Int J Burn Trauma 2026;16(5):187-190
www.IJBT.org /ISSN:2160-2026/IJBT0174181

Case Report

Chondroblastic osteosarcoma of the scapula in an adolescent: a rare case report with diagnostic challenges

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Received May 20, 2026; Accepted October 20, 2026; Epub October 25, 2026; Published October 30, 2026

Abstract: Osteosarcoma is the most common primary malignant bone tumor in adolescents, but the scapula is an exceptionally rare axial site that presents unique diagnostic and therapeutic challenges due to its hidden location beneath thick musculature. We report the case of a 15-year-old male who presented with a rapidly expanding left scapular mass, where imaging revealed an aggressive lesion with cortical destruction, deep muscular invasion, and synchronous bilateral pulmonary metastases. While an initial core needle biopsy suggested a Grade 1 chondrosarcoma, strong nuclear immunohistochemical positivity for SATB2 definitively confirmed high-grade chondroblastic osteosarcoma. Neoadjuvant chemotherapy via the MAP protocol (methotrexate, doxorubicin, cisplatin) was initiated, but the patient suffered severe systemic toxicity and absolute chemoresistance, rapidly succumbing to the disease during the second cycle. Scapular osteosarcoma frequently presents at an advanced stage with synchronous metastases due to delayed clinical detection, emphasizing the mandatory role of advanced immunohistochemical panels like SATB2 to prevent misdiagnosis of chondroblastic variants. Furthermore, this case underscores a critical therapeutic dilemma: in patients with a massive axial tumor burden and profound cachexia, standard high-dose chemotherapy can cause fatal systemic toxicity, highlighting an urgent need for risk-stratified, personalized treatment protocols.

Keywords: Chondroblastic osteosarcoma, scapula, SATB2 protein

Introduction

Osteosarcoma is the most common primary malignant tumor of bone and is defined by the formation of osteoid matrix by malignant mesenchymal cells [1, 2]. It accounts for approximately 15-20% of all primary bone sarcomas, with an incidence of about 2-4 cases per million population per year [1, 3]. The disease shows a characteristic bimodal age distribution, occurring most frequently in adolescents between 10-20 years of age, corresponding to periods of rapid skeletal growth, and less commonly in adults over 50 years, where it is often associated with underlying bone disorders [3, 4]. The tumor commonly arises in the metaphyses of long bones, particularly around knee, including the distal femur and proximal tibia [2, 5]. The genetic theory explains the development of the disease in the young population. Mutations involving tumor suppressor genes such as *TP53* and *RB1* play an important role, leading to uncontrolled proliferation of osteoblastic cells [3, 6]. Several predisposing factors have been identified, including prior exposure to ionizing radiation, pre-existing conditions such as Paget's disease of bone, and inherited cancer syndromes such as Li-Fraumeni syndrome [4, 6]. On histopathological examination, osteosarcoma is characterized by malignant spindle cells producing osteoid matrix, which is essential for diagnosis [5]. Definitive diagnosis relies on identifying highly pleomorphic, atypical mesenchymal cells with hyperchromatic nuclei, brisk atypical mitotic figures, and the deposition of amorphous, lace-like osteoid matrix directly produced by these tumor cells, which may be accompanied by areas of necrosis and haemorrhage [7]. Based on the predominant matrix, it may be further classified into osteoblastic, chondroblastic, or fibroblastic subtypes [5]. Radiologically, the tumor typically appears as an aggressive lesion with a mixed lytic and sclerotic pattern, ill-defined margins,

https://doi.org/10.62347/ABPN1931

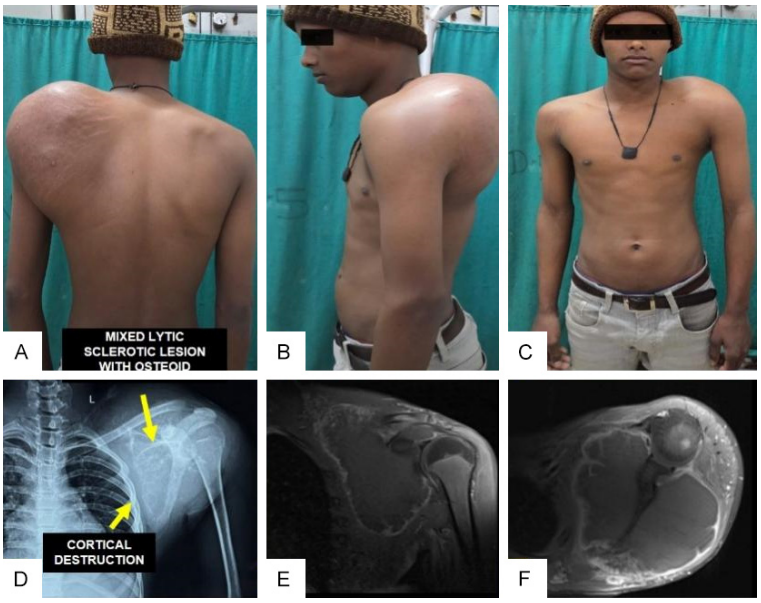


Figure 1. Combined clinico-radiological profile of a massive left scapular malignant tumor. (A-C) Gross clinical appearance: (A) Posterior view showing a massive hump-like mass filling the dorsal scapular region with overlying cutaneous striae; (B) Lateral view highlighting significant posterior projection; (C) Anterior view demonstrating shoulder girdle asymmetry and acromioclavicular joint disruption. (D) Anteroposterior radiograph: An aggressive, mixed lytic-sclerotic lesion arising from the scapular body, exhibiting extensive medial cortical destruction, trabecular disruption, and prominent tumor matrix mineralization. (E, F) T2-weighted MRI scans: (E) Axial cut displaying a lobulated, hyperintense mass arising from the scapula, infiltrating the subscapularis and infraspinatus muscles, and displacing the glenohumeral joint and humerus laterally beneath the stretched deltoid musculature; (F) Coronal oblique cut showing vertical expansion into the supraspinatus and teres major muscles, while demonstrating a clear tissue boundary with the spared glenohumeral joint line and proximal humerus.

cortical destruction, and periosteal reactions such as Codman's triangle or a sunburst appearance, often accompanied by an associated soft tissue mass reflecting extraosseous extension [2, 8]. Despite significant advances in treatment, including the use of neoadjuvant chemotherapy and surgery, osteosarcoma remains an aggressive malignancy with a marked tendency for early haematogenous spread to the lungs, which continues to be the most important determinant of prognosis. Current standard management utilizes a combination of multi-agent neoadjuvant chemotherapy (typically methotrexate, doxorubicin, and cisplatin), followed by wide surgical resection and adjuvant chemotherapy tailored to histological response. While this approach achieves a 5-year survival rate of 60-70% for localized disease, the prognosis drops significantly to under 20-30% for patients with metastatic or recurrent disease, highlighting a plateau in efficacy over recent decades [1, 2]. Thus, a multidisciplinary approach is necessary to diagnose the disease at the earliest for effective treatment and subsequent recovery.

Case presentation

A 15-year-old male presented with a two-month history of progressive left scapular pain and a rapidly growing dorsal mass that expanded from the size of a lemon to a melon. The mass was solitary and was accompanied by significant weight loss, anorexia, and a cachectic build. Physical examination revealed a massive tumor over the left dorsal scapular region, measuring 37 × 24 × 18 cm (**Figure 1A-C**). The overlying skin was hyperpigmented, shiny, stretched with striae, and exhibited visibly engorged veins with a local rise in temperature. On palpation, the mass had an irregular surface and a variegated consistency, being bony hard at some focal points and firm at others. The scapular spine was completely unpalpable. The lateral clavicle showed superior migration along the acromioclavicular joint. Significant left axillary lymphadenopathy was noted. The tumor caused gross restriction of left shoulder movement, but the distal neurovascular structures were spared.

Anteroposterior radiograph of the left shoulder girdle showed an aggressive, heterogeneous lesion with mixed lytic and sclerotic areas, ill-defined permeative margins, and visible osteoid matrix formation localized to the body of the scapula (**Figure 1D**). Marked cortical destruction was evident along the medial border of the scapular blade, disrupting the normal trabecular pattern, with enhanced extraosseous soft-tissue involvement.

Magnetic resonance imaging (MRI) confirmed a high-grade malignant sarcoma arising from the scapular body with internal calcifications

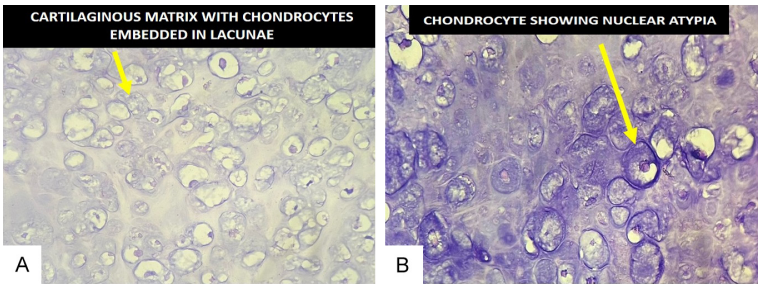


Figure 2. Histopathological images (100× magnification) showing a cartilaginous matrix with chondrocytes embedded in lacunae. There is diffuse growth and permeation of intratrabecular spaces with increased cellularity, nuclear atypia, and anisonucleosis, with peripheral displacement of nuclei in some cells. A. Cartilaginous matrix with chondrocytes embedded in lacunae (100×). B. Chondrocyte showing marked nuclear atypia (100×).

and direct infiltration into the supraspinatus, infraspinatus, and subscapularis muscles, while the glenohumeral joint and proximal humerus were spared (**Figure 1E**, **1F**). Thorax HRCT showed a large, calcified left scapular mass with multiple bilateral pulmonary nodules, confirming synchronous metastasis. Fine-needle aspiration cytology (FNAC) showed atypical cells having hyperchromasia, increased nuclear-to-cytoplasmic ratio, and prominent nucleoli, interspersed with osteoclast-like giant cells and material suggestive of osteoid. However, the core needle biopsy demonstrated a malignant cartilaginous matrix with atypical chondrocytes embedded within lacunae, intratrabecular permeation, hypercellularity, anisonucleosis, and focal myxoid changes, initially giving the impression of Grade 1 chondrosarcoma (**Figure 2**). This diagnostic discrepancy was resolved following immunohistochemistry (IHC) studies. The atypical stromal cells demonstrated strong nuclear positivity for SATB2 (immunoreactive score 3+), while the chondroid regions were positive for S100 (immunoreactive score 2+), definitively confirming the diagnosis of chondroblastic osteosarcoma.

The patient was initiated on a first-line neoadjuvant systemic chemotherapy regimen following the standard MAP protocol (High-dose Methotrexate, Doxorubicin, and Cisplatin). However, the clinical course was rapidly compromised by severe systemic toxicity and an absolute lack of clinical response. The patient exhibited immediate and profound intolerance to the therapeutic regimen, manifesting as intractable systemic weakness, severe myelosuppression, and worsening cachexia. During the second cycle of chemotherapy, the primary scapular mass continued to progress, causing acute mechanical distress, while the pre-existing synchronous bilateral pulmonary metastases expanded unchecked. Despite aggressive supportive care, the patient could not tolerate further therapeutic cycles and rapidly succumbed to the disease during this second phase of neoadjuvant therapy.

Discussion

Scapular osteosarcomas account for less than 10% of all cases and frequently present late due to their anatomical location [2, 3]. Because the scapula is a flat bone insulated by thick musculature, tumors can grow considerably before becoming clinically apparent. This is starkly illustrated in our case, where the patient presented only after the tumor had involved the entire shoulder girdle, accompanied by synchronous pulmonary metastases and severe cachexia [3, 4]. Based on this aggressive presentation, it is a crucial guiding opinion that clinicians must maintain a high index of suspicion and initiate rapid, low-threshold advanced imaging for any adolescent presenting with persistent, non-traumatic shoulder girdle pain. Plain radiographs typically reveal aggressive mixed lytic and sclerotic patterns, though classical periosteal reactions like Codman's triangle may be absent in flat bones. While MRI delineates local soft-tissue invasion, thoracic CT remains mandatory for detecting pulmonary metastases, the most common site of distant spread [8, 9].

Histopathologically, variants like chondroblastic osteosarcoma frequently mimic chondrosarcoma because initial biopsies may capture non-representative chondroid zones. Therefore, a vital diagnostic guiding opinion is that a multidisciplinary review integrating radiology and pathology is mandatory; when a biopsy yields a diagnosis of chondrosarcoma in an adolescent, clinicians must proactively utilize immunohistochemical panels including SATB2 to rule out a chondroblastic osteosarcoma variant [10].

SATB2 expression plays a decisive role in confirming osteogenic differentiation and preventing catastrophic treatment delays. The standard management of osteosarcoma utilizes the MAP protocol to shrink primary tumors and eradicate micrometastatic disease. While combination chemotherapy has raised 5-year survival rates for localized disease to 60-70%, patients presenting with synchronous metastatic disease experience significantly lower survival rates, ranging between 20-30% [1, 2]. Furthermore, axial location remains an adverse prognostic indicator due to the immense challenge of achieving wide surgical margins.

However, our case introduces a profound therapeutic dilemma and underscores a critical clinical guiding opinion: for patients presenting with massive axial tumor, poor performance status, and synchronous multi-organ metastasis, the immediate toxicity of first-line high-dose MAP chemotherapy can be completely fatal. As observed in our patient, who experienced rapid clinical decline and succumbed during the second cycle of neoadjuvant therapy, standard high-dose protocols can cause intolerable systemic toxicity without achieving a therapeutic response. Consequently, the management of high-risk, metastatic axial osteosarcoma requires a personalized, risk-stratified therapeutic approach; oncologists should heavily consider early treatment modifications, such as utilizing split-dose scheduling, incorporating less toxic alternative agents early on, or implementing upfront palliative radiation and aggressive supportive care rather than strictly adhering to standard high-dose systemic regimens in patients with advanced cachexia.

Conclusion

This case highlights the clinical presentation of chondroblastic osteosarcoma arising in an exceptionally rare anatomical site: the scapula. The case underscores the significant diagnostic challenges and histopathological pitfalls encountered when differentiating this variant from chondrosarcoma, highlighting the mandatory role of advanced immunohistochemical panels like SATB2 to achieve an accurate diagnosis. While literature emphasizes that early diagnosis and prompt combination chemotherapy are critical to optimizing survival, our patient's rapid decline and fatal outcome during the second cycle of neoadjuvant therapy reveal a harsh clinical reality. For patients presenting with massive axial tumor burden, synchronous bilateral pulmonary metastases, and profound cachexia, standard high-dose systemic regimens can cause intolerable toxicity. Ultimately, scapular osteosarcoma must be considered in any adolescent presenting with a rapidly expanding shoulder mass. Continued reporting of these advanced, high-risk cases remains vital to guide future, risk-stratified therapeutic protocols and personalized palliative approaches in patients unable to tolerate standard first-line protocols.

Disclosure of conflict of interest

None.

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