CASE REPORT

Myositis ossificans of the quadriceps femoris in a soccer player

Jose Pedro Marques,[1] João Páscoa Pinheiro,[2] Joana Santos Costa,[3] Diogo Moura[4]

[1]Department of Sports Medicine, Coimbra University Hospital, Coimbra, Portugal
[2]Faculty of Medicine, University of Coimbra, Coimbra, Portugal
[3]Department of Physical Rehabilitation Medicine, Coimbra University Hospital, Coimbra, Portugal
[4]Department of Orthopedics, Coimbra University Hospital, Coimbra, Portugal

Correspondence to
Dr Jose Marques,
joseppmarques@gmail.com

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SUMMARY

A young soccer player was diagnosed with myositis ossificans 6 weeks after a muscle strain in the right thigh. Radiographic and sonographic investigations initially helped to confirm diagnosis and later supported clinical improvement. We present our approach to the case and discuss pathophysiology, prevention and treatment of this rare condition.

BACKGROUND

Myositis ossificans (MO) is a rare disorder characterised by non-neoplastic heterotopic bone formation in soft tissue and skeletal muscle.[1] Three types can be differentiated: MO *progressiva* (hereditary and severe generalised form), MO without history of trauma (associated with burns, haemophilia and neurological disorders) and MO *circumscripta* or *traumatica* (either related to a direct blow or repeated minor trauma).[1] The latter is the most common, representing 60–75% of all cases.[2] Although it can be seen at any age, the highest incidence is reported in adolescents and young adults.[1] MO traumatica (MOT) mainly affects extremities, most often the anterior thigh and arm.[3] The clinical presentation encompasses a painful soft tissue mass usually with no inflammatory signs. The diagnosis of this condition may be difficult and requires radiological and/or histological findings. To accurately interpret these, one must be familiar with its aetiopathogenesis—MOT is essentially a proliferative mesenchymal response to an initiating injury to the soft tissue that leads to localised ossification.[3] In the first week after injury, proliferative fibroblastic cells are prominent. As maturation proceeds, a typical zonal pattern develops with three distinct zones: the centre, characterised by rapid proliferating fibroblasts; the intermediate zone, consisting of osteoblasts with immature osteoid formation and the periphery, composed of mature bone well separated from the surrounding tissue by myxoid fibrous tissue. Around the third to fourth week, calcifications appear inside the mass. By the sixth to eighth week, a well-organised cortical bone with cortex and marrow space can be seen at the periphery. The maturation process proceeds so that, by 6 months, the bone is indistinguishable from the normal skeleton.[4 5] As a self-limited condition, in most cases, conservative treatment with clinical and radiological follow-up is usually enough.[3] Surgical excision is an exceptional option when function is impaired or the mass is unusually large or painful.[6]

We present a case of a soccer player who developed MOT following a quadriceps femoris strain and an early return to practice. To the best of our knowledge, this is one of the few case reports of MOT secondary to an indirect muscle injury. We aim to emphasise the importance of recognising significant muscle injuries and to adjust rehabilitation and return-to-play schemes according to the extent of the lesion.

CASE PRESENTATION

A 25-year-old Caucasian man with anterior thigh pain was referred to the clinic. The patient recalled a muscle strain 6 weeks before, when sprinting to get to the ball at the end of a soccer match. He had to be substituted due to intense pain and spent the two following weeks being treated by the team's physiotherapist. Treatment consisted mostly of cryotherapy and massage. He resumed training but after 1 week had to stop due to increased pain, and a decrease in range of motion (ROM) and function of his right leg. The patient was an amateur soccer player with 10 h/week spent between training and competition. He denied having suffered a contusion to his right thigh in the previous weeks and also denied having any relevant personal or family history, constitutional symptoms or drug use.

Physical examination at the clinic revealed a right thigh perimeter 2 cm larger than the contralateral. He had limited ROM of his right knee, achieving no more than 90° flexion with preserved extension. Tenderness along the medial third of the thigh along with pain on passive stretching, and on static and dynamic quadriceps contraction, was evident. The patient had no inflammatory signs and neurovascular examination was unremarkable.

INVESTIGATIONS

Radiographs (figures 1 and 2) showed new bone adjacent to the proximal 2/3 of the right femur. A bedside ultrasound examination (figure 3) revealed a hyperechoic reflection of the calcified surface with posterior attenuation located at the *rectus femoris* myotendinous junction. Biarticular muscles (such as this) are commonly injured at this location in strain injuries. An MRI was performed in order to exclude non-benign causes of a soft tissue mass. It identified a mass located in the proximal 1/3 of *rectus femoris* muscle with characteristics compatible with MOT.

TREATMENT

The patient was instructed to use crutches, with partial weight-bearing allowed for 10 days. Static

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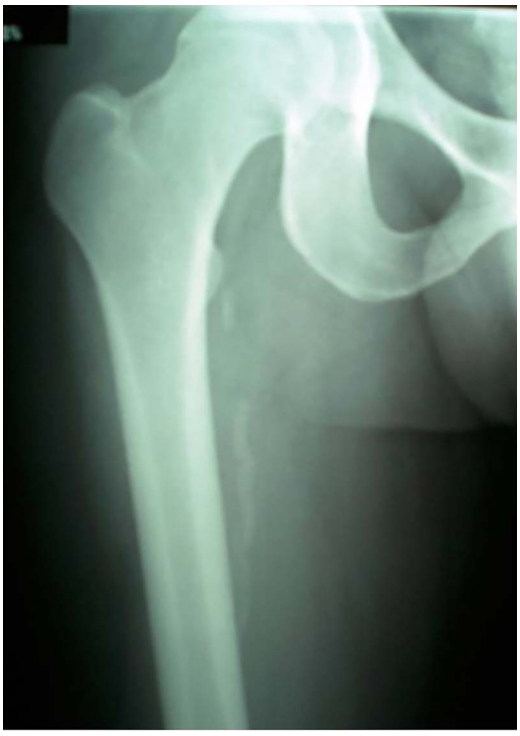

Figure 1 Calcifications along the medial border of the proximal femur (anteroposterior view).

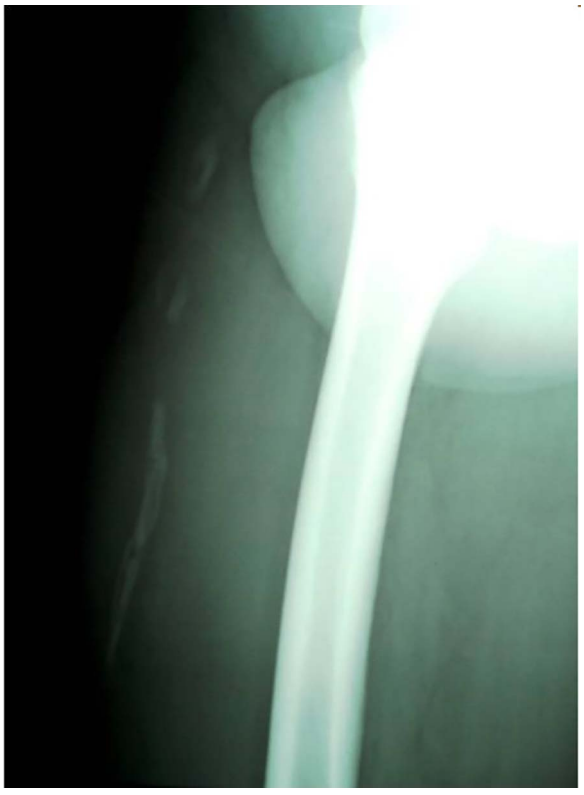

Figure 2 Calcifications along the medial border of the proximal femur (lateral view).

cryotherapy was performed and he received a prescription for etoricoxib 60 (1 pill/day for 2 weeks) and an elastic bandage. A rehabilitation programme was initiated, which in the first 4 weeks included quadriceps work (static contraction 0–30°; 25 and 45 Hz electrical stimulation), static stretching, intermittent ultrasound (1.5 Hz for 10 min) and manual therapy. Over the following 4 weeks, the (now asymptomatic) patient started closed kinetic chain muscular strengthening, proprioceptive training, deep transverse massage and aerobic reconditioning with cycloergometer (55% maximum heart rate; HRmax). Protein supplementation with branched-chain amino acids (BCAA; 1 g/kg) was started during this period. Between the 8th and 12th week, eccentric work of quadriceps and hamstrings was added alongside jogging and some on-field activities. From the 12th week on, the patient was allowed to begin sport-specific training, and by the 24th week, he started competing without restrictions.

OUTCOME AND FOLLOW-UP

Follow-up after 40 weeks revealed an asymptomatic patient. Ultrasound and X-ray showed marked decrease in the number and volume of calcifications.

DISCUSSION

Four pre-requisites (previous conditions) seem to be necessary for MOT formation: an initial traumatic injury, the secretion of a protein by injured tissue or inflammatory cells, a reservoir of not fully committed mesenchymal cells and a favourable environment to continued heterotopic bone deposition.[2]

Most case reports identify contusion as the initial injury. To the best of our knowledge, this report is one of the few where strain was the inciting event.[7–9] The relevance of this feature remains unknown and might not be considerable since the following event (intramuscular haematoma formation) is already a common step in the cascade of events.[10 11] The patient restarted training only 2 weeks after muscle injury. Premature return to activity is referred to by King[11] as a main risk factor for MOT development. He relies on the work developed with West Point cadets, where the incidence of MOT following thigh contusions decreased from 20% (in 1973) to 9% (in 1991).[11] A possible explanation could be an improvement in acute phase healthcare and optimisation of recovery periods. Prescription of non-steroidal anti-inflammatory drugs (NSAIDs) is often regarded as a prevention strategy for MOT. In fact, NSAIDs have shown some efficacy in MO prevention following spinal cord injury and joint replacement,[12] leading to an extrapolation of this benefit to MOT cases. Nonetheless, 'state of the art' treatment of muscle injuries no longer includes NSAIDs because the COX-2 pathway

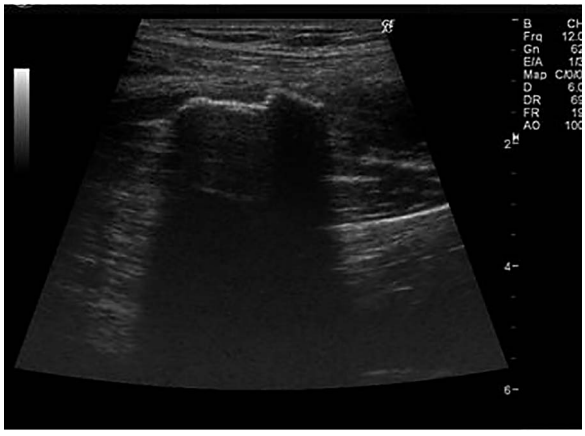

Figure 3 Ultrasound showing hyperechoic reflection of the calcified surface with posterior attenuation.

is seen as essential during early stages of skeletal muscle regeneration.[13] Hence, when weighing the ability to prevent the rare MOT and reduce pain in short-term against long-term deficit in force production and muscle repair, we tend to choose the latter.[14] In the future, a better understanding of risk factors for MOT's may allow differentiation of a subgroup in which the overall risk of developing this condition is high enough to deserve NSAIDs prophylaxis. Research focused on finding other risk factors has led to inconclusive/equivocal results.[12]

MOT diagnosis must be considered in the context of a muscle injury, especially when the symptoms intensify 2–3 weeks after the trauma. Additional clinical suspicion is warranted if the area becomes more indurated and loss of ROM is observed. Imaging studies differ in their sensitivity and specificity according to the phase of the maturation process.[15] While ultrasound is able to detect the early soft tissue changes of MO, conventional radiography and CT confirm the existence of heterotopic bone formation and may display specific patterns of MOT.[15] MRI is the technique of choice to evaluate soft tissue lesions, which makes it useful in the differential diagnosis with neoplasms.[15] The differential diagnosis of MOT includes malignant tumours such as lymphoma, osteosarcoma and rhabdomyosarcoma.[12] In this patient, 6 weeks after the inciting injury, ultrasound and X-ray revealed findings compatible with MOT. He denied constitutional symptoms (chills, fever or weight loss) and his laboratory tests (white cell count; erythrocyte sedimentation rate and C reactive protein; alkaline phosphatase levels; calcium levels; creatine kinase levels) were all within normal values. MRI confirmed our strong clinical suspicion that this was an MOT case. Radiography and ultrasound repeated at the 40th week showed marked improvement. The time to radiological and clinical resolution is in line with some of the well-resulting case reports found in the literature.[16] In complicated cases, symptoms and radiological findings may last more than a year. Whether this difference is due to treatment or to local/systemic factors remains unknown. Biopsy was not performed, as it is only required in atypical cases, in young patients with no previous trauma or when imaging studies are inconclusive.[2] Radiation therapy can be used to reduce size and accelerate the lesion's maturation, especially when planning surgery.[17] Given the favourable evolution, we found it unnecessary. Bisphosfonate therapy is another option often mentioned in the literature; we, however, did not explore it. Its inhibitory effect on bone formation is limited to the crystallisation process, leaving bone matrix formation unaffected. This means that, after cessation of treatment, the matrix resumes uninhibited mineralisation. It therefore follows that an early diagnosis is of utmost importance and treatment with bisphosfonate has to be continued for at least 6 months.[12] Surgery was not advised since there was no significant pain or functional limitation. The decision to give protein supplements was made according to recent evidence suggesting that exercise combined with BCAA improves muscle repair and growth in athletes.[18]

Treatment of MOT in the acute phase is mainly conservative, designed to control pain and inflammation, and later to stimulate flexibility, strength and fitness status.

Learning points

- Myositis ossificans is rarely reported after strain injuries.
- Optimisation of time to return to practice and rehabilitation programmes might be the key for prevention of myositis ossificans.
- A soccer player was able to return to competition after 24 weeks of conservative treatment.
- Ultrasound evidence of calcifications persists long after clinical resolution.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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