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Letters to the Editor - Comments

Comments for better understanding of the study clinical characteristics and prognostic factors in COVID-19 patients aged ≥80 years

Dear Editor,

We have read with interest the original article from authors Covino et al. entitled “Clinical characteristics and prognostic factors in COVID-19 patients aged ≥80 years.” The authors aimed to describe the clinical presentation of patients aged ≥80 years with coronavirus disease 2019 (COVID-19), and provide insights regarding the prognostic factors and the risk stratification in this population. Consequently, the authors conclude that the risk of death could not be age dependent in patients aged ≥80 years, whereas severe dementia emerged as a relevant risk factor for death in this population. However, we have some comments related to this study.

“Severe Dementia” was not defined in the methodology. The comparison was made between patients with severe dementia where two survived and six died, which was found statistically significant. However, the study found only eight of 69 (11.6%) patients with dementia. Importantly, we should not ignore 17 of 23 (74%) patients who had died because of other comorbidities without dementia.

The study concluded that risk of death could not be age dependent in patients aged ≥80 years. On the contrary, the case fatality rate of COVID-19 increases with old age.1 In addition, the presence of comorbidities was found associated with greater disease severity and poor clinical outcomes, hence risk increases with the number of comorbidities a patient has.3 While comparing comorbidities, the authors found there was no statistically significant difference between patients with COVID-19 that survived versus those that died. Age along with the number of comorbidities with duration and severity would have played an important role in the primary endpoint of the study, i.e., death.

One statement of confusion was that dementia was an independent risk factor for death in the discussion yet was a relevant risk factor in the conclusion. For an identical age group (≥80 years) using a study with this small sample size, severe dementia probably may not be the independent risk factor for death due to COVID-19, but definitely it may act as an independent risk factor if a study includes patients from all age groups, controlling for all possible risk factors.4

It would have been better if the study could include more descriptive statistics for statistically significant variables found in the patients who died. It might have helped us to understand more about the prognostic factors among elderly patients with COVID-19.

Disclosure statement

The authors declare no conflict of interest.

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Reply to comments on “Is sarcopenia primarily age- or renin–angiotensin system-related disorder?”

Keywords: age-related organ dysfunction, renin–angiotensin system, sarcopenia.

Dear Editor,

I wish to thank Gurcay and Kara1 for taking an interest in my recent review2 and I appreciate their constructive comments. They support a chain reaction among muscle loss with aging, physical inactivity and overactivity of the renin–angiotensin system (RAS). I strongly agree with this concept because RAS basically works as a thrifty hormone that regulates water and sodium balance in the body; however, longevity and satiation in the present day induce an imbalance of sodium retention, resulting in lifestyle-related diseases and age-related organ dysfunction. Their comments strengthen the relation between the RAS and age-related organ dysfunction (“senescence”), including sarcopenia and cognitive decline. In particular, in sarcopenia, the authors’ comments constructively support my review.2 I also propose that we should pay attention to the SPRINT-MIND study in which intensive blood pressure lowering management showed a significant reduction in the risk of probable dementia3 and a smaller increase in cerebral white matter lesion volume,4 in addition to the authors’ comments on the blood pressure lowering effects on brain senescence.

Moreover, the authors’ proposal of measuring total rather than regional muscle mass in sarcopenia to assess the control of RAS-related disease and evaluate the preventive effect of RAS blockade is also very interesting. Effective management of RAS in the elderly may contribute to prevention of age-related disease; however, to date we do not know the detailed activation of the systemic RAS or local tissue RAS clinically, particularly in skeletal muscle. I also totally agree with the authors’ proposal to investigate the relation between the RAS and muscle loss. I appreciate again the authors’ suggestions.

Disclosure statement

The authors declare no conflict of interest.

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