Skeletal Muscle MRI in Complex Regional Pain Syndrome

Yoichiro Nishida¹, Yuki Saito¹, Takanori Yokota¹, Takashi Kanda¹² and Hidehiro Mizusawa¹

Abstract

Background  Conventional magnetic resonance imaging (MRI) sequences of patients with complex regional pain syndrome (CRPS) have shown abnormal signals in skin, soft tissue, joints, bone, and bone marrow, but not yet in skeletal muscles, during the acute phase. The aim of this study was to clarify whether or not the affected muscles in CRPS patients show abnormal MRI signal intensities or signal enhancement by gadolinium dimeglumine during the acute phase.

Patients and Methods  MRI studies of skeletal muscles were performed on three patients of CRPS. Out of three patients, MRI was performed on three at stage 1, one in improving phase, two in remission phase, and one at stage 3. MRI was performed in the transaxial plane with both T2-weighted imaging (T2WI) and fat-suppressed T1-weighted imaging (T1WI) with or without gadolinium dimeglumine enhancement.

Results  All patients at stage 1 showed hyperintense muscle signals on T2WI and gadolinium dimeglumine enhancement on T1WI. Following clinical improvement, the hyperintense lesions reverted to near normal. Muscles in the chronic phase showed high signals on both T2WI and T1WI without gadolinium dimeglumine enhancement.

Conclusion  MRI abnormalities in the acute phase are consistent with muscular edema, interstitial edema, and vascular hyperpermeability. These MRI findings suggest the presence of hemodynamic abnormalities caused by microangiopathy, sympathetic abnormalities, or both, which may lead to ischemia of affected muscles. Chronic phase abnormalities indicated the presence of muscle atrophy and fibrosis or fatty infiltration of the affected muscle.

Key words: acute phase, complex regional pain syndrome, magnetic resonance imaging, microangiopathy, muscle, reflex sympathetic dystrophy

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Introduction

Complex regional pain syndrome (CRPS), which has also been called as reflex sympathetic dystrophy (RSD), causalgia, Sudeck’s atrophy, algodystrophy and shoulder-hand syndrome, presents as an array of painful conditions that are characterized by a continuing regional pain that is seemingly disproportionate in time or degree to the usual course of any known trauma or other lesion (1). The pain is regional and usually has a distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. CRPS is commonly divided into three stages. Stage 1, the inflammatory stage, lasts a few months and is characterized by diffuse locoregional burning pain, edema, redness, and warmth. In stage 2, the dystrophic stage, pain during exercise, decreased skin temperature, hair loss, and the beginning of skin and muscle atrophy are shown. Stage 3, the atrophic stage, is characterized by persistent pain on exercise, scleroderma-like skin changes, and muscle atrophy. Some patients with stage 1 or 2 CRPS show marked improvements after treatment with sympathetic ganglion block, nonsteroidal anti-inflammatory drugs, or corticosteroids. Excluding conversion disorder can be difficult in the acute phase, when the differential diagnosis must be made with non-invasive techniques, such as magnetic resonance imaging (MRI). MRI is one of the noninvasive techniques and its application has greatly increased the sensitivity and accuracy of radio-
logical diagnosis of various disorders. Nuclear magnetic resonance (NMR) spectroscopy reveals changes in skeletal muscle during the acute phase of CRPS (2), and conventional MRI indicates mainly muscle atrophy, fibrosis, or fatty infiltration in the chronic phase (3-5). We report the features of skeletal muscles in CRPS patients by conventional MRI, because it might prove useful for clinical investigation as well as give new insight into the pathophysiology of the disease.

Patients and Methods

Patients

CRPS diagnosis and staging were based on characteristic clinical symptoms and examination. Patients showed increased uptake of technetium methylene diphosphonate (99mTc-MDPT) on bone scintigraphy, and changes in skin temperature, blood flow, or sudomotor function. Patients with other neurological disorders, relevant comorbidity and conversion disorder, were excluded. Three patients participated in this study, and their age, sex, disease trigger, affected limb, clinical staging, and disease duration at the time of MRI are shown in Table 1. MRI was performed on three at stage 1, one in the improving phase, two in the remission phase, and one at stage 3.

Patient 1

A 65-year-old woman suffered swelling and pain in the right foot without any possible initiating triggers. Her skin became purplish red and she was diagnosed as CRPS. Her clinical symptoms were remitted by oral NSAIDs. Crural muscular MRI was done at stage 1 and in the remission phase (three and 13 months from symptom onset, respectively).

MRI

MRI studies of skeletal muscles using T1-weighted imaging (T1WI) and T2-weighted imaging with fast spin-echo technique (T2WI) in the transaxial plane were performed using a high field strength (1.5 T), superconducting General Electric Signa MRI system. In addition, fat-suppressed T1WI was performed before and after the administration of gadolinium dimeglumine. We visually determined the enhancement effect of the gadolinium dimeglumine by comparison with uninvolved limbs, and categorized signal change severities on T2WI in the affected muscle into four groups: normal, no high-intensity area; mild, 0% to 33% of the whole muscle area showed high intensity; moderate, 33% to 66%; and severe, more than 66%, which is a modified method of categorization from a previous one (6). Two authors (Y.N. and Y.S.) independently quantified these changes and yielded the same results.

Results

In patient 1, diffuse, abnormally high T2WI signals were observed in some muscles of the affected lower limb at stage 1 (Fig. 1A and Table 1). These muscles also showed high signals on fat-suppressed T1WI only after the administration of gadolinium dimeglumine (Figs. 1B, 1C). Follow-up T2WI and T1WI at stage 3 showed reduced muscle volume in the affected leg and abnormal, linear areas of high signal intensity (Figs. 1D, 1E). The area of T2WI signal changes was reduced in some muscles (Table 1), and there was no enhancement by gadolinium dimeglumine (Fig. 1F). In patient 2, diffuse, abnormally high signals were observed at stage 1 in some muscles of the right forearm, both on T2WI and on fat-suppressed T1WI after the administration of gadolinium dimeglumine. There were no abnormal muscular signals on non-enhanced T1WI. At 5 and 20 months after onset, the high-signal areas on T2WI were gradually reduced in size in parallel with clinical improvement, and gadolinium dimeglumine gave no signal enhancement (Table 1). Although the time course and appearance of the abnormal MRIs were similar in patients 1 and 2, the patients had different clinical outcomes. In patient 3, some muscles of the affected leg showed diffuse high signals on T2WI at stage 1. Gadolinium dimeglumine enhanced fat-suppressed T1WI, although some muscles showed slightly higher signals on T1WI even without enhancement. The high-signal areas on T2WI were decreased, and the enhancement effect of gadolinium dimeglumine disappeared by 13 months after onset, which coincided with recovery (Table 1).
Figure 1. Lower limb muscle MRI of patient 1 in the acute phase at two months after onset (A-C), and in the chronic phase at 22 months after onset (D-F). At stage 1, T2WI revealed diffuse, abnormally high signals in affected muscles in the left leg (arrows in A), while fat-suppressed T1WI was normal (B). Fat-suppressed T1WI after the administration of gadolinium dimeglumine showed enhancement on most of the muscles showing increased signals on T2WI (arrowheads in C). On the other hand, at stage 3, T2WI (arrows in D) and T1WI (arrowheads in E) revealed linear, abnormally high signals, and reduced muscle volume in the affected leg. Fat-suppressed T1WI revealed no enhancement by gadolinium dimeglumine (F).

Table 1. Summary of Profiles and MRI Findings in Five Patients with CRPS

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age (Sx)</th>
<th>Disease trigger</th>
<th>Affected limb(s)</th>
<th>Clinical stage</th>
<th>Disease duration</th>
<th>Distribution of affected muscles</th>
<th>Enhancement effect</th>
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<tbody>
<tr>
<td>1</td>
<td>31</td>
<td>traffic accident</td>
<td>left L/E</td>
<td>stage I stage III</td>
<td>22 months</td>
<td>FDL, FHL, So, Pe, TP</td>
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<td>(M)</td>
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<td>2</td>
<td>24</td>
<td>blood drawing</td>
<td>right U/E</td>
<td>stage I improving phase</td>
<td>5 months</td>
<td>APL/EPB, FDP, PT, Su</td>
<td>(+)</td>
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<tr>
<td></td>
<td>(F)</td>
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<td></td>
<td></td>
<td></td>
<td>3+, 3+, 3+, 2+, 2+</td>
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<tr>
<td>3</td>
<td>65</td>
<td>unclear</td>
<td>right L/E</td>
<td>stage I remission phase</td>
<td>13 months</td>
<td>FDL, FHL, TP, So</td>
<td>(+)</td>
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<td></td>
<td>(F)</td>
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<td>3+, 3+, 3+, 2+, 2+</td>
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U/E = upper extremity; L/E = lower extremity; 1+ = mild signal changes; 2+ = moderate signal changes; 3+ = severe signal changes, as defined in the text; (+) = present; (-) = absent; FDL = flexor digitorum longus muscle; FHL = flexor hallucis longus muscle; So = soleus muscle; Pe = pectoral muscle; TP = tibialis posterior muscle; APL/EPB = abductor pollicis longus and extensor pollicis brevis muscle; FDP = flexor digitorum profundus muscle; PT = pronator teres muscle; Su = supinator muscle.

Discussion

CRPS patients show abnormal MRI signals, either in T2WI, T1WI, or short-tau inversion recovery images, in skin, soft tissue, joints, bone, and bone marrow at various stages (3-5, 7). Only four reports refer to abnormal muscle MRI findings. Three of them primarily describe muscle atrophy, fibrosis, and fatty infiltration in later disease stages by conventional MRI sequences (3-5), and the other one shows abnormal muscular metabolism of high-energy phosphate by 31P NMR spectroscopy (2). To date, no reports have dis-
cussed the values of muscular abnormal signal changes in the acute phase shown by conventional MRI sequences. Here, we describe abnormal muscular signals in the acute phase of CRPS on T2WI and on T1WI with gadolinium enhancement. All three patients assessed at stage 1 showed hyperintense signals in affected muscles on T2WI, with most of these areas also showing enhancement with gadolinium dimeglumine on T1WI. These MRI features indicate that enhancement. All three patients assessed at stage 1 showed hyperintense signals on T2WI and on T1WI with gadolinium enhancement. These MRI features indicate that enhancement. All three patients assessed at stage 1 showed hyperintense signals on T2WI and on T1WI with gadolinium enhancement.

In conclusion, we report that muscle MRI of CRPS patients in the acute phase showed hyperintense signals on T2WI and on T1WI with gadolinium enhancement. These MRI findings are consistent with the presence of hemodynamic abnormalities caused by microangiopathy, sympathetic abnormalities, or both, which may lead to ischemia of the affected muscles, spreading from localized to adjoining muscles in acute pathophysiology. Although further studies of larger numbers of patients are needed, muscle MRI could be useful for the diagnosis and staging of CRPS.

References