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Research Article

CONVERSION OF MYOSITIS OSSIFICANS TRAUMATICA TO OSTEOSARCOMA

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A B S T R A C T

Myositis ossificansusually has a benign coursewithout major complications, but on rare occasions itmay transform into an osteosarcoma. Our patient first presented with leg pain and was diagnosed with myositis ossificans radiologically. She was pursued up by serial imaging for six years during which her lesion was stable. A month before this presentation, she noticed a small mass around the knee which rapidly grew into a massive protruding lesion. Imaging done now raised suspicion of malignant lesion which was confirmed pathologically. This prompts a need to closely follow a patient with myositis ossificans radiologically and be aggressive in managing it whenever there is a suspicion of malignancy.

INTRODUCTION

Myositis ossificans (MO) refers to aberrant ossification of muscles, ligaments and tendons. It possesses a relatively indolent course with no life threatening events. (Wei *et al.*, 2015). We report an unusual case of myositis ossificans transformed into a chondroblastic osteosarcoma after a stable course. Very few cases of such transformation are reported to our knowledge (Konishi *et al.*, 2001; Wheeler *et al.*, 2014).

Case

A 67-year-oldAfricanAmerican lady with no significant medical history presents with a mass around herleft knee which she first noticed three months ago. It began as a small 'knot' and rapidly advanced to a large protruding mass (32cmX 13cm).Patient did not report any previous chronic ulcer or infections at the site of the mass. Around fourteen years ago, she was in a motor vehicle accident (MVA) and sustained crushing injury in same leg; seven years later, she presented with left leg pain. X-ray done at that point showed abnormal periosteal new bone formation about the proximal fibular metaphysis and head with a very high suspicion for periosteal or paraosteal sarcoma. Further management was planned, but the patient did not follow up as her pain improved with analgesics.

One year afterwards, she came back with the same complaint. During that presentation, a computerized tomography (CT) and nuclear medicine scan (NM) was done.

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Bone scans showed increased uptake in soft tissues around the proximal tibia without the involvement of shaft of the left tibia. CT of the leg done on the same day also showed a large region of calcification within the soft tissues about proximal tibial and fibular shafts without evidence of intramedullary calcification or periosteal reaction or change in the size of the calcific mass, this was felt to represent heterotopic calcification, such as myositis ossificans rather than osteosarcoma. Follow up CT scans done over the next two years showed similar heterotrophic ossification process, butwith a small increase in size and without signs of malignancy. An NM bone scanthree months before this admission showed stable myositis ossificans (Fig 1).

During this admission, her labs were normal except elevated ALP. A CT of the left leg showed significant interval extension of abnormal soft tissue calcification within the proximal to mid left leg with the interval cortical destruction of the mid fibula shaft, findings which are highly suspicious for interval development of malignant transformation, such as osteosarcoma. Bone scans done now showed marked uptake in the area described aboveas shown in Fig 2. Excisional biopsy of the lesion was done and sent for microscopic examination, which showed several islands of atypical hyaline cartilage with areas of surrounding osteoid, all within a fibrotic stroma (Fig 3).

As clinical and radiological features were highly suggestive of malignancy, the patient was referredto anorthopedic oncologist who performed an above the knee amputation and sent the specimen for pathological evaluation which showed high grade chondroblastic osteosarcoma (pT2, pNX, pMX). CPM gene

amplification was not detected. She receivedfour cycles of Cisplatin and Adriamycin.

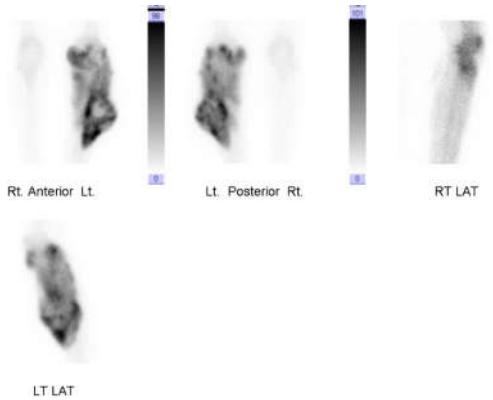

Fig 1 NM scan showing increased uptake in the area of osteosarcoma

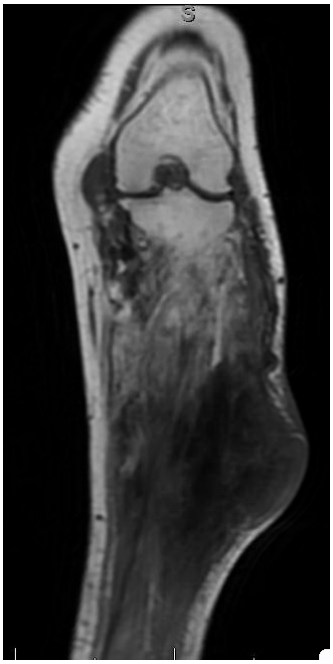

Fig 2 Abnormal calcification with destruction of shaft of the fibula by osteoscarcoma

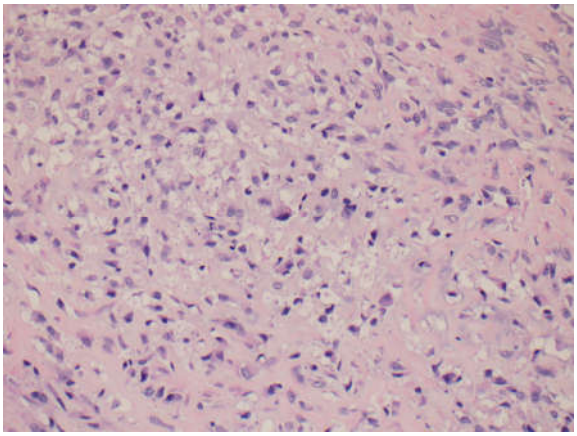

Fig 3 High power view of malignant osteoid production

DISCUSSION

MO can be classified based on etiology. Most common is traumatic MO, where injury/inflammation triggers ossification of tissues. Fibrodysplasia ossificans progressiva refers to a congenital condition where ossification can occur at multiple sites. Neurogenic MO refers to ossification of joints after spinal cord injury(Kruse *et al.*, 2009). We believe that our case is a traumatic type given her history of injury to the same leg in MVA.

The mechanism behind ossification tissues is unclear, but some studies done in animal models suggested constitutional activation of bone morphogenic protein (BMP) type 1 may be responsible for it at least in genetic types (Yu *et al.*, 2008). Another study showed injuries to muscle will trigger chondrocytes/osteoblast recruitment which carries out ossification of tissues (Aho *et al.*, 1988). Malignant transformation of MO might occur is one of the above processes goes unregulated.

Patients with MO present with classical symptoms like pain, tenderness, and swelling in the region of involved muscles. Any acute increase in pain/tenderness or rapid growth of mass associated with constitutional symptoms like weight loss or loss of appetite along with rising ALP along with other inflammatory markers should raise suspicion of malignant transformation (Goldman, 1976; Schultzel *et al.*, 2015).

Diagnosis of osteosarcoma in patients with a history of MO needs correlation of clinical, radiological as well as pathological features to avoid misdiagnoses (Nishio *et al.*, 2010; El Bardouni *et al.*, 2014). It is easy to appreciate these changes if we have done serial imaging with CT or MRI as in this case. Aggressive features like cortical bone destruction, incomplete translucent zone in periosteal lesion and absence of zone phenomenon differentiates osteosarcoma from MO (Goldman, 1976). On pathological examination, proliferating, immature fibroblasts with hyperchromatic nuclei and ill-defined borders differentiates it from MO which show zoning phenomenon (peripheral osteoid with a central layer of cells) (Ragunanthan e Sugavanam, 2006).

Management does not differ from primary osteosarcoma. The first step would be the biopsy of the lesion to confirm diagnosis followed by complete excision of the tumor with disease free margins. As in our case, if suspicion of malignancy is very high, we can go ahead even with surgery if the biopsy results are negative. Systemic chemotherapy with cisplatin and doxorubicin with or without ifosfomide is given to the patient on a case-by-case basis (Ruiz-Godoy *et al.*, 1999; Almeida *et al.*, 2014).

CONCLUSION

Scheduled, cautious monitoring of myositis ossificans for radiographic changes (like a cortical bone invasion) and aggressive management by excision biopsy/early resection in patients with worrisome clinical features, like a sudden increase in the size of the lesion, will aid in the identification of such malignant transformations, and ultimately be lifesaving.

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