Proliferative myositis presenting with a checkerboard-like pattern on CT

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Abstract

Proliferative myositis is a rare pseudosarcomatous inflammatory process. Radiological diagnosis of self-limiting proliferative myositis helps direct appropriate clinical management and avoiding unnecessary surgical excision. We present the ultrasonography, computed tomography, and magnetic resonance imaging findings in a case of proliferative myositis. In this case, malignancy was suspected, and complete excision was performed. A checkerboard-like pattern, a characteristic sonographic and pathological finding of proliferative myositis, was demonstrated by computed tomography in our patient; to the best of our knowledge, this is the first such case in the literature.

Key words: • magnetic resonance imaging • proliferative myositis • tomography, spiral computed • ultrasonography

Proliferative myositis is a rarely seen intramuscular inflammatory process. This pathology involves a fast-growing lesion showing diffuse infiltration to the muscular tissue; it is often misdiagnosed as a malignant sarcomatous tumor in clinical practice (1). In this article, ultrasonography (US), computed tomography (CT) and magnetic resonance imaging (MRI) findings in a case of proliferative myositis are presented. The patient underwent complete surgical excision to exclude malignancy. To the best of our knowledge, this is the first case of proliferative myositis with a checkerboard-like pattern diagnosed with the use of CT in the literature.

Case report

A 48-year-old male patient was admitted with a complaint of a painless, fast-growing swelling on the left side of the neck which occurred over several days, preceded by a sensation of slight itching on the involved region. The patient had no history of trauma. On physical examination, a 5 × 4 cm immobile mass without clear boundaries was palpated on the left sternocleidomastoid (SCM) muscle. The lump was not fixated to the overlying skin. It did not appear inflamed. Laboratory examination revealed leukocytosis, with a white blood cell count of 13,100/mm³.

US examination was carried out with a color Doppler US scanner (Shimadzu, SDU-2200, Japan) equipped with a 5–10 MHz linear transducer. Gray scale imaging in longitudinal plane revealed a fusiform expansion of the left SCM muscle; the fibrillary pattern of muscle was preserved without prominent discontinuity. The muscle fibrils were interspersed with hypochoic linear structures of non-uniform thickness, giving rise to a dry-cracked mud or a checkerboard-like appearance in the transverse plane (Fig. 1). Color Doppler US examination revealed no abnormal vascularization within the lesion.

CT examination performed with a single-slice spiral CT scanner (Pronto, Hitachi Medical Corporation, Japan) revealed the presence of an expansion on the SCM muscle, containing ill-defined, isodense to slightly hypodense areas with heterogeneous contrast enhancement and linear, reticular hypodense structures (Fig. 2). In keeping with US findings, the aforementioned hypodense linear structures resulted in a checkerboard-like appearance on CT.

MRI examination was performed by a 1.5 T MRI scanner (Signa Excite HD, GE Healthcare, Milwaukee, Wisconsin, USA). On pre-contrast FSE T1-weighted MR images no pathological signal intensity was detected, except for the presence of a fusiform expansion on the SCM muscle. On FSE T2-weighted and T2*-weighted images, a diffuse, irregular, heterogeneously increased muscle signal intensity was detected; the internal strands had a geometrical pattern isointense or slightly hyperintense compared to normal muscle, consistent with intact muscle fibers (Fig.
Although radiological findings suggested an inflammatory, rather than a neoplastic, process, the mass located in the SCM muscle was completely excised because of patient’s suspicious history. Pathological examination of the surgical specimen confirmed the radiological diagnosis of proliferative myositis (Fig. 4).

**Discussion**

Proliferative myositis, first described by Kern (1) in 1960, is a rare pseudosarcomatous process with a special predilection for the trunk and upper extremities, though it infrequently involves the head and neck muscles. It is characterized as a firm, painless, fast-growing soft tissue mass. The mean patient age is 50 years. Interestingly, the lesion can double in size within several days without erythematous change or induration and without fixation to the skin (1–3). Although the etiology of proliferative myositis is unknown, a local traumatic event has been considered to be a predisposing factor. Cases have been reported in association with rheumatoid vasculitis, desmoplastic fibroma, application of cast for fracture, and chromosomal anomalies (3–7).

Proliferative myositis (an intramuscular counterpart of proliferative fasciitis) involves the muscular tissue in a diffuse manner (8). Pathologically, there is an intense fibroblast proliferation in the stromal tissue (perimysium and epimysium), and the bundles of muscle fibers are interspersed with proliferative connective tissue. There are spindle-like cells and large basophilic cells similar to ganglion cells (9).

Radiologically, proliferative myositis manifests as a solid, ill-defined, soft tissue mass causing an expansion in the muscular tissue (3). Longitudinal US scanning reveals the continuity of muscle fibers. On transverse US scanning, hypoechoic linear structures roughly grouping the bundles of muscle fibers give rise to an appearance resembling a checkerboard, dry-cracked mud, or scaffolding, which is a valuable sonographic finding for an accurate diagnosis of proliferative myositis. The relevant pattern defined for the rough grouping of the muscle fibers with preserved continuity is evident on gross pathological examination as well (3, 8–11). Pagonidis et al. (3) described a case with hyperechoic appearing muscle tissue grouped by hypoechoic geographic lines and accordingly they defined it as hyperechoic dense muscle. In literature, two cases with atypical proliferative myositis have been reported; one with a heterogenous mass

3a). After intravenous gadolinium injection, FSE T1-weighted images demonstrated an ill-defined, irregular, marked, heterogeneous contrast enhancement (Fig. 3b).
that had calcifications, as in myositis ossificans (11), and the other with a well-defined, expansile, lobulated, hypechoic intramuscular lesion (12). Sarteschi et al. (8) detected some arterial structures with high-resistance flow within scaffolding by color Doppler US. Wlachovska et al. (12), on the other hand, described an anarchic central vascularization in a case with atypical US findings. However, our patient, like the patient presented by Pagonidis et al. (3), showed no abnormal vascularization.

No characteristic CT finding has been described for proliferative myositis. It has been reported that an ill-defined, expansile lesion appearing isointense or hypointense compared to surrounding muscle may be demonstrated on unenhanced CT scanning. Although it may show homogeneous or heterogeneous enhancement after contrast material administration, no enhancement may be detected in some cases (11, 12). Thus, CT has not been considered a significant tool in the diagnosis of proliferative myositis (3). In our case, spiral CT demonstrated linear reticular hypodense structures, giving rise to a pattern similar to the sonographic checkerboard-like pattern. This finding has not been reported previously, possibly because of technical limitations of the CT equipment used and the lack of extensive inflammatory process. Use of single slice or multislice CT scans as an imaging tool will be needed to assess the nature and predictability of this phenomenon.

On MRI, proliferative myositis is not well demarcated, as with other imaging methods, and is associated with an expansion of the muscle. The lesion appears hypointense or isointense on T1-weighted images and moderately or markedly hyperintense on T2-weighted images (3, 4, 11–14). After intravenous injection of the contrast agent, the lesion demonstrates marked enhancement. On T2-weighted and postcontrast T1-weighted images, relatively hypointense areas with geometrical pattern consistent with the bundles of intact muscle fibers are seen within the lesion with diffuse increased signal intensity, an appearance suggesting the aforementioned checkerboard-like pattern (3, 14). Another MRI finding of proliferative myositis is perilesional edema and contrast enhancement extending to the surrounding fascia (3, 11, 13).

Before the description of the histological features and the benign, inflammatory character of the disease by Kern (1), and Enzinger and Dulcey (2), proliferative myositis was believed to have a malignant course, and patients were usually treated with an aggressive protocol consisting of radical excision with lymphadenectomy, and, in some cases, radiotherapy and chemotherapy. Later,
a marginal excision instead of radical surgery became a standard surgical option (15). The current therapeutic approach reflects the understanding of the self-limited nature of the disease. Investigation includes incisional biopsy or fine needle aspiration biopsy to exclude malignancy, and close follow-up. However, complete excision of the lesion may be necessary in cases with inconclusive biopsy findings (3, 11, 12).

The differential diagnosis of proliferative myositis should include myositis ossificans, trauma, muscle denervation, rhabdomyolysis, polymyositis, dermatomyositis, and soft-tissue malignancies (12). It is possible to differentiate the majority of these entities with clinical and laboratory findings.

The radiological diagnosis of proliferative myositis is of particular importance in order to avoid unnecessary radical surgical excision. It is quite possible to reach a diagnosis with the aid of the findings of US and MRI studies, although histopathological confirmation is necessary for a definitive diagnosis.

References