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## Alcoholic Myopathy Accompanied with Acute Reversible Encephalopathy

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Patients of chronic alcoholism may show many kinds of complications such as myopathy, nutritional problems, peripheral neuropathy, withdrawal seizure and encephalopathies. We report an unusual case of alcoholic myopathy diagnosed with typical laboratory and pathological findings initially manifested as acute reversible encephalopathy showing transient abnormalities on brain MRI.

**Key Words:** Alcohol, Myopathy, Encephalopathy

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There are many kinds of complications about chronic alcoholism such as myopathy, nutritional problems, neuropathy, withdrawal seizure and encephalopathies. Among them, it is well known that alcoholic myopathies have characteristic pathological features according to their types.<sup>1</sup>

Acute alcoholic myopathy is caused by severe alcoholic binges. It shows rapid onset muscle pain and swelling with myoglobinuria. On the other hand, chronic alcoholic myopathy complicates years of alcohol abuse. It is characterized by slowly progressive and painless proximal weakness correlated with lifetime consumption of ethanol<sup>1</sup>.

We report a case of acute alcoholic myopathy superimposed on chronic alcoholic myopathy initially presented as acute reversible encephalopathy showing transient abnormalities of brain magnetic resonance images (MRI).

### CASE REPORT

A 59-year-old woman admitted due to acute mental change without definite history of seizure episodes because she was found by her family member. On first neurological examinations in regional hospital, she showed stuporous mentality for several minutes. Her mental status recovered fully after one or two hours during transfer to our hospital.

She was a chronic alcoholic for about 10 years. In history, she noticed progressive motor weakness especially during walking and standing since 6 months ago. And she experienced several episodes of much more severe disability with pain

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on legs remitted for spontaneously. From the before 3 days on admission, she reduced amount of alcohol consumption due to exacerbated weakness and pain on her legs.

Neurological examinations revealed quadriparesis dominant on proximal limbs muscles (MRC Grade IV+). Laboratory findings showed markedly elevated serum creatine kinase (CK) and lactate dehydrogenase level (over 16000 and 5068 U/L, respectively) and positive urine myoglobin. Routine complete blood counts, electrolytes and chemistry including renal function tests showed no definite abnormal finding except for elevated hepatic enzymes. Thyroid function tests, rheumatological laboratories (RA, ANA, Anti-Ro and Anti-La) and vitamin levels (thiamine, folate, pyridoxine, cobalamin) were within normal limits. Cerebrospinal fluid examinations revealed no other abnormal finding.

Electroencephalography (EEG) on admission showed mild diffuse slowing without epileptiform discharges. Needle electromyography (EMG) studies revealed chronic active myopathic discharges especially in proximal limb muscle group. Brain MRI of diffusion weighted and FLAIR sequences images showed multiple diffuse high signal intensities in bilateral frontoparietal cortical and subcortical areas (Fig. 1) and these findings were regressing rapidly in follow-up images (Fig. 2). Pathological findings from right biceps muscle revealed severe atrophic changes and size variation of type I muscle fibers compatible with chronic alcoholic myopathy (Fig. 3).

On education and psychiatric support, after abstaining from alcohol, her motor functioning was improved slowly and she was discharged without any other complication.

## DISCUSSION

Patients with alcoholic myopathy show a variety of manifestations. They are classified into two types of myopathies, acute and chronic!

Acute alcoholic myopathy with myonecrosis develops suddenly in the context of binge drinking with muscle pain and swelling. Its severity ranges from asymptomatic transient elevation of creatine kinase to frank rhabdomyolysis with myoglobulinuria. Occasionally, it is associated with acute alcohol withdrawal. Frequently, EMG

findings show no abnormality. Pathological findings show scattered type I predominant muscle fibers (high oxidative, low glycolytic, slow twitch) necrosis suggesting mechanisms of oxidative damages.<sup>2</sup>

Chronic alcoholic myopathy is a gradually evolving syndrome of proximal weakness and atrophy characterized by slowly progressive proximal weakness and muscle atrophy. In pathology, there are striking muscle atrophy and degeneration of type I fibers, especially type I B muscle fibers (high glycolytic, low oxidative, fast twitch). EMG may show myopathic potentials, neuropathic features, or both. In some cases, acute muscle injury, manifested by muscle tenderness and creatine kinase elevation, may be superimposed on chronic alcoholic myopathy.<sup>3</sup>

Our patient showed typical history and findings of chronic alcoholic myopathy but, she was manifested with acute alcoholic myonecrosis. It is explained by the fact that chronic alcoholic myopathy is accompanied by acute type I in some times if there is abrupt reduce or abstinence of alcohol consumption.

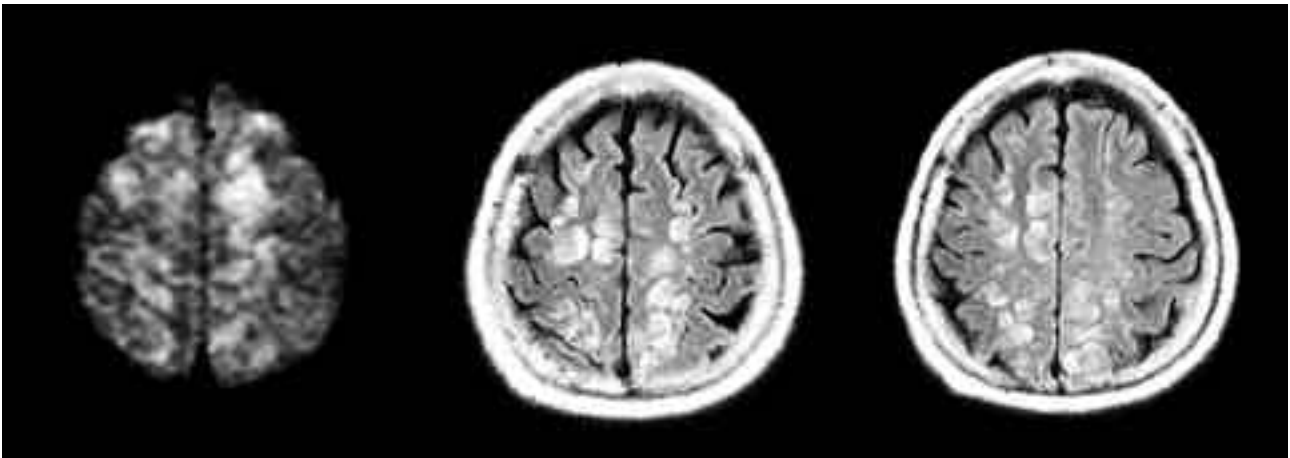
Unusual findings of brain MRI in this patient are interesting. Though there are many kinds of abnormalities associated with alcoholism, we cannot explain the exact nature of these acute transient multiple cortical and subcortical abnormalities of brain images because we meet this kind of findings infrequently.<sup>4</sup> In concerning the time relationships, it may be proposed that multiple high signal intensities of brain MRI are closely related with acute mental change and it may be provoked by recent alcohol abstinence or alcoholic rhabdomyolysis not by vitamin deficiency.

There are several kinds of complications manifested as encephalopathy in chronic alcoholics. Among them, there is a rare type of disease such as subacute encephalopathy with seizures in chronic alcoholism (SESA syndrome). It is characterized by focal seizures in chronic alcoholics and can be diagnosed by periodic lateralized epileptiform discharges (PLEDs) in the EEG with or without abnormalities in brain images.<sup>5,6</sup> Same as reported cases of SESA syndrome, our patient's mentality was recovered without remained neurological deficits except for muscle weakness. But, there was no definite evidence of seizure episode in our patient by history. Also we could

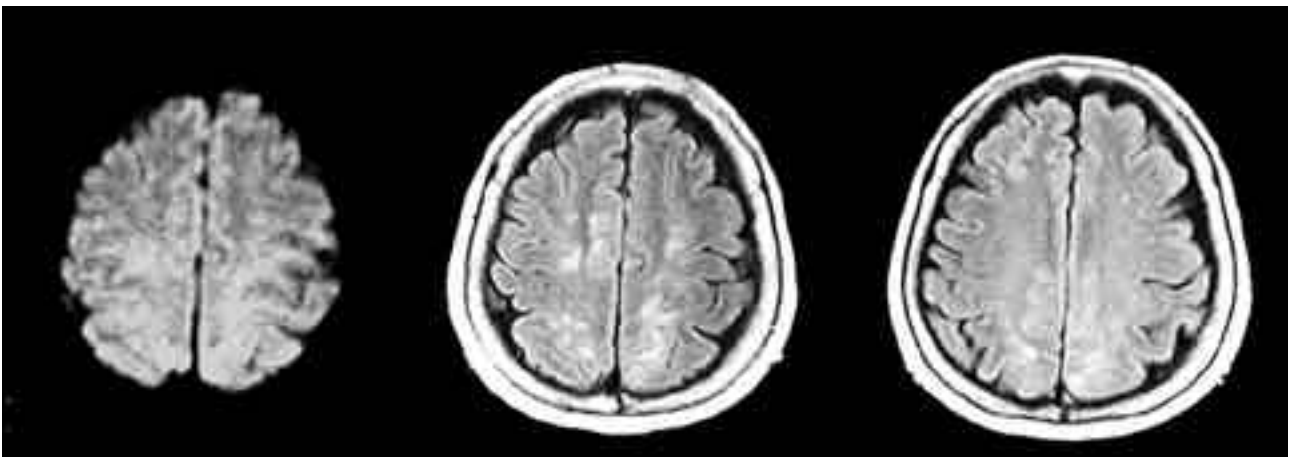
not get adequate information from EEG though this may be due that it was done in recovering state.

There are other possibilities that the findings

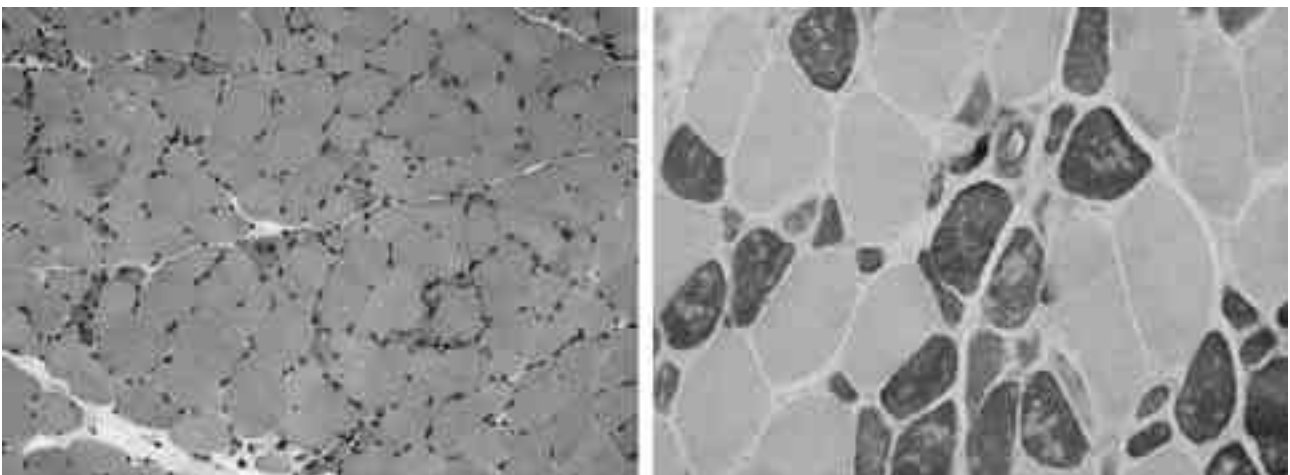
are results of ischemia associated with hypermyoglobinemia, hyperCKemia or alcohol withdrawal seizures because the patterns of images resemble the one of watershed infarction. But



**Figure 1.** Diffusion and FLAIR images of brain MRI showed diffuse multiple high signal intensities in bilateral frontoparietal cortical and subcortical areas mimicking cerebral infarctions.



**Figure 2.** Follow up images after 3 days showed decreased amounts of the abnormal high signal intensities on the same regions.



**Figure 3.** Pathological findings from right biceps muscle showed severe atrophic changes and size variations of type I muscle fibers (H&E,  $\times 100$ , ATPase 9.4,  $\times 200$ ).

abnormal signals of MRI were regressed rapidly and this fact can't explain cerebral infarctions.

Though it is extremely rare, there is another chance of transient encephalopathy associated with rhabdomyolysis, acute renal failure and hypertension<sup>7</sup>. But our patient showed no evidence of hypertension and renal failure. Even if there are some other possibilities such as direct toxic mechanism, this is remained to be resolved in the future time until meet other similar cases.

We report an unusual case of acute alcoholic myopathy superimposed on chronic alcoholic myopathy showing acute reversible encephalopathy with evidences of brain MRI and muscle pathology.

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