

Proliferative Myositis: A Case Report¹

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We report a case of proliferative myositis arising in the pectoralis major muscle of a 59-year-old man who presented with palpable mass. The initial clinical impression was a malignant tumor. Ultrasonography revealed the lesion as a spindle-shaped hypoechoic mass, and MR imaging of the left pectoralis major muscle showed hypointensity at T1-weighted imaging, hyperintensity at T2-weighted imaging, and strong enhancement at contrast-enhanced T1-weighted imaging.

Index words : Myositis

Muscles, US

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Proliferative myositis, a rare benign pseudosarcomatous lesion (1), usually appears as a rapidly growing mass in the proximal muscles of the extremities of adults. Clinically, it can be mistaken for a malignant tumor. Since it shows no tendency to recur, it is, however, probably self-limiting (2), and treatment by simple local excision is recommended. We present a case of proliferative myositis arising from the pectoralis muscle.

Case Report

A 59-year-old man presented with a hard, tender, fixed palpable mass in the superolateral aspect of the left anterior chest wall near the anterior axillar fold. There was no history of trauma to the chest. Four years earlier he had become aware of the presence of a small lump in the left anterior chest wall, and recent rapid growth had occurred. Clinically, a sarcomatous muscular neoplasm

was suspected.

For ultrasonographic examination, an HDI 3000 imager (ATL, Bothell, Wash., U.S.A.) with a wide-band small-part probe (frequency range, 5 to 10 MHz) was used. In the pectoralis major muscle, a relatively well-demarcated spindle-shaped hypoechoic mass, 3.3 × 2.8 × 1.4 cm in size and with roughly grouped muscle-like echogenicity and thick-lined echolucencies, was observed (Fig. 1). Power Doppler sonography depicted arterial vessels in the central portion of the mass, and the Doppler waveform showed a high resistive index.

For magnetic resonance (MR) imaging, a 1.5-T Magnetom Vision scanner (Siemens, Erlangen, Germany) was used. The images depicted a well defined mass in the left pectoralis major muscle. Low signal intensity with central iso-signal intensity to muscle was observed at T1WI, high signal intensity with central iso-signal intensity at T2WI, and strong enhancement with a central less enhanced portion at T1-weighted contrast-enhanced imaging (Fig. 2).

Wide excision of the lesion revealed, grossly, a poorly demarcated mass, 2.5 × 2.0 × 1.1 cm in size, located in the pectoralis muscle. Normal muscle tissue was separated by bands of pale gray scar-like induration, and light microscopic examination revealed that muscle

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fibers were separated by an excessive proliferation of fibroblast-like cells. At a higher magnification, the mass showed two main features; poorly-demarcated spindle cell proliferation and large basophilic ganglion-like cells that diffusely infiltrated skeletal muscle fiber while preserving individual fibers (Fig. 3). Immunohistochemically, both spindle and ganglion-like cells showed diffuse immunoreactivity in the presence of vimentin, but neither type of cell reacted to desmin, - smooth muscle actin, factor VIIIa, or myoglobin. Electron microscopy revealed that the large ganglion-like cells had abundant cytoplasm dominated by dilated and vacuolated cisternae of rough endoplasmic reticulum (Fig. 4). Abundant intermediate cytofilaments were present in the cytoplasm, suggesting myofibroblastic differentiation. Flow cytometric DNA analysis revealed a diploid

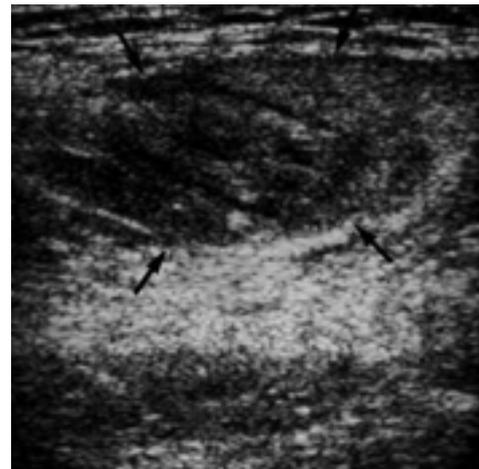


Fig. 1. Transverse sonogram shows a relatively well demarcated, ovoid mass in the left pectoralis muscle with central linear echolucencies resembling dry and cracked mud (arrows).

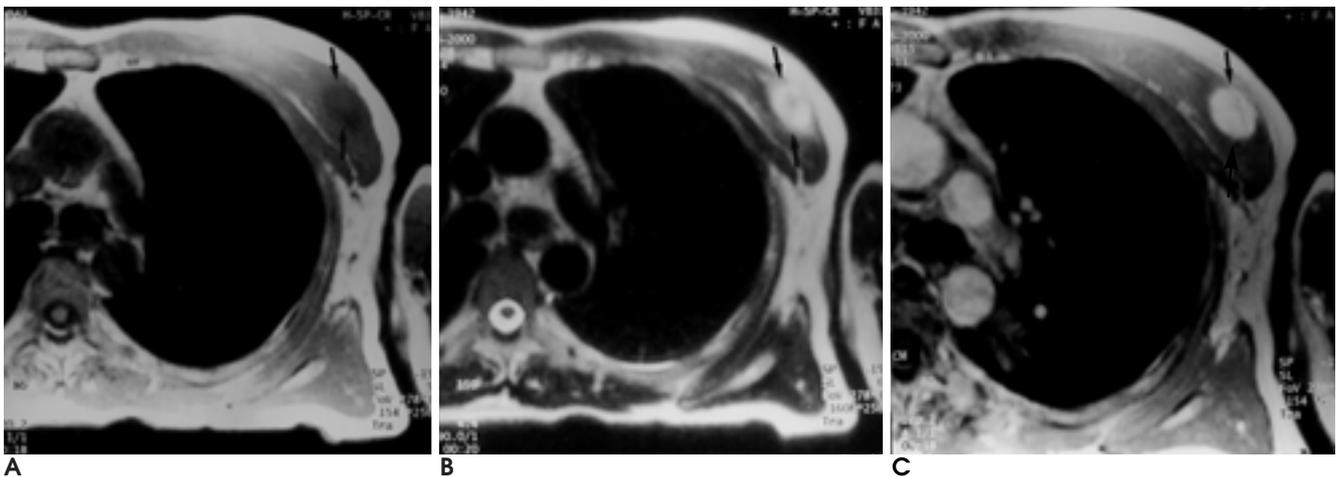


Fig. 2. A. Axial T1-weighted image shows an ovoid, target like hypointense mass in the left pectoralis major muscle (arrows).
B. Axial T2-weighted image shows target like hyperintensity with a tapered edge (arrows).
C. Axial contrast-enhanced T1-weighted image shows a strong enhancement with central less enhancement portion (arrows).

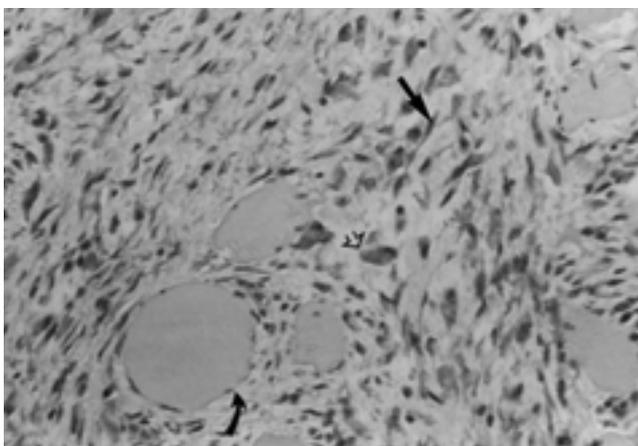


Fig. 3. Light microscopic examination reveals proliferation of the spindle cells (arrow) and ganglion like giant cells (open arrow) among the atrophic muscle fibers (curved arrow) (H & E, $\times 20$).



Fig. 4. Electron microscopic examination shows ganglion like cell with abundant cytoplasm dominated by dilated and vacuolated cisternae of rough endoplasmic reticulum (arrows).

pattern.

Discussion

Proliferative myositis, a non-neoplastic pseudosarcomatous proliferative lesion involving skeletal muscle and a self-limiting intramuscular pseudosarcomatous inflammatory process was first described by Kern in 1960. It appears as a rapidly growing mass that may or may not be painful. Common sites for proliferative myositis in adults are shoulder, chest wall and thigh (2), and those affected are, predominantly, aged 40 - 50 years. The etiology of proliferative myositis is unclear, but trauma and ischemia have been suggested as possible etiologic factors (2, 3).

Ultrasonography demonstrates roughly grouped muscle tissue delineated by a thick hypoechoic scaffolding which in transverse section resembles dry, cracked mud. Color Doppler sonography does not appear to add any significant information (4), and no characteristic CT pattern has been identified (3). MR imaging has demonstrated the presence of an ill defined mass that was hyperintense at T2WI and hypointense at T1WI, while Gd-enhanced T1-weighted imaging has revealed a contrast enhanced nodular lesion (5, 6). An inhomogeneous structure has occasionally, been observed, together with calcification that may be mistaken for malignancy or myositis ossificans (5).

Local excision is curative, and neither recurrence nor metastasis has been reported (1 - 6).

Grossly, proliferative myositis manifests as a poorly demarcated, gray-white scar-like lesion with diffusely infiltrated growth. Histopathologically, the typical diagnostic feature is ganglion-like cells that have abundant basophilic cytoplasm. At the periphery, the perimysium and endomysium are infiltrated by loose tissue consisting of elongated spindle shaped cells. In the intermediate area, giant ganglion-like cells are admixed with spindle shaped-cells. The central area of the lesion consists,

predominantly, of giant cells in a delicate network of collagenous fibers (5, 7). Immunohistochemically, the giant cells show immunoreactivity in the presence of vimentin, but do not react to desmin, -smooth muscle, actin or myoglobin. Rhabdomyosarcomas have an eosinophilic cytoplasm and react to desmin and myoglobin (7). In our case, the mass had a well defined margin, and the initial impression was benign soft tissue mass. Because sharp margination is not a reliable point of differentiation between a benign and malignant neoplasm, sarcoma could not, however, be ruled out (8). Probably because of the high cellularity of the central scar-like component, the target-like signal intensity seen at MR imaging varied.

Proliferative myositis is an uncommon benign condition occurring in adults. Correct identification of the condition is essential. In order to avoid misdiagnosis of a soft tissue tumor. When radiologic examination reveals a soft tissue mass, proliferative myositis should thus be included in the differential diagnosis.

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