Magnetic resonance imaging of skeletal muscle involvement in limb restricted vasculitis

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CASE REPORTS

Patient 1

A 40 year old man with no medical history of consequence was referred to our department in December 1998 because of a three month history of painful induration of the external face of the left calf and arthralgia of the left ankle.

Physical examination found no fever, a blood pressure of 140/80 mm Hg, and a palpable tumour about 2 cm in diameter on the external face of the left calf. Laboratory data showed an erythrocyte sedimentation rate (ESR) of 39 mm/1st h. Creatine kinase (CK) and aldolase levels, liver function tests, and serum complement levels were normal, and there was no creatininemia or proteinuria. Antibody tests for HIV and hepatitis C virus were negative, and hepatitis B virus serology detected only anti-HBs antibodies. Tests for antinuclear antibodies (ANAs), antineutrophil cytoplasm antibodies (ANCAs) and cryoglobulins were negative, and a chest radiograph was normal. Magnetic resonance imaging of the calves showed normal unenhanced T1 weighted sequences, and hyperintense signals on T2 weighted and slow tau inversion recovery (STIR) images of the peroneus longus, peroneus brevis, and tibialis anterior muscles of the left calf (fig 1); the same areas were slightly enhanced by gadolinium in T1 weighted sequences. Histological examination of biopsy performed in the pathological area disclosed vasculitis of medium sized (250 µm) arteries with fibrinoid necrosis of the vessel wall, and a polymorphous inflammatory infiltrate containing predominantly neutrophils and eosinophils. Limb restricted PAN-type vasculitis of skeletal muscle was diagnosed.

In January 1999, prednisone (40 mg/day) was started, and provided rapid relief of myalgia and arthralgia, but the symptoms recurred during dose tapering. Serial MRI of the calves showed persistent signal abnormalities of the left anterolateral compartment. In June 2000, azathioprine (100 mg/day) was added to the prednisone. One year later, the patient is still taking azathioprine and prednisone (20 mg/day) and has only moderate pain.

Patient 2

In June 1995, a previously healthy 41 year old woman was referred to our department for myalgia of the right calf, arthralgia of the right ankle of 10 months' duration, and subsequent development of bilateral skin nodules in the legs.

Temperature and blood pressure were normal, but there were multiple tender subcutaneous nodules on both lower limbs with slight inflammatory oedema and tenderness of the underlying muscles. Her ESR was 42 mm/1st h. Values for CK,
aldolase, and serum complement were normal and there was no creatininaemia. Serological assays for hepatitis B virus (HBV) and hepatitis C virus (HCV), ANA, ANCA, rheumatoid factor (RF), and cryoglobulins were negative. Radiographs of the calves and a technetium-99m bone scan were consistent with bilateral but predominantly right fibular periostitis. Magnetic resonance imaging of the calves showed bilateral and patchy distributions of hyperintense signals on T2-weighted images of the anterolateral and posterior compartments. The involved area was enhanced by gadolinium in T1-weighted images. Histological examinations of a muscle biopsy guided by MRI and an excised cutaneous nodule showed fibrinoid necrosis of a medium sized artery wall, an inflammatory cell infiltrate with lymphohistiocytes, and epithelioid giant cell granuloma. Lower limb restricted PAN was diagnosed.

Prednisone (50 mg/day) was started in September 1995. The symptoms regressed but myalgia recurred in October 1996 on 5 mg prednisone a day. Colchicine (1 mg/day) was prescribed and allowed transient discontinuation of prednisone. In June 1998, prednisone (10 mg/day) was reintroduced because of the reappearance of cutaneous lesions and was continued as maintenance treatment. In April 1999 a new disease flare up occurred with MRI of the calves showing the same abnormalities as previously.

**Patient 3**

In August 1998, a 45 year old woman was referred to our department because of an 11 month occurrence of arthralgia of the ankles and severe myalgia with swelling of the calves. The patient had a history of chronic HCV infection (serotype 3) diagnosed in 1997. A needle biopsy of the liver performed in April 1998 was consistent with chronic mildly active hepatitis. Physical examination showed a normal temperature, a blood pressure of 140/80 mm Hg, and bilateral tenderness with a slight, inflammatory oedema of the calves, which had several small sized erythematous macular lesions. Laboratory tests showed an ESR of 30 mm/1st h, and normal CK and lactate dehydrogenase values. Liver enzymes, a chest radiograph, and urinary sediment were normal and there was no creatininaemia or proteinuria. Serum HCV RNA was detected at 13 000 000 copies/ml. Serological assays for HBV, HIV, ANA, RF, ANCA, and cryoglobulins were negative. Magnetic resonance imaging of the calves showed normal T1-weighted sequences but hyperintensities on T2-weighted or STIR sequences involved one or several muscle groups of the gastrocnemius muscle and the medial head of the right gastrocnemius muscle. The lateral head of the left gastrocnemius muscle is still swollen and shows a mildly intense signal.

**DISCUSSION**

Our findings in these three patients suggest that MRI can assess the muscle involvement occurring in PAN or PAN-type vasculitides restricted to the limbs. Indeed, muscle vasculitis appears as hypersignals in T2-weighted or STIR sequences. In T-weighted sequences, the affected areas remained normal but were slightly enhanced by gadolinium. In all three patients, the hyperintensities involved one or several muscle groups of one or both lower limbs. These results are in accordance with those of Hofman et al, who reported the MRI findings in a patient with limb restricted PAN.19

Hyperintensities appearing predominantly or exclusively in T-weighted or STIR sequences indicate increased muscle fluid content. Our patients' radiological findings are most likely consistent with oedema, which is the principal feature of muscle inflammation.10,14 However, because myopathies of
traumatic, metabolic, and degenerative origin and peripheral neuropathies.\(^{12–15}\) exhibit these same changes, some authors prefer the term oedema-like abnormalities rather than oedema.\(^{16}\) It is therefore unlikely that a diagnosis of limb restricted vasculitis may be evoked based solely on MRI findings. In particular, the patchy, asymmetric, and distal distribution of the lesions resembles that found in focal myositis.\(^{13}\)

Despite an apparently poor specificity, MRI of skeletal muscle could constitute a useful complementary examination for limb restricted PAN or PAN-type vasculitides. These diseases are often difficult to recognize because of their indolent course and the poor specificity of clinical and laboratory signs. Myalgia and stiffness are the main clinical signs of muscle involvement.\(^{14,15}\) Levels of CK are normal or only slightly increased\(^{7}\) and electromyograms inconsistently show a myogenic pattern.\(^{2}\) Hence, our findings would indicate that MRI has a higher ability to detect vasculitic muscle disease. None the less, further investigation needs to be undertaken to evaluate the precise sensitivity of MRI in this setting. Another application of MRI could be to select the optimal site for muscle biopsy to ensure that the sampled tissue comes from an affected area, whereas serial MRI might be helpful for monitoring the course of the disease and response to treatment. As suggested by the normalisation of the oedema-like abnormalities of patient 3 under treatment and the patient reported by Hofman et al.,\(^{16}\) it would seem that the MRI results coincide with disease activity, although these findings require confirmation.

It would also be informative to investigate the usefulness of skeletal muscle MRI for systemic PAN or other systemic vasculitides. In systemic PAN, skeletal muscles are often involved and represent one of the preferential biopsy sites for histological confirmation.\(^{1}\) The technique of MRI could therefore contribute to diagnosing paucisymptomatic disease or selecting the muscle biopsy site. It remains, however, to be shown that the muscle involvement occurring in systemic PAN generates the same changes in MRI as those seen in the more longstanding, limited disease.

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