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Letter to the Editor

Creatine-kinase is a poor biomarker to monitor the severity and predict the outcome of COVID-19

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We read with interest the article by Friedman *et al*. about a retrospective, single-center, cross-sectional, observational, study of 289 patients admitted for confirmed SARS-CoV-2 infection, who were evaluated for the relationship between creatine-kinase (CK) levels and skeletal muscle symptoms/signs, disease severity, complications, and death [1]. Hyper-CKemia was reported in 45.7% of patients, 18% had myalgia, and 32% muscle weakness [1]. Hyper-CKemia was positively correlated with disease severity [1]. The study is appealing but raises concerns.

We disagree with the conclusions that CK can be used as an "additional data point in predicting the trajectory of the COVID-19 disease process" [1]. Hyper-CKemia is a poor biomarker for monitoring COVID-19 and predicting its outcome for several reasons. First, COVID-19 is often not a mono-organ disease but can manifest in a number of organs other than the lungs. Other organs particularly affected are the brain and the heart. Since both, heart and cerebral disease may be associated with hyper-CKemia, CK has a highly variable expression level. Second, CK levels are highly dependent on comorbidities. Therefore, those with a previous hereditary or acquired neuromuscular disorder may present to the hospital already with hyper-CKemia. Third, as in the index study, a number of patients are either already taking different myotoxic drugs at different dosages prior to enrolment or are receiving myotoxic drugs for COVID-19. These drugs may obscure the importance of CK as a biomarker of the COVID-19 progression and as an outcome parameter. Fourth, CK levels are highly dependent on a subject's physical condition. Those who exercise regularly may have chronic muscle damage that precludes reliable use of CK as a biomarker.

Surprisingly, none of the patients were diagnosed with myositis, although myositis is one the most common complications of SARS-CoV-2 infections as well as SARS-CoV-2 vaccinations [2]. Ocular myositis and dermatomyositis, both possible complications of COVID-19, were also not mentioned [3]. What is the reason for this unexpected absence?

SARS-CoV-2 can worsen a pre-existing neuromuscular disease. However, there is no discussion as to whether myasthenia remained stable during the COVID infection in the one included patient with myasthenia. It is also not mentioned whether any patient has developed new-onset myasthenia, as has been previously reported in COVID-19 patients [4].

CK may also be elevated in case of cardiac involvement. We should know whether patients with CK but without clinical muscle manifestations have been evaluated for myocarditis, Takotsubo syndrome, or myocardial infarction, which are known to be common cardiac complications of SARS-CoV-2 infection [5].

Strokes are also a common complication of SARS-CoV-2 infections and can be associated with CK-elevation. Surprisingly, none of the included patients had a stroke or venous sinus thrombosis. Were patients with a central nervous system (CNS) involvement excluded?

Four percent of enrolled patients had a history of seizures [1]. How was it ruled out that in these patients CK on admission was not due to a seizure that occurred just prior to admission?

Overall, the interesting study has limitations that challenge the results and their interpretation. CK should not be used to assess severity and outcome of COVID-19.

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