

Chronic Alcoholic Myopathy: An Atypical Presentation

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Case Description

Case of 48 y/o male with chronic liver disease secondary to ethanolism, who had remained abstinent for the past two months, complaining of gradual proximal weakness and generalized myalgias. He had no family history of neuromuscular diseases and workup was negative for polymyalgia rheumatica or inflammatory myopathies. Initial electrodiagnostic (EDX) study revealed a proximal subacute myopathy with increased recruitment (*Figure 1*). Follow up EDX one year after revealed a subacute to chronic condition without clinical improvement.

Discussion

Chronic alcoholic myopathy (CAM) presents in ages 40–60 and relates to cumulative long-term high-dose alcohol consumption with alcohol-related organ dysfunction. It is characterized by symptomatic improvement during subsequent months following complete alcohol abstinence. Normal insertional activity and absent denervation potentials are expected during routine EDX in view of type II muscle fiber involvement. In this case, initial EDX showed denervating potentials and increased recruitment of small/brief polyphasic motor unit action potentials (*Figures 2, 3, 4*). Subsequent EDX revealed normal to increased duration and few increased amplitude MUAPs, some which were serrated/polyphasic (*Figures 5, 6*). Persistent denervation potentials have not been documented in CAM patients

Persistent denervation potentials, have been documented in other type II myopathic conditions (e.g., severe steroid myopathy), but not in CAM. It is thought that disease involving type I fibers could explain poor clinical improvement one year after initial EDX. Findings are suggestive of a non-resolving myopathic process, therefore making this case different from previously documented literature.

EDX

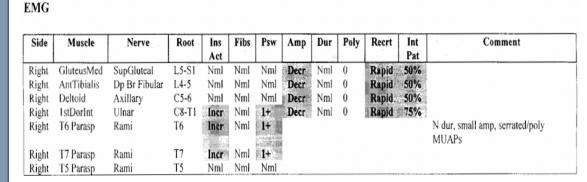
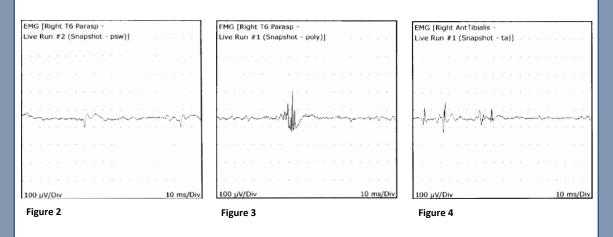
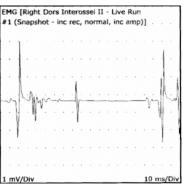


Figure 1







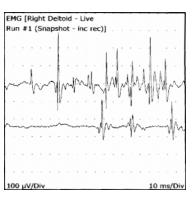


Figure 6

Conclusion

CAM is the most common clinical manifestation of alcohol use disorder frequently under diagnosed due to its variability of symptoms and normal laboratories. The precise mechanism transforming an alcohol induced reversible muscle injury into a permanent one is poorly understood. Furthermore, insufficient literature describing EDX findings in this population makes it an excellent topic for further investigation.

References

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