primarily solid, though larger lesions may display cystic spaces containing fluid–fluid levels without internal septations. The differential diagnosis includes giant cell tumor (GCT), chondromyxoid fibroma, osteosarcoma, chondroblastoma, fibrous dysplasia, and metastasis.[16] In our case, imaging showed a large, heterogeneously enhancing, flurodeoxyglucose (FDG)-avid solid cystic mass arising from the left third rib, initially raising suspicion of Ewing sarcoma.

Macroscopically, ABCs typically appear as multiloculated lesions with blood-filled cystic spaces separated by tan-white, gritty septa. However, the presence of extensive solid areas, as observed in our case, suggests a solid variant of ABC.[17]

Microscopically, ABCs are well-circumscribed lesions composed of blood-filled cystic spaces and fibrous septa containing multinucleated giant cells and reactive woven bone.[18] Solid ABCs are distinguished by extensive fibroblastic proliferation with scattered osteoclastic giant cells and occasional focal osteoid production, but without overt malignant features. Given their histologic overlap, giant cell-rich osteosarcoma and GCT must be considered in the differential diagnosis (►**Table 1**). GCT is characterized by a uniform distribution of giant cells among stromal cells, whereas giant cell-rich osteosarcoma exhibits cytologic anaplasia and tumor osteoid production. Our case demonstrated features more consistent with solid ABC rather than GCT or osteosarcoma.[18]

Chondromesenchymal hamartoma of the chest wall, a benign pediatric tumor composed of hyaline cartilage, woven bone, spindle cells, and blood-filled cystic spaces, was not considered due to the absence of chondroid elements in the resected specimen. Ewing sarcoma, the most common primary malignancy of the chest wall in the pediatric population, represents an important radiologic differential diagnosis. This entity was excluded due to the absence of uniform small round cells showing immunopositivity for CD99 and NKX2.2, which are characteristic of the disease. The most frequent genetic abnormality in Ewing sarcoma is the t(11;22)(q24;q12) translocation, resulting in the EWSR1–FLI1 fusion transcript, seen in approximately 85% of cases. This is followed by the t(21;22)(q22;q12) translocation, producing the EWSR1–ERG fusion in about 10% of cases.[18]

Primary osteosarcoma of the rib is an uncommon entity, accounting for only 1% to 2% of all osteosarcomas. Radiographic findings typically include aggressive bone destruction with a large extraosseous soft tissue component containing osteoid matrix. Histopathologic confirmation relies on the identification of neoplastic osteoid and markedly pleomorphic malignant stromal cells. Primary bone lymphoma constitutes less than 5% of primary bone malignancies in children and most commonly affects the long bones, with primary involvement of the ribs being exceedingly rare.[17] Langerhans cell histiocytosis (LCH) is characterized by the proliferation of abnormal dendritic histiocytes and most frequently presents between 1 and 4 years of age. Approximately 80% of cases demonstrate osseous involvement, predominantly affecting flat bones such as the skull, vertebrae, pelvis, mandible, and ribs. Diagnostic LCH cells are identified by grooved, folded, indented, or lobulated nuclei with fine chromatin, inconspicuous nucleoli, and thin nuclear membranes. The characteristic histologic background includes variable numbers of eosinophils, histiocytes (both multinucleated LCH-type and osteoclast-type cells, particularly in bone), neutrophils, and small lymphocytes.[16,17]

Complete surgical resection is essential for achieving a cure in both benign and malignant chest wall tumors, as incomplete removal can lead to recurrence. The recurrence rate following curettage ranges from 20% to 70%, though the presence of *USP6* fusion does not impact prognosis.[19] In the

Table 1 Distinctive pathological features of common giant cell-rich bone lesions

Histological features	Solid aneurysmal bone cyst	Osteosarcoma-giant cell-rich variant	Giant cell tumor of bone
Distribution	Metaphysis of long bones, posterior body of vertebrae	Metaphysis of long bones, infrequently diaphysis	Epiphysis, rarely metaphysis of long bone, sacrum, and vertebral body, rarely small bones
Radiology findings	Lytic, expansile lesion with well-defined margins, shell of reactive bone formation with fluid–fluid levels	Permeative bone destruction with mineralization—a mixed lytic/sclerotic appearance Periosteal reaction and extraosseous extension	Eccentric lytic lesion, no mineralisation
Neoplastic stromal proliferation	Absent	Present	Absent
Osteoid	Reactive osteoid±	Malignant osteoid+	Reactive osteoid±
Atypia	Absent	Marked	Absent
Distribution of giant cells	Irregular	Irregular	Uniform
Mitotic activity	Maybe brisk	Brisk	Rare
Atypical mitoses	Absent	Present	Absent If present, suspect malignancy in GCT
Aneurysmal areas filled with haemorrhagic fluid	Present	May be present	May be present
Specific immunohistochemistry/ molecular profile	*USP6* rearrangements	No specific IHC or molecular markers	H3.3 p.Gly34Trp (G34W) IHC *H3-3A* gene mutation

Abbreviation: GCT, giant cell tumor.

present case, considering the advanced presentation and questionable resectability and proximity to great vessels and the cardiac border, Denosumab was given in the neoadjuvant setting. Although the National Comprehensive Cancer Network (NCCN) guidelines do not include a standardized protocol for Denosumab in the treatment of ABCs because of the lack of large-scale clinical trials, Denosumab *is*, however, approved for the treatment of GCT of bone, a histologically similar tumor. The use of ABCs is an extrapolation from its success in GCT. However, there was no radiological response to Denosumab therapy.[20,21]

This case study focuses on the misleading clinical presentation of this rare rib tumor and the importance of distinguishing this entity histomorphologically from the more common pediatric bone tumors like osteosarcoma and Ewing sarcoma. Although young age and open physes are associated with an increased risk of local recurrence, the pathological factors associated with recurrence in ABC remain elusive, as does the proportion and significance of the solid component. There is immense scope for the study of the molecular profile of the recurrent ABCs for better prognostication and management of these cases. The limitations of the study include the unavailability of molecular tests, especially to determine the partner genes for *USP6* associated with aggressive disease.

In conclusion, diagnosing solid ABCs on needle core biopsy can be challenging, particularly in distinguishing them from GCTs and giant cell-rich osteosarcomas in pediatric cases. The rib as the site of origin may not be evident without extensive radiologic assessment. A comprehensive panel of immunohistochemical markers can aid in ruling out other mesenchymal neoplasms. This case highlights the rarity of solid ABCs in the rib and underscores the importance of integrating histopathologic and radiologic findings for accurate diagnosis. The successful multimodal approach, combining targeted therapy and surgical resection, illustrates the complexity of managing such rare pediatric cases.[18,19]

Authors' Contributions

V.V.G.: Concept, design, definition of intellectual content, literature search, data acquisition, data analysis, and manuscript preparation.

D.V.: Definition of intellectual content, literature search, data analysis, manuscript preparation, and manuscript editing.

S.M.S.: Definition of intellectual content, data acquisition, and data analysis.

R.V.K.: Definition of intellectual content, literature search, data analysis, manuscript editing, and manuscript review.

A.K.C.: Definition of intellectual content, literature search, clinical studies, data analysis, and manuscript review.

G.N.: Clinical studies.

V.P.D.: Clinical studies.

Conflict of Interest

None declared.

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