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REVIEW

Focal myositis: a literature review of clinical and immunopathological aspects

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Objectives. Focal myositis (FM) is a rare and restricted skeletal muscle inflammation, presenting as a solid mass with a typical lower leg localization and benign prognosis. In most cases the process solves spontaneously or after immunosuppressant therapy, but sometimes it recurs or progresses to a systemic inflammation. The basis of the disease are mostly unknown.
Methods. Hence, we provide an update of histopathological features of FM, in order to better define the underlying pathomechanisms of this disorder. A PubMed literature search was focused on the case reports published in English from July 1977 to December 2023.
Results. FM and other myositis may show similar morphological features. Emerging studies on MMP molecules and future eventual research on microRNAs (miRNAs) could help in differential diagnosis.
Conclusions. Clinical, laboratory, neurophysiological and imaging findings can allow a correct diagnosis. However, muscle biopsy seems to be the only diagnostic tool to differentiate among FM and other localized soft tissue masses.

Key words: focal myositis, histopathology, immunohistochemistry, fibrosis, inflammation.

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Introduction

Focal myositis (FM) is a rare, confined muscle inflammation presenting as a benign pseudotumor. In 1977, this disorder was first identified as a new clinicopathologic entity by Heffner R.R. et al., who classified FM apart from the other inflammatory pseudotumors of skeletal muscle, such as proliferative myositis and nodular pseudosarcomatous fasciitis [1]. The mass is usually painful, moveable, and unattached to the surrounding tissues. It is typically localized at thighs or lower legs, growing during a two to eight-week period [1].
The pathogenesis is still not clear and the disease results frequently idiopathic [1]. Furthermore [2-45]. FM can remain a localized process or, rarely, generalize to a polymyositis [15]. An increased risk of recurrence or progression to a multifocal inflammatory myopathy could be suggested by the occurrence of multiple nodules, high level of serum Creatine Kinase (CK) and/or Erythrocyte Sedimentation Rate (ESR), and limb atrophy [16].
Electromyography (EMG), muscle MRI (Magnetic Resonance Imaging), and muscle biopsy may help to reach the correct diagnosis [16].
In this review, we provide an update on clinical and histological features of FM. We conducted a PubMed literature search, selecting the case reports published in English from July 1977 to December 2023. Used search terms were "*focal myositis*" combined with "*histopathology*" and "*immunohistochemistry*". We excluded manuscripts supplying a global description of inflammatory myopathies. Furthermore, search results were screened for relevant studies which can potentially contribute to better define immunopathological aspects of FM.

Etiopathogenesis

The precise mechanism behind FM is unknown, but different hypotheses have been made.

Firstly, several FM cases have been described in association with chronic radiculopathy. However, it is not clear if the denervation is responsible for neurogenic muscle hypertrophy, or it is secondary to inflammation, on the contrary [2-4]. If chronic stimulation triggers the hypertrophy, muscle fibre necrosis can be postulated as result of fibre size increase and splitting. Consequently, the necrosis engages inflammatory cells, as demonstrated in mice models in spontaneous myositis [4,17].

Less frequently, infectious agents, including viruses, bacteria, fungi, protozoa, and worms, have been found in the affected muscle, causing both direct infection and immune- or toxin-mediated injury. Clinical manifestations depend on the type of pathogen and are heterogeneous, such as local muscle abscesses, diffuse infectious myositis, generalized myalgias, and acute rhabdomyolysis. The term "infective myositis" should be used for these patients [5,6,18].

A variable percentage of patients was reported as affected by FM related to neoplasms or autoimmune diseases, suggesting an immunity disorder at the basis of the muscle injury [7-10]. In this regard, a specific profile of matrix metalloproteinases (MMPs) in the muscle have been postulated as possible pathogenic mechanism of FM [19].

A rarer cause of FM lies in the ischaemic condition secondary to atheromatous emboli, diabetic angiopathy, and vascular malformation. In these patients lymphohistiocytic cells infiltrated the muscle endomysial tissue and surrounded arterioles and capillaries, where amorphous material deposits were evident, similar to that seen in the muscle [11-13].

Finally, Asbach P. et al. reported a case due to a long-term statin intake and solved after drug discontinuation [14]. The mechanisms leading to myopathy under statin treatment have not been elucidated; however, it could be postulated that myofibers apoptosis, induced by this medication and involving mitochondrial functions, have a role in the development of the muscle lesion [20].

Clinical, instrumental features and therapy hints

FM typically presents as an inflammatory pseudotumor, restricted to one skeletal muscle [10]. However, the inflammation can involve a muscle part, an entire muscle, or two or more muscles, not necessarily in the same area [21]. Pain, erythema, and fever can be symptomatic of FM, usually without muscle weakness. Furthermore, systemic disorders such as immune-mediated inflammatory diseases, neoplasms, radiculopathies, have been associated with FM [10].

The exact prevalence of this disorder remains unknown [22]. FM occurs in males as well as in females, affecting all ages with a few differences between children and adults. In children, FM is commonly painless, localized in the calf, and with no correlation between recurrence and CK levels [23]. In adults, it has been often reported at thighs and lower legs, less frequently at arms [1,7]. Head and neck muscles are rarely affected. A 72 and 48-year-old men presented a completely asymptomatic FM of the tongue [24,25]. A 55-year-old woman with macroglossia and difficulty in moving her tongue has also been reported [26]. Other few cases of FM localized to perioral and masticatory musculature have been reported, occurring with fever, pain, and trismus in some cases [27-32]. Sternocleidomastoid [33-37] and deltoid muscles [38] FM have been described. Interestingly, Urayoshi et al. reported a man who developed myositis of the deltoid muscle eight days after influenza vaccination [39]. Finally, abdominal and trunk musculature can be similarly interested.[7]

The size of the lesion can vary approximately from 1 to 20 cm, and it may grow over a period of weeks [40]. The prognosis is good with a spontaneous regression of the mass in most cases. A relapsing of the lesion is possible, and it usually involves the same muscle of the first episode. [7,23] However, that is not a rule. Gordon M.M et al. reported a 52-year-old man with a benign pseudotumor in his left thigh, which spontaneously disappeared and reappeared in his left arm after six months [41]. Moreover, several cases of focal inflammation developed into generalized myositis as polymyositis [15,16,42].

An increase of phlogosis markers can be present, as well as myonecrosis markers, but no specific autoantibodies have been identified, in contrast to other idiopathic inflammatory myopathies [43].

EMG and muscle MRI can be useful for differential diagnosis with other benign pseudotumor or malignancy, although muscle biopsy cannot be avoided in most cases [4,21,40,44,45].

Up to date, there are no specific guidelines for the treatment of FM. Glucocorticoids have been efficiently used [40]. Other immuno-

Table I. Clinical, laboratory, neurophysiological and histopathological features of FM representative case series.

Pts n.	Clinical features	Laboratory findings	Neurophysiological examinations	Imaging	Histology	Immunohistochemistry	Ref.
8 pts	Solid mass in: lower limbs (6 pts) upper limbs (1 pt) abdominal wall (1 pt) Pain in 5 pts	Elevated CK levels in 4 pts	EMG in 5 pts: complex repetitive discharges (3 pts) myopathic pattern (5 pts)	Muscle MRI in 5 pts: edema patchy gadolinium enhancement	Fibre size variation Lobulated pattern Necrosis/fibrosis Internalized nuclei	T cells (invading muscle fibres) macrophages	16
4 pts	Solid mass in: lower limbs (1 pts) upper limbs (1 pt) Pain in	Normal CK levels in 3 pts, slightly elevated in 1 pt	EMG in 1 pt: insertional activity increase	Muscle MRI in 4 pts: edema inflammation	Fibre atrophy Fibrosis	CD4+ cells CD8+ cells macrophages MHC-1 upregulation	46

Pts n.	Clinical features	Laboratory findings	Neurophysiological examinations	Imaging	Histology	Immunohistochemistry	Ref.
7 pts	Solid and painful mass in lower limbs (7 pts)	Slightly elevated CK levels in 7 pts	EMG in 7 pts: myopathic pattern with fibrillation potentials	Muscle MRI in 7 pts: edema inflammation	Degeneration/ regeneration Internal nuclei Fibre splitting Rare necrosis	CD4+ cells (2 pts) CD8+ cells (7 pts) CD22+ cells (2 pts) CD68+ macrophages (6 pts) MHC-I expression (7 pts) MAC deposits (1 pt) Slight MMP2/MMP7 immunoreactivity in some endomysial and perimysial vessels MMP9 expression in scattered atrophic fibres	19
115 pts	Solid mass in: lower limbs (70 pts) upper limbs (18 pts) head (16 pts) trunk, hip, abdomen (11 pts) Tenderness or pain (31 pts)	n.r.	n.r.	n.r.	Myopathic/neurogenic changes Endomysial/perimysial fibrosis Fibre size variation Internalized nuclei Germinal centers Vacuolar change Amorphous substance	Performed in 20 pts: CD163+ macrophages (90%) CD3+ cells (18 pts) CD4+ cells more than CD8+ CD20+ cells (9 pts) MHC-1 expression (15 pts) weak IgG4 expression (13 pts)	7
4 pts	Solid and painful mass in lower limbs (4 pts)	Elevated CK levels in 2 pts	EMG in 4 pts: neurogenic pattern (acute and chronic denervation)	Muscle MRI in 4 pts: edema, atrophy, and fat muscle hypertrophy	Atrophic/hypertrophic fibres Internal nuclei Regenerating fibres No fibres type predominance Necrosis/fibrosis	Lymphocytes and macrophages (within necrotic fibres) Marked MHC-1 upregulation	4
37 pts	Circumscribed mass (23 pts) or multi-monofocal myositis (12 pts) in: lower limb (26 pts) upper limb (8 pts) head or neck (3 pts) Pain, erythema, and fever (variably associated)	Elevated CK levels in 6 pts	EMG in 20 pts: normal (4 pts) myopathic pattern (12 pts) neurogenic pattern (4 pts)	Muscle MRI in 27 pts: focal inflammation (25 pts) fascia impairment (7 pts)	Myopathic/neurogenic changes Fibre size variation Internalized nuclei Necrosis/fibrosis	CD3+ cells as prominent cells CD4+ cells in all pts CD8+ cells (56% of pts) CD68+ macrophages (93%) CD20+ cells (81%) MHC-I overexpression C5b9 staining (21 pts)	10

Abbreviations: EMG = electromyography, MRI = magnetic resonance image, MHC-1 = major histocompatibility complex-1, MAC = membrane attach complex, MMP = matrix metalloproteinase.

suppressive drugs such as azathioprine or cyclophosphamide were prescribed as second line, in case of clinical worsening, steroid dependence, or relapse [10].

Representative case series of FM are reported in Table I.

Histological features

The first FM histologic description was that of a "*severe myopathy with inflammation*" [1], resulting in normal muscle architecture loss and moderate vascularity [47]. Galloway H.R et al. reported also a marked endothelial swelling of medium-sized vessels without evidence of vasculitis [48].

Muscle fibre size variation has been shown with both hypertrophic and rounded-angular atrophic fibres. Internal nuclei were also frequent and necrosis and regeneration with complete/uncomplete splitting could occur simultaneously [1,5,40,49]. Regenerating cells were recognized as having basophilic sarcoplasm when stained with Hematoxylin and Eosin (H&E) [1]. Indeed, hyaline vacuolated or fragmented muscle fibres were randomly alternated with floccular necrotic fibres [24]. *Ring fibres* have been shown in Phosphotungstic Acid Haematoxylin (PTAH) or Periodic Acid-Schiff (PAS) preparations, due to perpendicular orientation of affected myofibrils to their longitudinal axis, around the inner normal portion [1]. Another common feature was represented by fibrosis involving perimysium and endomysium, especially in older cases [7,50]. Real "lobules" of muscle fibres, compactly grouped and fibrosis-surrounded, have been reported [16]. ATPase and oxidative enzyme reactions (Nicotinadmide Adenine Dinucleotide-NADH, Succinic Dehydrogenase-SDH) evidenced a normal checkerboard profile with regular fibre types representation and no clear type's predominance [1]. However, a predominance of type 1 fibres was rarely noted and *moth-eaten fibres* were apparent with oxidative enzyme reactions [49].

These histological findings are consistent with an unspecific myop-

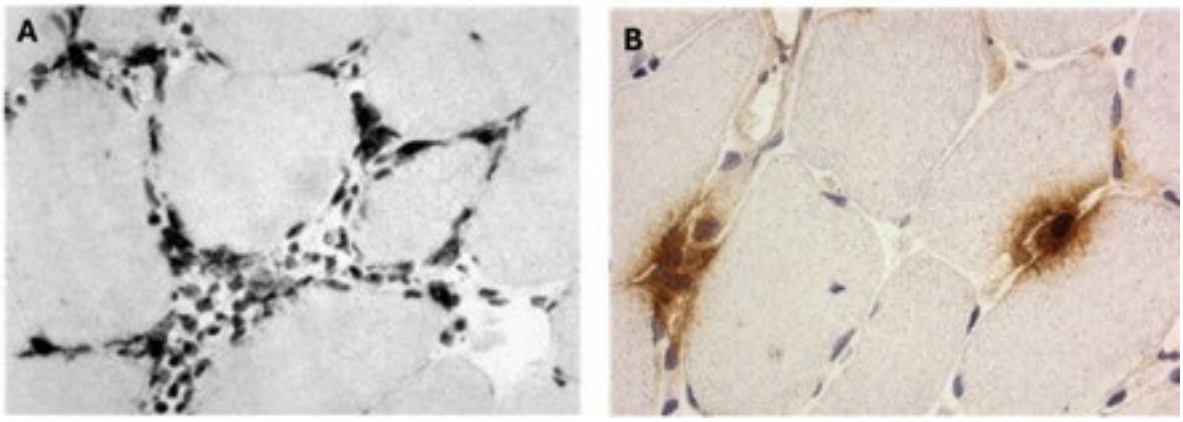

Figure 1. Muscle biopsy immunoistochemical images. T-cell CD8+ endomysial infiltrates surrounding muscle fibres in splenius capitis muscle (A) [53]; scattered macrophages CD68+ in gemellus medialis muscle (B; image from authors' personal database) (Magnification: 340).

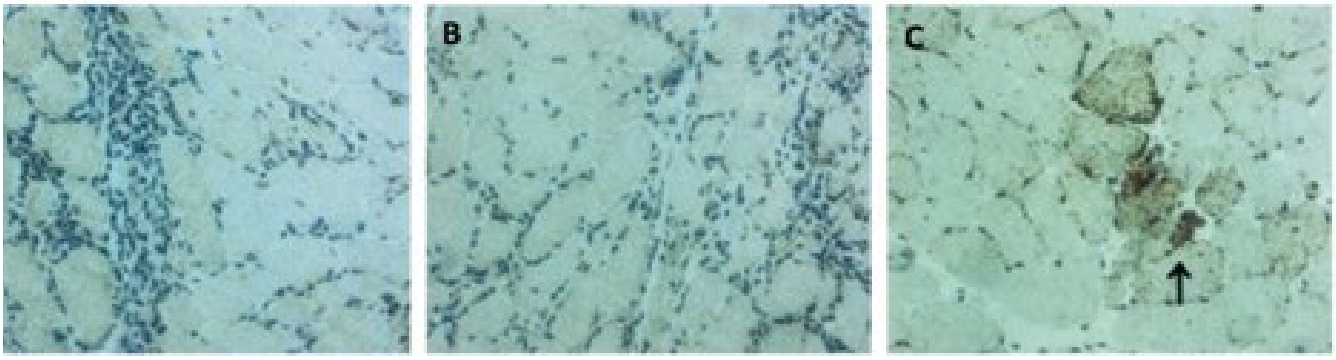

Figure 2. MMPs role in differential diagnosis among inflammatory myopathies: absent MMP2 (A) and MMP7 (B) immunoreactivity in FM specimens; MMP9 expression in scattered atrophic muscle fibres (C, arrow) (Magnification: 280) [19].

athic process, making the differential diagnosis a real challenge [30].

Immunohistochemical features

FM immunohistochemical findings have not been deeply investigated. Neutrophils, lymphocytes, plasma cells, macrophages, and a small percentage of eosinophils have been encountered, infiltrating perimysial and endomysial compartments or collecting nodules within the interstitium [7,27]. However, the predominant elements were T cells (CD3+, CD8+) and macrophages (CD68+), with a minor B-cell component (CD20+) (Fig.1) [5,16,19,30,35,48,51]. Furthermore, Gallay L. et al. noticed that B cells and macrophages were mainly present in FM cases associated with autoimmune disorders and neoplasia [10].

MHC-I (major histocompatibility complex-1) was variably overexpressed on the sarcolemma of muscular fibres, with a distribution pattern different from the perifascicular one, typical of dermatomyositis, and the diffuse positivity usually found in inclusion body myositis [7,10,29,52]. MHC-I seemed to be marked in specimens with a higher number of CD8+ cells [19,52,53]. Some clusters of muscle fibres, especially vacuolated fibres, were positive for S100. The expression of IgG4 could be also detectable, explaining FM fibrosis and its autoimmune aetiology [7]. Finally, C5b9 staining was observed in different profiles, although its relevance remains unknown, contrary to dermatomyositis or necrotizing myopathies [10].

Interestingly, a role in differential diagnosis among inflammatory myopathies has been recognized to MMPs. Rodolico C. et al. detected a slight MMP2 and MMP7 immunoreactivity in some endomysial and perimysial vessels in patients with FM. Muscle fibres and infiltrates were negative, contrarily to polymyositis and dermatomyositis in which MMP2 and MMP7 were identified in atrophic myofibres (MMP7 was revealed only in polymyositis). Nonetheless, MMP9 was expressed in scattered atrophic muscle fibres in patients with FM, polymyositis, and dermatomyositis. (Fig. 2). The same fibres were also positive for MHC class I antigens and were considered as regenerating fibres [19]. In another paper, Cain A.J. et al. detected immunopositivity for myoglobin and desmin in regenerative fibres, confirming skeletal muscle differentiation [35].

Further studies have been conducted on tissue transglutaminase or transglutaminase 2 (TG2), which is involved in several pathological process such as inflammation and fibrosis. Indeed, TG2 appeared overexpressed in endomysial vessel walls in dermatomyositis, polymyositis, sporadic inclusion body myositis and FM. However, TG2 expression pattern in FM was similar to the other inflammatory myopathies, with a variable expression degree as regard to the amount of necrotic and degenerating/regenerating muscle fibres. Moreover, it is still not clarified TG2 role as proinflammatory proteins or as inflam-

mation-reducing agent [52].

Conclusion

The principal aim of this review is to collect clinical and histological features of FM.

Although limited data are currently available about this disorder and its pathogenesis, we tried providing basic principles to distinguish this specific entity from the other inflammatory myopathies. However, FM and other myositis may show similar morphological features. On one hand, inflammatory cells' infiltrate mainly consists of CD3+ and CD8+ lymphocytes, and MHC-I is expressed in muscle fibres as well as in polymyositis and dermatomyositis. On the other hand, it seems that emerging discoveries on MMP molecules could help in differential diagnosis. MMP2 and MMP7 are positive in some endomysial and perimysial vessels in FM, while muscle fibres and infiltrates are negative [19,52].

Nevertheless, further insights should be gained to deeply investigate FM pathomechanism. A future prospective could be represented by research on microRNAs (miRNAs), small noncoding RNAs regulating different physio-pathological processes, such as autoimmunity and inflammation. Muscle-specific miRNA (myomiRs) have been recognized in inflammatory myopathies, suggesting a role in myofibre damage [54,55]. Then, miRNA profiling could be used as potential biomarker of different myositis and, what is more, they could correlate with disease generalization and response to therapy.

Abbreviations

FM	Focal myositis
CK	Creatine kinase
ESR	Erythrocyte Sedimentation Rate
EMG	Electromyopgraphy
MRI	Magnetic Renonance Imaging
H&E	Hematoxylin and Eosin
PTAH	Phosphotungstic Acid Haematoxylin
PAS	Periodic Acid-Schiff
NADH	Nicotinadmide Adenine Dinucleotide
SDH	Succinic Dehydrogenase
MHC-I	Major Histocompatibility Complex-I
MMP	Metalloproteinase
TG2	Transglutaminase2

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Conflicts of interest statement

All authors declare no conflicts of interest.

Author's contributions

AP wrote the first draft of the manuscript; AM, AB, and FB participated to the literature review preliminary to draft the paper. CR, AT, and OM critically revised the manuscript. All authors approved the final version of the manuscript.

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