Skeletal Radiol (2004) 33:237–240
DOI 10.1007/s00256-003-0715-0

CASE REPORT

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# Proliferative myositis in a patient with AIDS

Received: 24 June 2003
Revised: 29 September 2003
Accepted: 1 October 2003
Published online: 4 December 2003


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**Abstract** We report a case of proliferative myositis in the right biceps of a 56-year-old man with acquired immune deficiency syndrome (AIDS). Imaging methods included sonography, computed tomography and magnetic resonance imaging. The diagnosis was made by a core-cut biopsy and fine needle aspiration biopsy with immunohistochemical analysis. The lesion disappeared after 2 months without treatment. It is particularly important to determine whether intramuscular masses arising in patients with AIDS are due to an infectious or malignant process.

**Keywords** Proliferative myositis · AIDS · US · CT · MRI

## Introduction

Proliferative myositis (PM) is a rare intramuscular pseudosarcomatous inflammatory process of unknown cause, first described by Kern in 1960 [1]. It usually manifests as a very rapidly growing soft-tissue mass in an adult patient (median age at onset 50 years); the mass is often painful, and can be confused with a malignant tumor. Biopsy and histological analysis are required for proper diagnosis.

We report a case of PM in a patient with acquired immune deficiency syndrome (AIDS). The type of mass was determined after biopsy, and disappeared without treatment.

## Case report

A 56-year-old man with AIDS (history of *Pneumocystis carinii* pneumonia) who was receiving highly active antiretroviral therapy (zidovudine, lamivudine and nelfinavir) presented 4 days after noticing a swelling in his right arm. There was no history of recent trauma, inflammation or cancer.

Physical examination revealed a tender but painless mass in the lower third of the right biceps. There was no local hyperthermia, and no change in the color or structure of the overlying skin. Extension of the right arm was limited to 160°. The temperature was normal. Axillary nodes were palpable bilaterally.

The white blood cell count was $3.9\times10^9$/l and the red cell count $3.82\times10^9$/l. The hemoglobin level, platelet count and clotting tests were normal. The CD4 cell count was 296/mm$^2$. HIV viral load was below 50 copies/ml. The total creatinine kinase was 214 mU/ml (normal range 10–180 mU/ml) and the alkaline phosphatase was 132 mU/ml (normal range 30–120 mU/ml).

The right arm was examined with sonography, CT and MRI. Ultrasound showed an inhomogeneous, expansive mass in the right biceps. The mass was hyperechoic, with distinct contours, a mildly lobulated structure and anarchic central vascularization. There was

**Fig. 1** Sagittal T2-weighted MR of the right arm (3500/128), showing a hyperintense lesion in the biceps muscle

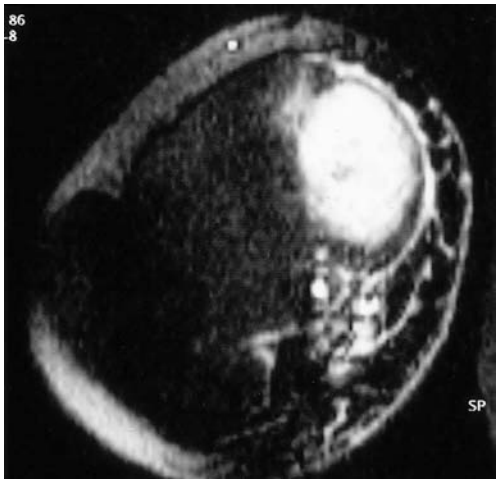


**Fig. 2** Axial STIR MR (3915/29) images showing the high signal intensity of this intramuscular lesion

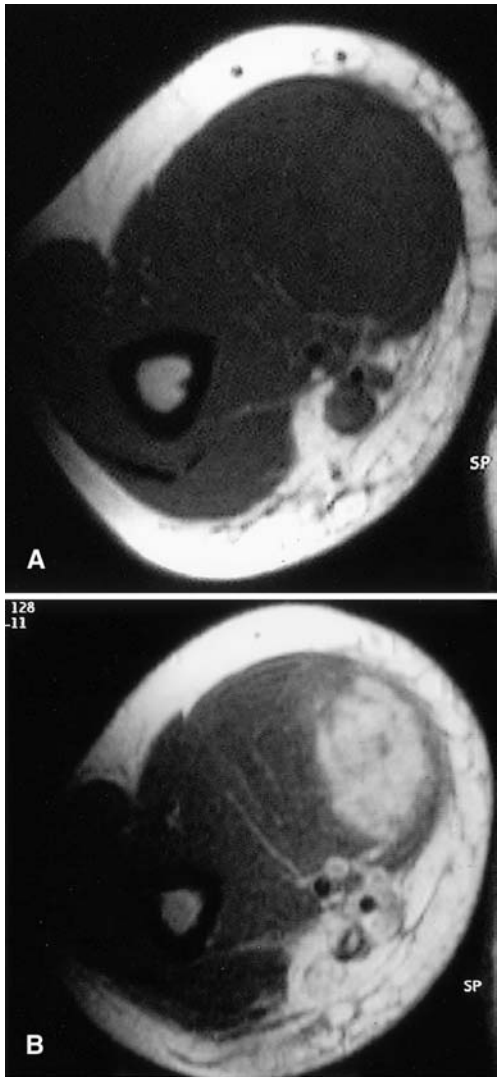


**Fig. 3** **A** T1-weighted axial MR image (644/12) obtained at the same level, showing a muscle-isointense lesion. **B** Note the homogeneous enhancement after intravenous gadolinium

no apparent infiltration of surrounding muscles. Sarcoma or lymphoma was suspected. MRI studies and biopsy were prescribed.

T2-weighted (Fig. 1) and STIR (Fig. 2) MR images showed a hyperintense lesion 7 cm in diameter in the right biceps, with an isointense signal on T1-weighted images (Fig. 3A) and homogeneous enhancement after intravenous gadolinium injection (Fig. 3B).

CT scans without intravenous contrast injection showed a faint, mildly hypodense lesion. The lesion was isodense with muscle after contrast enhancement . Core-cut biopsy and fine needle aspiration biopsy (FNAB) of the lesion were performed under sonographic control.

Histological examination of the specimens showed skeletal muscle components, fibroblast proliferation and a myxoid base; muscle fibers persisted in the bundles. A group of ganglion-like giant cells with large nuclei and basophilic cytoplasm was seen. Some mitoses were noted (Fig. 4).

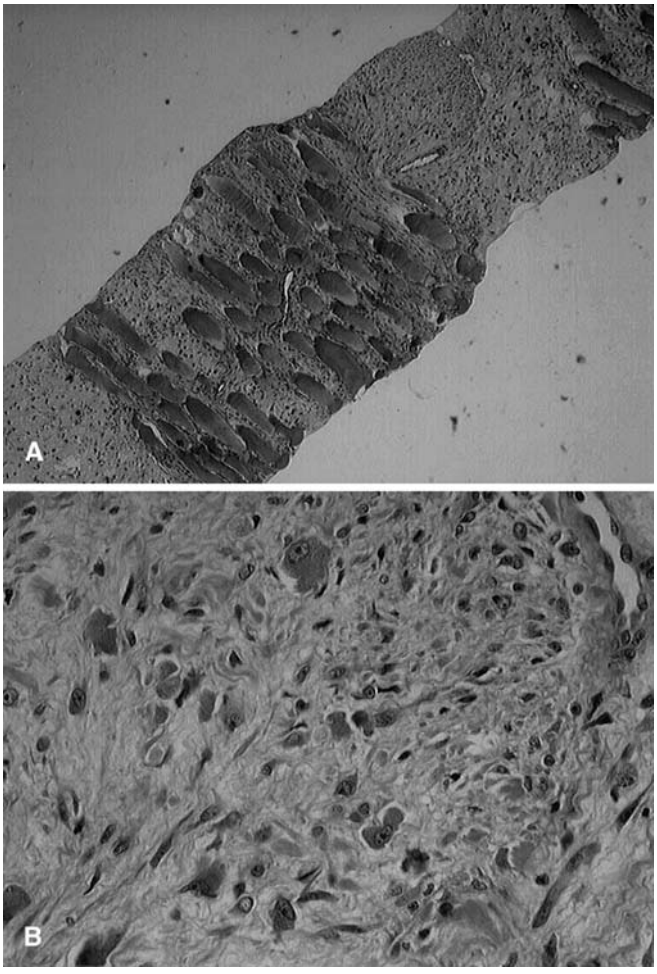


**Fig. 4** **A** Low-power view of proliferative myositis (×10). The skeletal muscle bundles are separated by the proliferation of connective tissue from endomysium and epimysium. The connective tissue is arranged in a checkerboard-like fashion. **B** High-power view of proliferative myositis (×40). Detail of characteristic large basophilic giant cells (modified fibroblasts) resembling ganglion cells

Immunohistochemical analysis showed that the ganglion-like cells stained positively with anti-vimentin and anti-smooth-muscle-cell antibodies, and negatively with anti-cytokeratin AE1-AE3 antibodies.

Benign proliferative myositis was diagnosed. No medical treatment was prescribed.

Two months later the mass was no longer palpable. The temperature was normal and the patient remained pain-free. Total creatine kinase and alkaline phosphatase levels were normal. Ultrasound showed a small hyperechoic zone of 25 mm in the bicipital muscle, with discrete local vascularization. The rest of the bicipital muscle had a normal appearance. The MRI examination was normal (not shown), both before and after intravenous gadolinium injection.

## Discussion

Proliferative myositis (PM) is a rare inflammatory myopathy. Clinically, it presents as a very rapidly growing solitary soft-tissue mass, with or without pain [1, 2, 3, 4]. The etiology is unknown, but there is often a history of local trauma [1, 2]. Average age at onset is 50 years [2, 3, 4]. PM usually arises in the trunk region or upper extremities [3, 4].

The rapid growth, compatible with a malignant process, often previously led to radical surgery. However, once the benign, inflammatory nature of PM had been established by Kern [1], the standard therapy became marginal excision.

Recently, incisional biopsy, or FNAB, followed by a watch-and-wait policy was recommended, as PM lesions disappeared spontaneously in all reported patients [2] without specific treatment.

Radiological findings are nonspecific. Sonography usually shows an inhomogeneous mass, with or without calcifications [2].

CT usually shows a poorly demarcated lesion that is hypodense or isodense relative to the surrounding muscle tissue. Contrast enhancement may be homogeneous, heterogeneous or absent [2, 3].

MRI findings have only been reported in four cases including ours. MRI generally shows hypointensity on T1-weighted images and hyperintensity on T2-weighted images [5]. Contrast studies have been reported in two cases (including ours), and showed enhancement of the lesion in both cases [2]. Recently, T2-weighted fat-suppression sequences were recommended for better visualization of signal intensity changes [6].

A firm diagnosis can only be established by biopsy and immunohistochemical analysis. Microscopic examination of biopsy specimens reveals three different features [4]: (a) a peripheral zone in which the muscle fibers are generally unaltered and the perimysium and endomysium are infiltrated by spindle-shaped cells; (b) an intermediate zone in which the muscle fibers are dissociated and giant ganglion-cell-like cells and spindle-shaped cells are present, the giant cells typically with a basophilic cytoplasm and large nucleus; (c) a central zone containing giant cells and collagenous fibers replacing muscle tissue. Immunohistochemically, the giant cells stain positive for vimentin and smooth-muscle actin [2, 4].

Differential diagnoses include infectious myositis, myositis ossificans, trauma, muscle denervation, rhabdomyolysis, polymyositis, dermatomyositis and soft-tissue malignancies (especially sarcomas and lymphomas). Most of these diagnoses can be ruled out by clinical and laboratory findings [7, 8]. Myositis ossificans is also an inflammatory myopathy. It presents as soft-tissue swelling with gradual ossification. There is frequently a history of local trauma, burns or immobilization due to traumatic paralysis or coma. During the acute phase, the area that

may later ossify is swollen and painful. The peripheral ossification typically develops within 6–8 weeks and is readily identified on serial radiographs; MRI typically shows a low-signal-intensity rim [9].

Accurate diagnosis of rapidly growing masses in patients with AIDS is vital, as delayed or inappropriate treatment of an infectious or malignant process can lead to life-threatening complications. Soft-tissue lesions potentially associated with AIDS include infections (bacterial, viral or parasitic myositis), neoplasms such as non Hodgkin lymphoma, leiomyosarcoma and Kaposi sarcoma [10], polymyositis [4, 7], multiple myopathies including sarcoid myopathy [11], and neurogenic myopathy [12].

Pyomyositis (due to *Staphylococcus aureus* in 90% of cases [10]) can develop in several muscles, with abscess formation and, in most cases, low-grade fever and leukocytosis. Most lymphomas in AIDS patients are non-Hodgkin lymphomas. Bone lesions are most common, and can be accompanied by soft-tissue masses. Biopsy is necessary to establish the diagnosis [7, 8]. Leiomyosarcoma of the vascular subtype (attachment to and spread along the surface of a vessel wall) is associated with Epstein-Barr virus infection in young AIDS patients, and with organ transplantation [8]. In Kaposi sarcoma, MR images can show involvement of both muscle and subcutaneous tissue [10]. Diagnosis is made by muscle biopsy. Symmetric, bilateral proximal muscle weakness is the rule in polymyositis, along with elevated serum levels of creatinine kinase. Proximal muscle weakness with elevated serum levels of creatine kinase has also been reported in sarcoid myopathy [11] and neurogenic myopathy [12] in HIV-infected patient. Biopsy is necessary for the diagnosis. Toxoplasmosis can cause acute, painful myopathy in HIV-infected patients, and is confirmed by detection of *T. gondii* cysts in a muscle biopsy specimen [13]. Acquired mitochondrial myopathy has been linked to zidovudine therapy [14].

Thus, rapidly growing intramuscular masses in patients with AIDS may be due to proliferative myositis, and should be biopsied. Correct diagnosis of PM can avoid needless resection; these lesions healing spontaneously.

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