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Case Report

Myositis Ossificans at the Left Deltoid Muscle. Case Report of Uncommon Lesion and a Brief Review of the Literature

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Abstract

Myositis ossificans (MO) is a benign condition of heterotopic ossification of soft tissues, typically secondary to trauma. The most common areas affected by MO are the thigh and upper arm. Involvement of the shoulder is uncommon. It may have clinical and radiological similarities with other benign and malignant soft tissue tumors, thus making diagnosis problematic. We report a case of a 29-year-old healthy athletic male who had been experiencing a painful enlarging mass of the left shoulder over the past three months. There was no recent history of trauma; however, the patient reported a previous history of left shoulder dislocation managed conservatively with no complications. Imaging demonstrated a well-defined heterogeneous calcified intramuscular mass with peripheral ossification. A multidisciplinary tumor board advised a tissue biopsy. Histologically, the typical zonal pattern of MO with peripheral mature bone and central immature osteoid was observed, without cytologic atypia. En bloc surgical excision was conducted because of increasing pain, the large size of the lesion, and the lesion's maturity. There were no postoperative complications with an uneventful postoperative course, and 4 years after follow-up, there was no recurrence. This case illustrates the importance of integrating clinical, radiological, and pathological findings in diagnosing MO, particularly at an unusual site, as in our case, the shoulder. It is important to recognize its characteristic features to avoid misinterpretation and unwarranted aggressive management with the diagnosis of malignancy.

Keywords: Myositis ossificans, Heterotopic ossification, Benign, Zonal pattern, Nontraumatic

Introduction

Myositis ossificans (MO) is a benign, non-neoplastic entity representing an abnormal heterotrophic ossified process in which bone is formed outside the skeleton, especially in the muscles [1]. First described in the 18th century, it remains a diagnostic dilemma because the clinical and radiological findings can resemble malignant tumors, particularly in their early or atypical forms [2,3]. MO is typically divided into three subtypes: traumatic myositis ossificans (the most frequent), non-traumatic (idiopathic) myositis ossificans, and the inherited myositis ossificans progressiva, better known as fibrodysplasia ossificans progressive (FOP) [4]. The importance of MO is that it may be misdiagnosed as other benign or malignant entities with unnecessary aggressive management. This is even more relevant for uncommon locations, such as the shoulder, where most soft tissue masses are typically considered to be of malignancy [1]. Although MO is not rare and has been well described in the literature, large, symptomatic lesions in the deltoid, as was seen in the present case, are relatively rare.

This case provides a rare chance to review the conventional knowledge about MO. It highlights the importance of a combined diagnostic workflow focusing on imaging, histopathology, and clinical correlation in differentiating this benign entity from its malignant counterparts. It also enhances the existing literature on shoulder-related MO and emphasizes the need to identify this condition when it presents outside of typical anatomical locations.

CASE PRESENTATION

A 29-year-old male athlete was admitted with a mass in the left shoulder that had been growing progressively and became increasingly painful three months before the admission. The pain was first self-treated with over-the-counter analgesics, but recently had become unbearable and required medical assessment. The patient reported no recent trauma to the left shoulder, but he remembered a dislocation of the left shoulder two years before, managed conservatively with no complications. He had a past medical history notable for well-regulated type 1 diabetes mellitus and an appendectomy in the past. There was no significant medical family history. Physical examination revealed a solid, tender mass, 8 × 5 cm in diameter, in the left shoulder are. The mass was hard and palpable, with much difficulty in moving it.

Plain radiography demonstrated the presence of a heterogeneously calcified soft tissue mass with peripheral mineralization. Chest and upper arm CT revealed one well-demarcated intramuscular mass in the deltoid region, which showed heterogeneous enhancement and a peripheral rim of calcification, but no periosteal reaction around the adjacent humerus. An MRI revealed a heterogeneously enhancing lesion with T2 hyperintense, T1 hypointense, and focally isointense foci. The muscles exhibited

edema and no lymphadenopathy. On imaging, a broad differential was entertained, including both benign and malignant processes: posttraumatic hematoma, soft tissue sarcoma, bursitis, calcific tendonitis, infection (abscess), osteochondroma, myositis ossificans (MO), extraskeletal osteosarcoma, and chondrosarcoma. The case was discussed in a multidisciplinary tumor board, with MO being the favored diagnosis, but a tissue biopsy was recommended for definitive diagnosis before local therapy could be implemented.

A tissue biopsy was carried out. On histology, a characteristic zonal architecture was observed with mature lamellar bone at the periphery showing decreased cellularity and central immature osteoid and fibrous tissue. Focal cystic changes and bone remodeling with hematopoietic marrow were occasionally recognized. Although there was increased cellular proliferation, the lesion showed no striking pleomorphism, nuclear atypia, or abnormal mitotic activity. These features were typical of late myositis ossificans. Histologically, the differential diagnosis included nodular fasciitis, fibrous dysplasia, heterotopic ossification, and extraskeletal osteosarcoma. MO's lone characteristic feature that helped differentiate between the two was a well-organized zonal pattern. In contrast, sarcomas usually do not show this pattern and often are characterized by significant cytologic atypia and brisk mitotic activity. Immunohistochemical stains, including SATB2, CD34, MDM2,CDK4 and Ki-67, favored the benignity of the lesion. No molecular testing was performed based on the typical imaging and histology findings.

Due to the lesion's large size, advanced maturation, and worsening pain, likely due to impingement on nearby neural structures, surgical intervention was undertaken. An en bloc resection was performed with clear margins, avoiding piecemeal excision. Pathology examination of the excised lesion confirmed complete excision of a mature myositis ossificans lesion with clear margins more than one cm. Post-operatively, the patient was enrolled in a physical therapy program of range of motion, muscle strengthening, and functional progression. Recovery was smooth and provided no recurrence at the four-year follow-up. This good result emphasizes the value of well-designed surgical planning, accurate diagnosis, and a soundly considered timing of surgery.

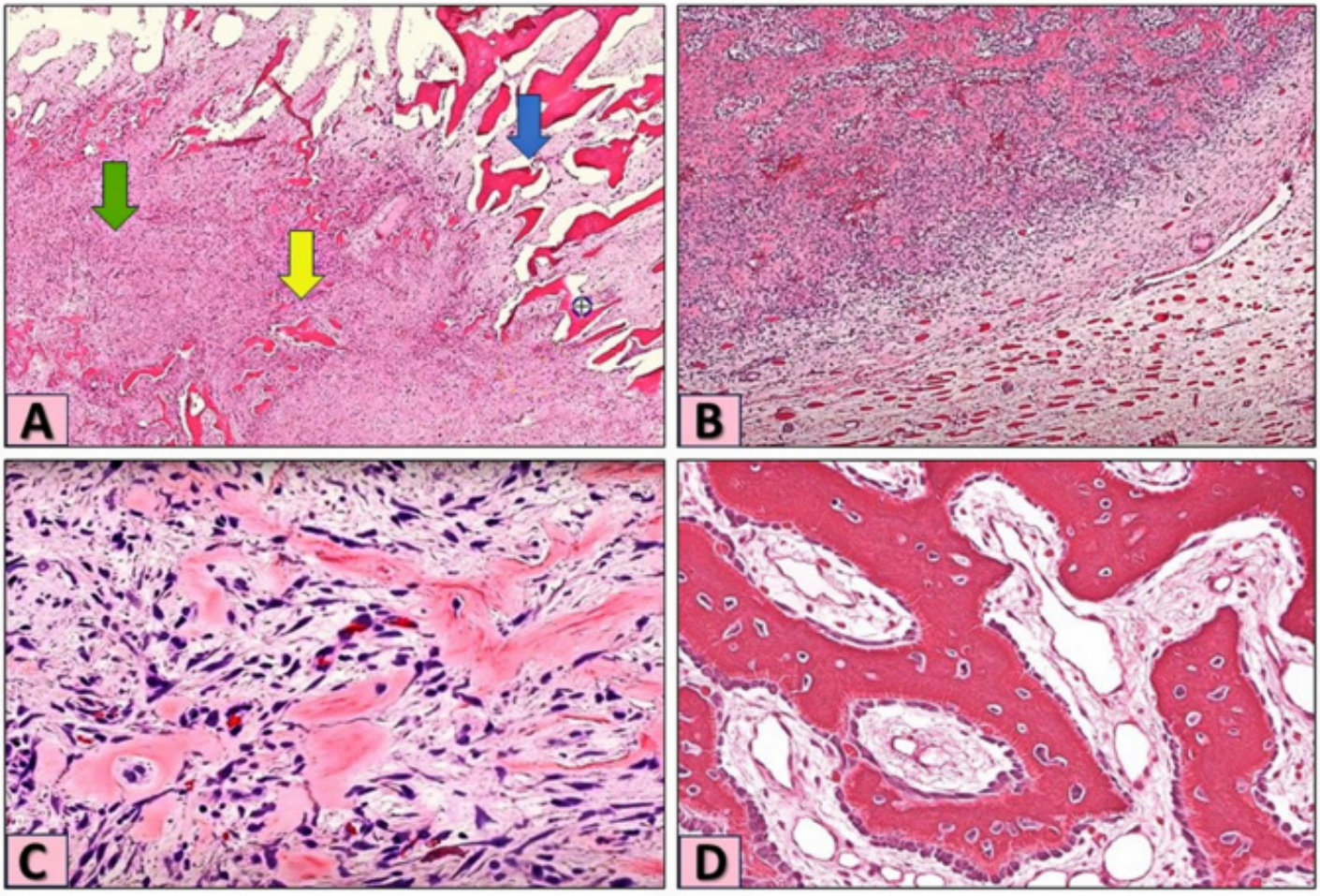

Figure 1: Histomorphologic features of the excised myositis ossificans
1A Low power view showing characteristic zonal architecture, mature lamellar bone at the periphery (Blue arrow) followed by immature osteoid (Yellow Arrow) and central cellular fibrous tissue (Green arrow)(H&E stain X20)
1B: Intermediate power view showing myositis ossificans (upper left) within the deltoid muscle (Lower right) (H&E stain X20)
1C: High power view showing immature osteoid and fibrous tissue (H&E stain X40)
1D: High power view showing mature bone trabeculae (H&E stain X60)

DISCUSSION

History, epidemiology, and risk factors: Myositis ossificans (MO) has been described for over 2 centuries, and its first detailed accounts date back to the 18th and 19th centuries. The term was initially used as a generic descriptor for soft tissue ossification [2,3] MO is now defined as a non-neoplastic, reactive phenomenon characterized by the formation of heterotopic bone within a muscle or soft tissue [1]. The term "myositis ossificans traumatica" was later coined to distinguish it from its genetic/persistent counterparts, most notably fibrodysplasia ossificans progressiva [5]. Epidemiologically, MO occurs more frequently in young adolescents and adults, especially in the second and third decades of life. Males are more commonly involved than females, probably due to more exercise practice and traumatic risks. It is most common in active or athletic people but can also affect non-athletes [6].

Our patient is a 29-year-old athletic male with no definite memory of experiencing recent trauma, but in whom a remote history of shoulder dislocation was discovered, who fits the common demographic profile of MO, despite an atypical location. His case illustrates the importance of including MO in the differential diagnosis of a soft tissue mass in a young adult, even without a history of acute trauma, especially in an athletic individual who is previously injured.

Clinical/radiographic presentation, and differential diagnosis: MO usually manifests as a painful, enlarging soft tissue mass, developing days to weeks following trauma. The clinical findings can be deceptive in the early disease, particularly when the lesion is large, rapidly enlarging, firm, and tender, features commonly suggestive of an aggressive or malignant lesion [7]. X-ray findings usually show soft tissue mass with a zonal ossification pattern, i.e., dense peripherally and lucent centrally [8]. CT affords superior anatomical detail, demonstrating peripheral ossification and excluding direct bone involvement or cortical destruction, and the latter would be at odds with a typical MO [8]. MRI findings can be deceptive, especially in lesions of the early stage. Typical appearances include T2 hyperintensity, peritumoral edema, and heterogeneous enhancement. These are nonspecific and may appear similar to soft tissue sarcomas, especially extraskeletal osteosarcoma [9]. Hence, histological correlation is necessary. MRI can also detect accompanying muscle edema and assist in determining proximity to neurovascular structures [10].

The differential of MO is wide-ranging and includes benign and malignant entities. The benign differentials include posttraumatic/organized hematoma, calcific tendinitis, bursitis, nodular fasciitis, and heterotopic ossification (non-neoplastic) [11]. Malignant mimics include extraskeletal osteosarcoma, parosteal or periosteal osteosarcoma, extraskeletal chondrosarcoma, and soft tissue sarcoma with calcified matrix (e.g., synovial sarcoma, liposarcoma) [12,13]. The lack of periosteal reaction and bone destruction can exclude MO from the malignant osseous tumors. Also, the zonal pattern of ossification—central cellularity with a peripheral plane of maturation—is a characteristic that inclines the diagnosis of MO versus a malignancy, in which the calcification is often central and amorphous [14,15].

In our case, the atypical site of the lesion in the shoulder and the lack of significant trauma had made the diagnosis uncertain. Imaging findings, particularly a peripheral rim of calcification seen on CT and MRI, generated concern for neoplastic causes. A multidisciplinary tumor board astutely recommended a biopsy for histologic confirmation before definitive therapy. This case emphasizes the necessity of correlating clinical, radiologic, and pathologic findings in the approach to such cases.

Pathology and immunohistochemistry findings: Histopathological study is the best tool to confirm the diagnosis of myositis ossificans (MO), especially when there is an atypical presentation or equivocal imaging [15]. The histomorphologic signature of MO is the well-defined zonal pattern, and it reflects the maturing process of the lesion; it can be used to distinguish it from malignant neoplasia [14]. Its zonal architecture consists of three concentric layers- a central, more cellular zone of osteoid and immature bone formation, and a peripheral rim of mature lamellar bone [14,15]. The histology demonstrated classic mature MO lesion features. No pleomorphism, abnormal mitoses, or infiltrative growth effect would have been found to suggest malignancy. Immunohistochemistry (IHC) is an adjunct tool that can be helpful, most notably in ruling out sarcomas or other mimics. In MO, the proliferative spindle cells are usually vimentin-positive and occasionally positive for smooth muscle actin (SMA), indicative of their myofibroblastic nature. However, this finding is nonspecific and needs context to be interpreted [16]. On the other hand, high-grade sarcomas may express MDM2, CDK4, and high Ki-67 proliferation index (e.g., dedifferentiated liposarcomas) or non-S100, non-desmin, or non-keratin in certain types of sarcomas [16]. In our case, negative immunostaining for MDM2 and CDK4, low Ki-67, and the absence of sarcomatous markers favored a benign reactive process. The final diagnosis was myositis ossificans.

Management, prognosis, and outcomes: Myositis ossificans (MO) treatment is mainly determined by the lesion stage, symptom intensity, functional limitation, and diagnostic confidence. In most such cases (especially those with a known history of trauma and characteristic radiological findings), MO is self-limited and can be treated conservatively [17]. In most cases, simple observation, rest, nonsteroidal anti-inflammatory agents, and physical therapy will suffice as the lesion becomes mature and stable in a spontaneous process that typically takes 6–12 months [17]. Surgery may be offered to some individuals in whom the lesion is painful, compressing the adjacent structures (e.g., the nerves or joints), is function-altering, or cannot definitively exclude malignancy [18]. Surgery should not be performed until the lesion is mature because premature resection could result in incomplete removal, recurrence, and potential for pathological incorrect diagnosis [19].

In our case, and even though our patient did not recall any specific traumatic event, the lesion increased in size over three months, causing increasing pain, most probably secondary to nerve compression or mechanical pressure. Imaging did not rule out a malignant process, and while biopsy showed it to be benign, continued symptoms and lesion size justified surgical excision. Complete en bloc excision was achieved with a soft tissue cuff to reduce the chances of recurrence. Crucially, piecemeal or partial removal is discouraged, resulting in refractory symptoms and recurrence.

Since it is adequately diagnosed and treated, the prognosis of MO is good. Recurrence is few and far between after total excision of a mature lesion. MO is non-malignant. However, extended follow-up may be required in cases that occur in unusual sites, have incomplete resection, or have residual functional limitation [20]. Adjuvant treatments such as bisphosphonates, low-dose irradiation, or NSAIDs have been investigated for prophylaxis against heterotopic ossification, especially following orthopedic interventions or in spinal cord-injured subjects. However, their use in the control of spontaneous or posttraumatic MO is limited without universal endorsement in a potentially select subgroup of patients [6].

Pathophysiology and pathogenesis: The pathophysiology of MO is the heterotopic bone formation in the soft tissues, typically the skeletal muscle, as a response to injury or repetitive mechanical stress. It is ectopic bone formation, i.e., bone formed outside the usual skeleton. [14,15]. It is different from other forms of calcification in how it becomes structured and progressively organized into mature lamellar bone. Pathogenesis is the key to diagnosis and differentiation between it and malignant tumors, as well as the manner of treatment. MO is based on a disturbed tissue re-

pair mechanism. The inciting event, usually trauma, results in necrosis of muscle fibers and a hematoma with inflammation [21]. In the absence of obvious trauma, such as in our case, subclinical microtrauma or repeated mechanical stress in athletes may elicit this same response. This site-specific insult results in a cytokine cascade and cell recruitment events that closely resemble the events of fracture healing, particularly endochondral ossification, bone formation from a cartilage template [1].

MO is not a neoplasm. While it may appear aggressive histologically, hypercellular, and mitotically active in its early phase, it does not exhibit genomic instability, atypia, or the invasiveness of true malignancies [6]. With molecular analyses of MO, it is demonstrated that BMP signaling, pro-inflammatory cytokines, and mesenchymal stem cell differentiation contribute to the cascade of ectopic bone formation [1]. Although not invariably a result of genetic mutations (as in fibrodysplasia ossificans progressive), knowledge of these pathways helps differentiate MO from other malignant lesions, and insights may be applied to therapeutic development [6]. This underlines the important role of pathology in the proper clinical and radiologic context. MO pathogenesis involves local injury, inflammatory signaling, and osteogenic differentiation. The transition from soft tissue injury to solid bone formation is a tightly controlled, but abnormal form of the physiological process of bone healing. Knowledge of these mechanisms leads to better diagnostic potential and can also point toward therapeutic strategies that target ectopic bone formation in certain clinical settings.

Lessons learned from this case: This case illustrates several important clinical and educational points, particularly in diagnosing and managing atypical MO presentations.MO is generally regarded as a benign, self-limiting entity that is commonly due to trauma. However, occurrence of MO in an atypical location,without clear trauma history or with atypical imaging findings, may represent a significant diagnostic challenge, such as that in our patient. A valuable teaching point learned from this case is that having a wide differential diagnosis when confronted with a rapidly growing soft tissue mass is essential. Our patient's lesion, in the setting of no identifiable trauma and concerning radiologic findings raised the initial suspicion for soft tissue sarcoma or other malignancy.

Another vital aspect is the diagnostic significance of histopathologic examination. The characteristic zonal architecture in mature MO remains the most helpful feature in distinguishing it from malignant mimics, such as extraskeletal osteosarcoma.This case further supports the importance of the pathologist being aware of the chronological progression of MO and not overcalling MO based on mitotic activity or cellularity alone, as in early lesions. We also demonstrated that the clinical history and imaging findings must be considered thoughtfully. The patient was also an athlete and had no recollection of injury, and his history of remote shoulder dislocation did not suggest association with the lesion. This is a reminder that microtrauma and overuse, especially in the active form, are potential inciting events and should not be underestimated when developing a clinical story.In addition, the case highlights the importance of early and judicious surgical intervention. After the diagnosis of MO had been established and the lesion had shown radiologic maturity, en bloc excision was associated with excellent relief of symptoms and long-term control. This is particularly so for surgical intervention, which should only be undertaken with a clear indication (pain, function restriction, or diagnostic doubt) and not before the immature phase of the lesion.

Lastly, the present case enriches the scarce literature on MO involving the shoulder, a rare location, and expands the diversity of its clinical presentation. Evidence of such presentations may assist our improvement of diagnostic pathways and prevent unwarranted anxiety or unnecessary concern when seeing benign yet complex lesions such as MO.

Conclusion

This case highlights the value of combined multidisciplinary management, a high index of suspicion for benign malignant-appearing lesions, the diagnostic yield of histopathology, and the benefits of personalized patient care. These fundamentals are widely applicable to MO and to assessing soft tissue masses in general.

Statements of compliance with ethical standards

Human subjects and ethics statement

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.

Conflicts of interest

All authors declare the following.

Payment/services information

All authors have declared that they received no financial support from any organization for the submitted work.

Financial relationships

All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Other relationships

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

Data access statement

All relevant data are included in the paper.

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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