Mesenchymal Chondrosarcoma Presenting as Deep Vein Thrombosis: A Case Report Highlighting Diagnostic Pitfalls and the Role of Imaging–Pathology Correlation

Rokaya Elamrati[1]*, Valerie Sanchez[1], Jonathan Lavezo[2],[5], Uttamkumar Savariya[1],[2], Rajiv Rajani[3],[5], Shaked Laks[4],[5]

[1]*Department of Pathology, Texas Tech University Health Sciences Center, USA*
[2]*Department of Orthopedic Surgery, Texas Tech University Health Sciences Center, 79905, USA*
[3]*Department of Radiology, Texas Tech University Health Sciences Center, USA*
[4]*University Medical Center of El Paso, USA*

***Correspondence:** Rokaya Elamrati, Texas Tech University Health Sciences Center, 5001 El Paso Dr, El Paso, TX 79905, USA

rokaya.elamrati@ttuhsc.edu

Radiology Case. 2026 May; 20(5):1-9 :: DOI: 10.3941/jrcr.5995

AUTHORS' CONTRIBUTIONS
Rokaya Elamrati, B.S.: Data collection, manuscript writing, figure preparation
Valerie Sanchez, B.S.: Assisted with data collection, manuscript writing, and figure preparation
Jonathan Lavezo, M.D.: Provided pathological analysis and figure contributions
Uttamkumar Savariya, M.D.: Provided pathological analysis and figure contributions
Rajiv Rajani, M.D.: Contributed surgical perspective and clinical interpretation
Shaked Laks, M.D.: Supervised radiological analysis, manuscript review, and corresponding author duties

ACKNOWLEDGMENTS
We thank the clinical, pathology, orthopedic, and radiology teams at Texas Tech University Health Sciences Center and University Medical Center of El Paso for their support in managing the patient.

DISCLOSURES
The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CONSENT
Informed consent was obtained from the patient for publication of this case report and accompanying images.

HUMAN AND ANIMAL RIGHTS
All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1975 Helsinki Declaration and its later amendments.

FUNDING
No external funding was received for this study.

ABSTRACT

Mesenchymal chondrosarcoma (MCS) is a rare, aggressive malignancy that can arise in either bone or soft tissue, typically affecting young adults in the second to third decades of life. Because clinical symptoms and radiographic features are often nonspecific, accurate diagnosis requires careful integration of imaging, histopathologic, and immunohistochemical findings.

We report the case of a healthy 26-year-old woman with a large juxtacortical mass arising from the left superior pubic ramus causing compression of the iliofemoral vein. Imaging revealed a lobulated, mineralized lesion with cortical origin and matrix calcification. Pathologic diagnosis was made based on the presence of biphasic histology—comprising undifferentiated small round cells interspersed with islands of cartilaginous differentiation and detection of a specific genetic fusion on molecular testing. This case emphasizes the importance of imaging–pathology correlation in establishing an accurate diagnosis and guiding multidisciplinary management for mesenchymal chondrosarcoma.

CASE REPORT

BACKGROUND

Mesenchymal chondrosarcoma is a rare, high-grade malignant cartilage-forming tumor that primarily affects adolescents and young adults and accounts for approximately 3–10% of chondrosarcomas. It is characterized by aggressive behavior and propensity to metastasize. MCS demonstrates a biphasic histology composed of undifferentiated small round or spindle cells mixed with islands of hyaline cartilage, making diagnosis challenging due to overlapping features with other malignant bone and soft-tissue tumors. However, molecular testing with detection of the HEY1::NCOA2 genetic fusion is considered diagnostic.

Computed tomography (CT) and magnetic resonance imaging (MRI) are essential for evaluating mineralization patterns, cortical involvement, and soft-tissue extension; however, atypical locations may further complicate recognition. MCS can originate in bone or soft tissues; however, juxtacortical and pelvic presentations are uncommon and rarely reported. This case is notable for its rare pelvic site and juxtacortical location, as well as its unusual initial presentation with iliofemoral deep venous thrombosis due to extrinsic compression. These findings underscore the diagnostic complexity of MCS and highlight the importance of considering this entity in the differential diagnosis of juxtacortical pelvic masses in younger patients.

CASE PRESENTATION

Clinical and imaging findings

A 26-year-old woman presented to the emergency department with left leg swelling and pain and was diagnosed with a deep vein thrombosis (DVT). Records of a duplex ultrasonography performed at an outside hospital also suggested extrinsic compression of the left external iliac vein by a pelvic mass. However, images could not be obtained. She denied abdominal or pelvic pain, weakness, numbness, or tingling. Physical exam was notable for fullness of the left anterior pelvis compared with the right and unilateral left lower-extremity swelling.

Subsequent pelvic radiographs demonstrated a large partially calcified mass in the left anterior pelvis, which appears to extend from the cortex of the left superior pubic ramus. There was cortical thickening of the superior pubic rami with periosteal reaction without evidence of bone destruction (Figure 1). CT of the pelvis without contrast demonstrated a large soft-tissue mass with dense peripheral calcification and central ring-and-arc chondroid matrix (Figure 2A). Cortical origin of the mass from the superior pubic rami, as well as periosteal reaction, was also demonstrated (Figure 2B).

Pelvic MRI with and without contrast showed an approximately 10-cm heterogeneous mass centered in the extraperitoneal space of the left hemipelvis along the left superior pubic ramus. The mass was predominantly T1 isointense to skeletal muscle with a heterogeneous T2 hyperintense signal. Nodular areas of T1 and T2 hypointense signal were seen within the lesion, consistent with areas of calcification (Figure 3A, 3B). On post-contrast imaging, the lesion demonstrated heterogeneous enhancement and compression of the external iliac vein (Figure 3C, 3D). There was cortical thickening but normal bone marrow signal in the superior pubic rami, which confirmed the juxtacortical origin of the mass (Figure 4).

Management and follow-up

The patient was referred to orthopedic oncology and underwent an open bone biopsy of the left pelvic mass for pathologic diagnosis. A staging CT scan of the chest and abdomen was negative for metastasis. She was referred to medical oncology for adjuvant chemotherapy, including seventeen alternating cycles with a combination of Vincristine/Doxurubicin/Cyclophosphamide and Ifosfamide/Etoposide (VAC/IE). Repeat imaging after chemotherapy showed minimal decrease in size and enhancement. The patient is planned to undergo resection using a patient-specific three-dimensional printed cutting guide and a pelvic allograft for medial acetabulum reconstruction. Surgical resection is to be performed in conjunction with vascular surgery for vein compression and plastic surgery for wound closure.

Histopathology

H&E-stained sections showed a primitive mesenchymal tumor with biphasic morphology consisting of a hypercellular small round blue cell tumor with islands of calcified hyaline cartilage (Figure 5A). There was a branching “staghorn-like” pattern of blood vessels with small areas of matrix mimicking osteoid deposition. (Figure 5B). Special laboratory stains highlighted a protein called CD99 on the surface of the tumor cells (Figure 5C) and showed a few cells positive for a muscle-related protein called desmin (Figure 5D).

Molecular diagnostics

HEY1::NCOA2 fusion was detected in the tumor.

DISCUSSION

Etiology and demographics

Mesenchymal chondrosarcoma is a rare, aggressive cartilage-forming malignancy with a characteristic biphasic histology that poses significant diagnostic challenges [1, 2]. Chondrosarcomas are a heterogeneous group of malignant cartilage-forming tumors with several subtypes. Approximately 80% of chondrosarcomas are the conventional subtype. These tumors tend to occur in older patients (> 50 years old) and are composed of chondrocytes in a background of cartilaginous matrix [3]. Mesenchymal chondrosarcoma is a rare high-grade subtype comprising 3-10 % of chondrosarcomas, which primarily affects younger patients, most diagnosed in their late teens to early thirties (mean age of 25) [3, 4].

Mesenchymal chondrosarcomas can originate in bone and soft tissues. Compared to conventional chondrosarcoma,

skeletal involvement is more common in the axial than the appendicular skeleton [3]. Approximately one-third of mesenchymal chondrosarcomas are extraskeletal, and the CNS/meninges are among the most common extraskeletal sites [5]. MCS is characterized by aggressive local behavior and a propensity for early metastasis, most commonly to the lungs [6].

Histologically, MCS is characterized by a biphasic pattern with solid, highly packed areas containing small round cells or spindle-shaped undifferentiated mesenchymal cells adjacent to isolated cells or islands of hyaline cartilage [3,5,7]. In addition, foci of osteoid formation or calcification can be seen [5,7].

Tumors may contain areas of small round cells described as "Ewing-like" surrounding staghorn vasculature or spindle-shaped cells arranged around blood vessels described as "hemangiopericytoma-like." Diagnostic pitfalls arise when limited biopsies capture only one component of the tumor, risking misclassification of MCS as other cartilaginous or small round cell tumors [5,6]. This challenge is well documented in intracranial [8,9] and in orbital [10] MCS, where biopsies were inconclusive, and diagnosis required careful imaging–pathology correlation.

Immunohistochemical detection of certain biomarkers can also aid in distinguishing MCS from other tumors. Cartilage components typically express S100 and SOX9, which are seen in other chondrosarcomas, whereas the undifferentiated and small cell components are often positive for mesenchymal markers such as CD99, as seen in our case [5,7,11]. Rarely, a tumor can show scattered aberrant expression of myogenin and desmin, as was the case for desmin in this case example.

The etiology remains unclear; however, most cases involve recurrent gene fusions—most commonly the canonical HEY1::NCOA2 fusion, supporting a translocation-driven pathogenesis [5,12]. In our case, molecular confirmation of a HEY1::NCOA2 fusion was detected and helped establish the diagnosis. Recent reports have described novel HEY1::NCOA2 variants in intracranial cases [9]. Rare alternative fusions, such as IRF2BP2::CDX1, have also been identified, reflecting underlying molecular heterogeneity, and underscoring the role of genomic testing when morphology and immunohistochemistry are equivocal [13].

Clinical and imaging findings:

Due to variable locations, patients with MCS tend to present with a range of non-specific symptoms, which often delays diagnosis. Many patients present with mass-like swelling or some type of pain [5]. Similar to our case, patients may present with symptoms related to compression of nearby structures by the lesion [5]. To our knowledge, this is the first case presentation with iliofemoral DVT due to mass effect on the distal left external iliac vein. Compressive symptoms in other cases have been mostly neurological, resulting from mass effect on cranial nerves from extraosseous MCS located in the orbit [10] and intracranial dura [8].

Our patient presented several radiographic features that have been described previously in MCS. This includes a large mass (often > 10 cm) with a soft tissue component demonstrating areas of calcifications. CT better delineates intratumoral calcifications, which in MCS are variable in appearance, with several patterns often coexisting within the same lesion [14]. The most commonly reported is the typical chondroid "ring-and-arc" type of matrix, which was seen centrally in our case. Some lesions have a more "fine stippled" or granular calcification, which may only be visible on CT, while others (as seen in the periphery of the lesion in our case) appear heavily calcified [6]. Some authors have described a pattern of biphasic calcification, which can be seen in the MCS and was also visualized in our patient. In these cases, a clear delineation between the calcified and noncalcified soft tissue portions of the tumor can be made and is best seen on CT. Inclusion of this feature in the radiology report, when present, may be helpful in biopsy planning [6,14].

Typical osseous involvement, including cortical remodeling and periosteal reaction, suggesting aggressive behavior, was also observed. Of note, the commonly described destructive lytic and sclerotic appearance was less conspicuous in our case, likely secondary to minimal marrow involvement and juxta-cortical location [3,6].

On MRI, mesenchymal chondrosarcoma (MCS) typically appears as a lobulated lesion with well-defined margins [5,15]. Calcified components demonstrate low signal intensity on both T1- and T2-weighted images, whereas uncalcified areas show low signal on T1 and high signal on T2-weighted sequences [14]. The T2 signal is often heterogeneously hyperintense depending on the degree of T2 hypointense calcifications within the lesion [6]. In some cases, well-delineated T2-hypointense calcifications surrounded by T2-hyperintense chondroid matrix are seen, producing a speckled appearance on MRI (referred to as the "black pepper sign") [15-17]. Of note, MCS lesions—including our case—are generally less T2 hyperintense than conventional chondrosarcomas and lack the characteristic near-fluid T2 hyperintense lobules typically seen in conventional chondrosarcoma [5,6].

As demonstrated in our case, the enhancement pattern in MCS has been described as different from conventional chondrosarcoma with the absence of the typical chondroid septal and peripheral enhancement [3]. On post-contrast imaging, most authors have reported heterogeneous avid enhancement with variability in the extent of enhancement in the calcified versus noncalcified components [14]. Similar to other reports, our lesions showed avid and heterogeneous enhancement that appears to involve both the calcified and non-calcified components.

Prognosis and treatment:

Prognosis in MCS remains guarded. Historically, 5- and 10-year survival rates were 54.6% and 27.3%, respectively [18]. More recent data from a large multicenter series demonstrate

improved outcomes, with 5- and 10-year survival rates of approximately 70% and 54%, respectively [4]. Pulmonary metastases and local recurrence are common, and long-term survival continues to decline over time [18]. Case reports highlight this variability: a thigh primary metastasizing to the pancreas demonstrated the tumor's unpredictable spread and poor outcome despite multimodal therapy [2]. In contrast, an intracranial case remained disease-free for five years following complete excision [8]. Collectively, these findings underscore both the aggressiveness and heterogeneity of MCS outcomes, emphasizing the need for long-term surveillance and systemic staging at diagnosis.

There are no standardized treatment guidelines due to the rarity of MCS. Nonetheless, wide surgical excision with negative margins is consistently emphasized as the cornerstone of therapy and the factor most strongly associated with improved survival [5]. The benefits of chemotherapy and radiotherapy remain uncertain; some studies suggest a role in controlling residual disease or recurrence, whereas others report minimal impact on overall outcomes. Consequently, a multimodal approach combining surgery with adjuvant therapy is often considered, particularly in cases of incomplete resection or advanced disease [1,2,4,5,19].

Differential diagnosis:

Based on the imaging appearance, the differential diagnosis for a partially calcified soft-tissue mass with osseous involvement includes other primary cartilaginous tumors such as high-grade chondrosarcomas or juxtracortical chondrosarcoma, or conventional osteosarcoma and variants like chondroblastic osteosarcoma and small-cell osteosarcoma [17]. In cases of extraosseous mesenchymal chondrosarcoma, the differential diagnosis includes other primary soft-tissue neoplasms such as synovial sarcoma and, less commonly, liposarcoma, given overlapping imaging features and occasional calcification [5,6,15,16]. Evaluation of mineralization patterns can assist in narrowing the differential, as osteosarcomas typically demonstrate a 'cloudlike' or 'fluffy' osteoid matrix [17]. When present, the distinct pattern of well-demarcated transitions between calcified and non-calcified components in MCS described by some authors may also be helpful [6,14]. MRI features may aid in distinguishing MCS from conventional chondrosarcoma, as MCS typically demonstrates moderate to high signal intensity on T2-weighted imaging, which is lower than that observed in most chondroid neoplasms [15]. Enhancement patterns can also assist in distinguishing MCS from conventional chondrosarcoma, as MCS typically demonstrates heterogeneous enhancement and lacks the characteristic cartilaginous septal and peripheral enhancement. Additional distinguishing features include the predilection of MCS for the axial skeleton and its occurrence in younger patients. In comparison to extraosseous MCS, liposarcoma variants, especially those with a myxoid component, typically demonstrate regions with more uniformly high T2 signal that do not enhance as avidly after contrast injection [6]. Nonetheless, there is significant overlap in imaging features between these lesions, making correlation with histopathology and clinical findings crucial for establishing the correct diagnosis [5,6].

TEACHING POINT

Mesenchymal chondrosarcoma is a rare, aggressive chondrosarcoma affecting younger patients, characterized on imaging by a large (>10 cm) lobulated mass with biphasic calcified/ossified and soft-tissue components, moderate T2 hyperintensity, and absence of classic chondroid enhancement, and pathologically by high-grade small round blue cells admixed with well-differentiated hyaline cartilage. Accurate diagnosis is challenging due to overlap with other small round blue cell tumors on limited biopsy and therefore requires careful imaging–pathology correlation, molecular confirmation such as HEY1::NCOA2 fusion testing, and a multidisciplinary approach to management and surveillance.

QUESTIONS

Question 1: Which imaging features are characteristic of mesenchymal chondrosarcoma?

1. Variable calcification and extraosseous extension (applies)
2. Well-circumscribed, non-enhancing fat-density mass
3. Purely cystic lesion with fluid–fluid levels
4. Cloudlike/fluffy osteoid matrix
5. "Ground-glass" opacification with well-defined sclerotic borders

Explanation: MCS commonly appears as a lobulated mass with mixed mineralization; CT best delineates calcifications, including ring-and-arc matrix. It shows heterogeneous avid enhancement, lacking the classic chondroid septal/peripheral pattern.

Question 2: Which histopathologic/molecular features are typical of mesenchymal chondrosarcoma?

1. Presence of the HEY1::NCOA2 fusion (applies)
2. Cartilage component negative for S100 and SOX9 on immunohistochemistry
3. EWSR1::FLI1 fusion as the defining alteration
4. Diffuse cytoplasmic ALK protein expression
5. Presence of the BCOR-CCNB3 or CIC-DUX4 fusion

Explanation: Characterized by biphasic histology with undifferentiated small round or spindle cells adjacent to hyaline cartilage, which typically expresses S100 and SOX9; many cases show the HEY1::NCOA2 gene fusion specific to this tumor.

Question 3: Which imaging modalities are most useful for diagnosis and planning in MCS?

1. Ultrasound as the primary diagnostic test
2. Non-contrast MRI only is sufficient for diagnosis
3. CT to define mineralization patterns and help target biopsy and MRI with contrast to assess marrow and enhancement pattern (applies)
4. Plain radiography alone is sufficient for diagnosis and operative planning

5. Radionuclide bone scan to differentiate between MCS and conventional chondrosarcoma

Explanation: CT best depicts intratumoral calcifications and can reveal sharp transitions in mineralization that aid biopsy planning. MRI (with contrast) characterizes marrow involvement and the heterogeneous, non-septal enhancement typical of MCS.

Question 4: What differential diagnoses commonly overlap with MCS on imaging?

1. Giant cell tumor
2. Synovial sarcoma (applies)
3. Osteoid osteoma
4. Nonossifying fibroma
5. Osteochondroma with a thin, stable cartilaginous cap

Explanation: Partially calcified soft-tissue masses with bone involvement suggest differentials such as high-grade or juxtacortical chondrosarcoma, osteosarcoma variants, and synovial sarcoma. Mineralization, enhancement, age, and MRI features help narrow the diagnosis.

Question 5: Which treatment/prognosis statements are correct for MCS?

1. Pulmonary metastases and local recurrence are uncommon; therefore, long-term surveillance is less critical
2. Wide surgical excision with negative margins is the cornerstone and correlates with improved survival (applies)
3. Systemic staging at diagnosis is unnecessary
4. Recent reports of 5- and 10-year survival are ~30% and 15%
5. Five-year disease-free survival is considered a definitive cure

Explanation: Because MCS is rare, guidelines are limited, but wide excision with negative margins is consistently emphasized; the role of chemo/radiation is variable/uncertain.

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FIGURES

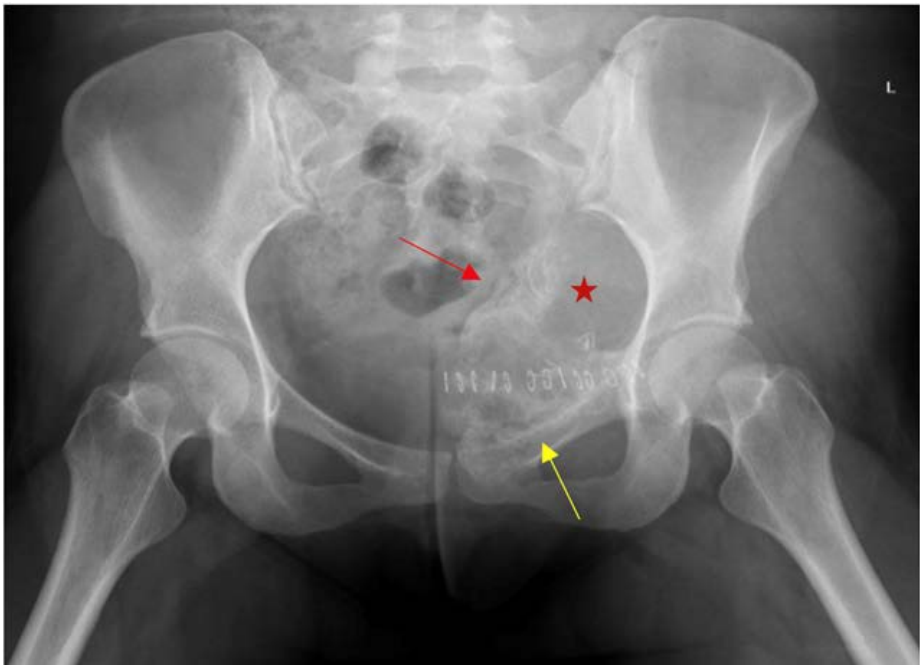

Figure 1: 26-year-old female with a mass arising from the superior border of the left superior pubic ramus.

Findings: Frog-leg lateral pelvic X-ray shows a large partially calcified mass arising from the superior border of the left superior pubic ramus (yellow arrow) with associated cortical thickening and periosteal reaction. The lesion demonstrated predominantly peripheral areas of calcification (red arrow) with a distinct central non-calcified component (red star)..

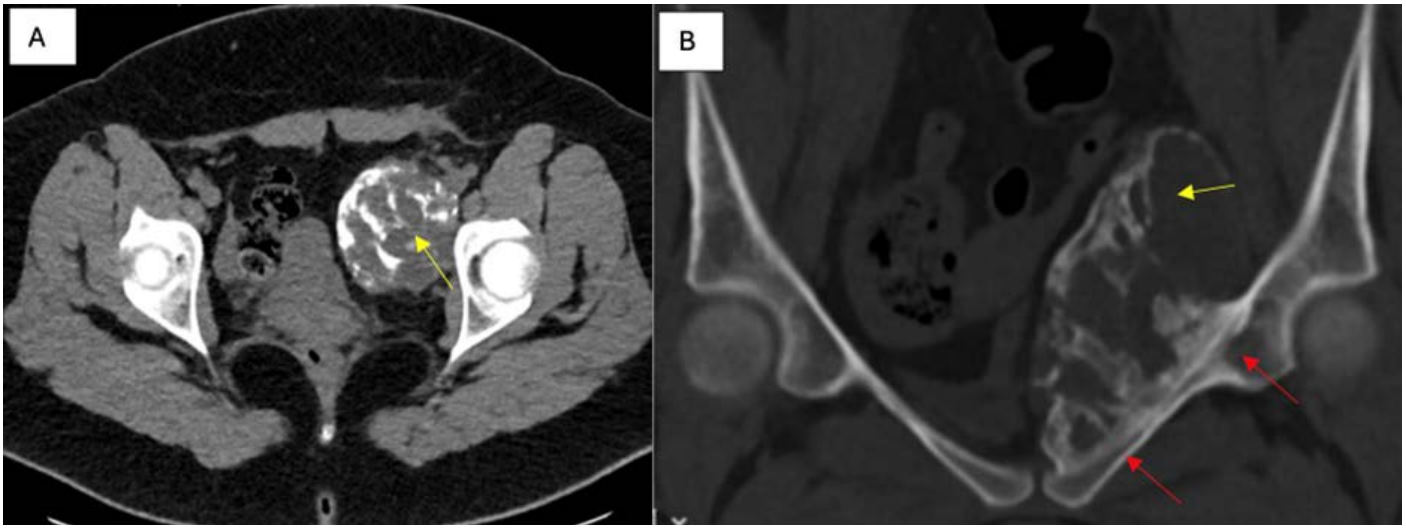

Figure 2: 26-year-old female with partially calcified left pelvic mass

Findings: CT pelvis without IV contras**t.** Axial soft tissue window (A) and Coronal bone windows (B) demonstrate a large lobulated partially calcified soft mass. Ring and arc type calcifications are noted centrally (yellow arrows, A). Dense calcification along the periphery of the mass and sharp transition between calcified and noncalcified components (yellow arrows, B). Cortical thickening and periosteal reaction are also well visualized with CT (red arrows, B)

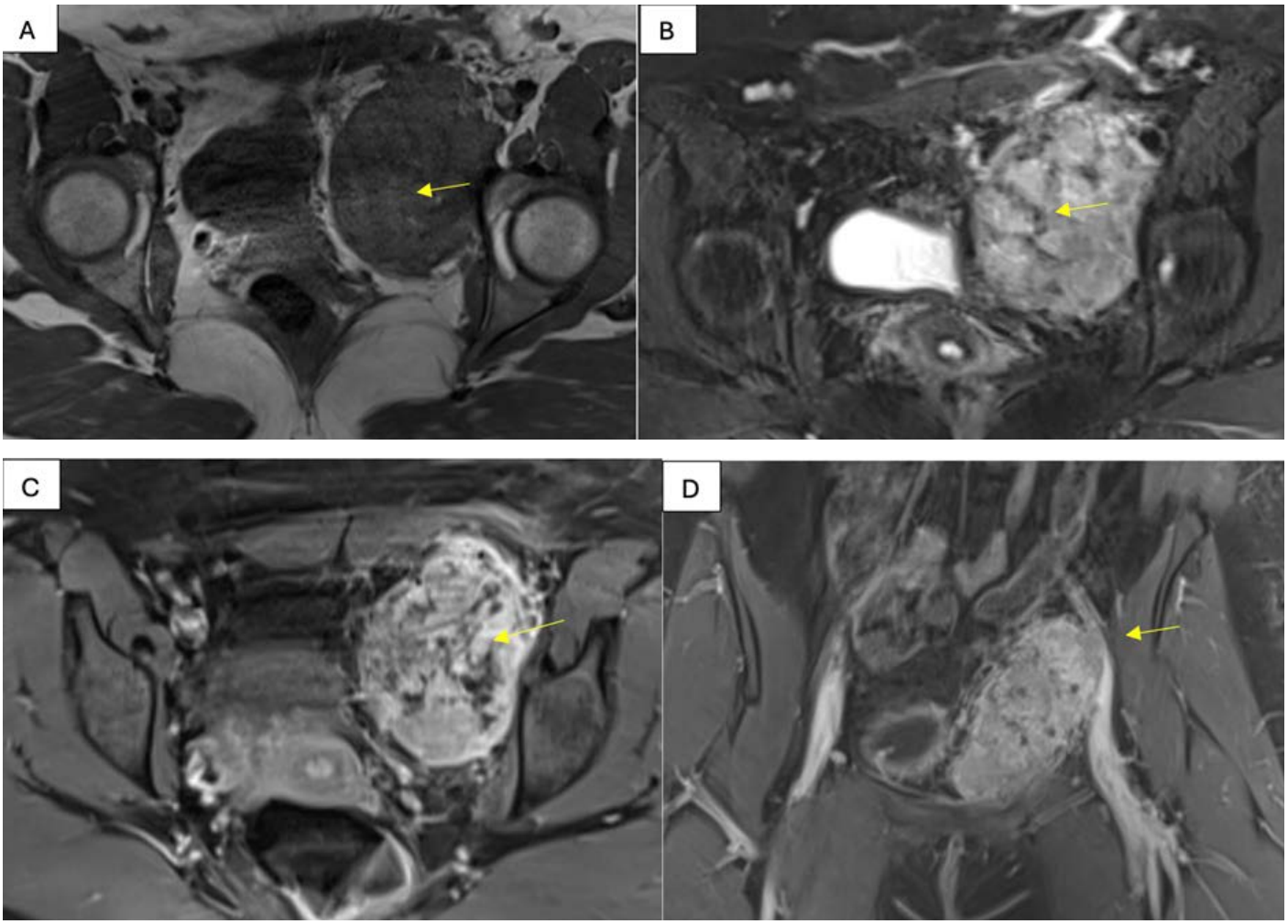

Figure 3: 26-year-old female with left pelvic mass compressing the external iliac vein.

Findings: MRI pelvis with and without contrast. Axial T1-weighted (A), Axial T2-weighted fat-suppressed (B), Axial (C), and Coronal (D) contrast-enhanced T1-weighted fat-suppressed images demonstrate a large lobulated left pelvic mass centered in the extraperitoneal space. Mass is predominantly T1 isointense and T2 hyperintense. Punctate foci of hypointense signals corresponding to calcifications (yellow arrow A,B). This appearance on T2-weighted images has been described as the "black pepper" sign. There is heterogeneous enhancement corresponding to calcified and noncalcified components (yellow arrows, C) and compression of the external iliac vein by the mass (yellow arrow, D).

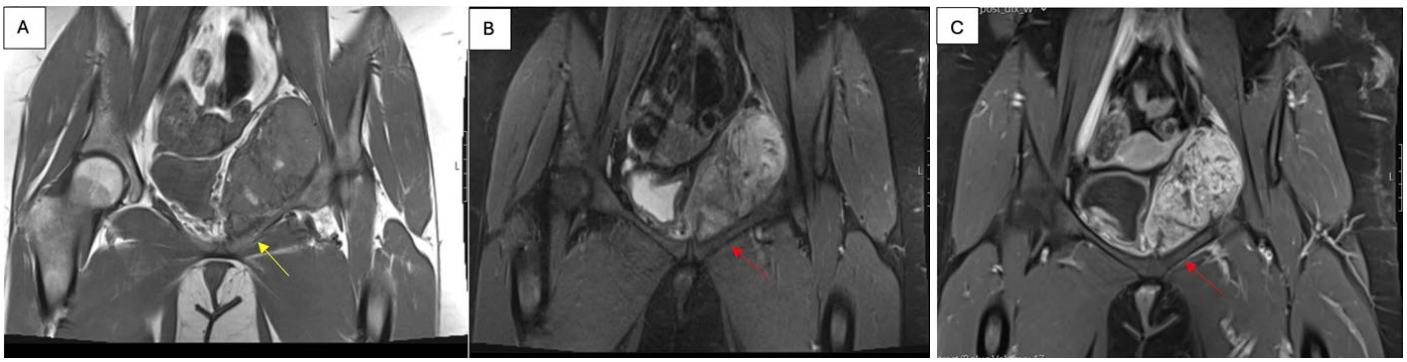

Figure 4: Juxtra-cortical pelvic mass arising from the superior border of the left superior pubic ramus without medullary extension.

Findings: MRI pelvis with and without contrast. Coronal T1-weighted (A), T2-weighted fat-suppressed (B), and T1-weighted fat-suppressed contrast-enhanced (C) images demonstrate a large heterogeneous mass arising from the superior pubic ramus. There is cortical thickening (yellow arrows, A). Normal bone marrow signal is noted (red arrows B, C), confirming the periosteal origin of the lesion.

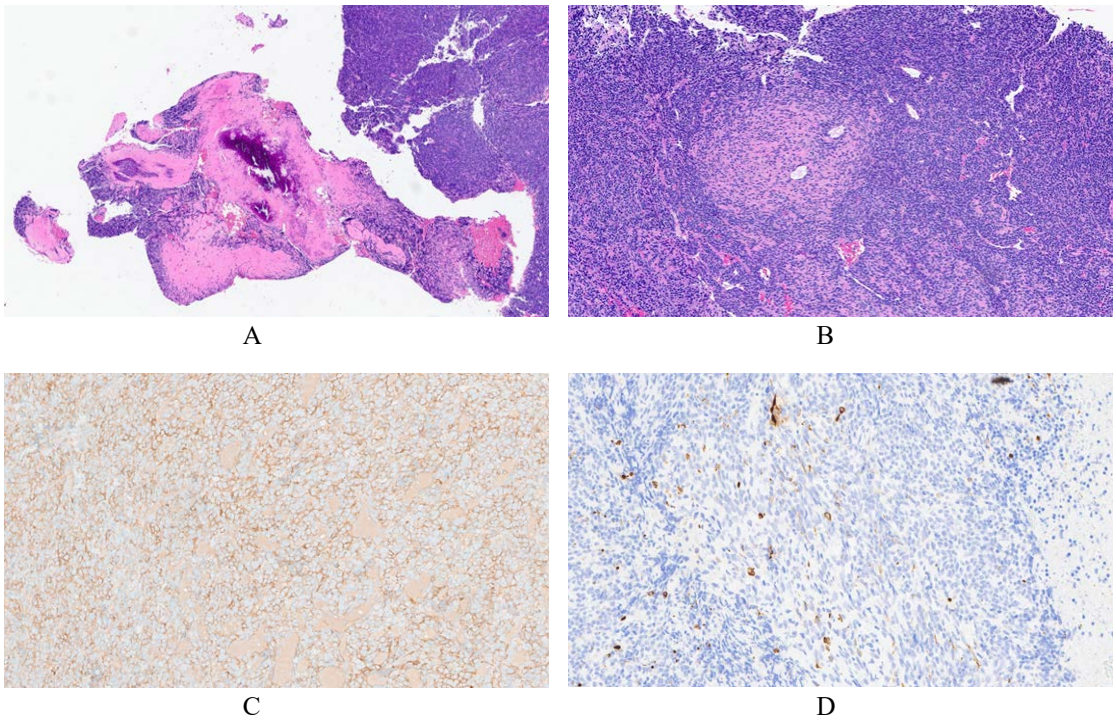

Figure 5: Biopsy left pelvic mass. **(A)** Hematoxylin and eosin stain, 40x magnification showing a hypercellular small round blue cell neoplasm with islands of calcified hyaline cartilage. **(B)** Hematoxylin and eosin stain 100x magnification showing staghorn-like vasculature with small osteoid-like islands. **(C)** CD99 immunohistochemical stain, 200x magnification. Immunohistochemical stains performed show membranous CD99 expression. (**D)** Desmin immunohistochemical stain, 200x magnification, which shows scattered rare desmin-positive cells.

KEYWORDS

Mesenchymal chondrosarcoma; Chondrosarcoma; Biphasic bone tumors; Ring-and-arc calcification; Magnetic resonance imaging; Computed tomography

ABBREVIATIONS

MCS = Mesenchymal Chondro Sarcoma
MRI = Magnetic Resonance Imaging
CT = Computed Tomography
DVT = Deep Vein Thrombosis