Neurocrit Care
https://doi.org/10.1007/s12028-024-02103-1

NEUROCRITICAL CARE SOCIETY

LETTERS TO THE EDITOR

Letter to the Editor: Acute Skeletal Muscle Wasting is Associated with Prolonged Hospital Stay in Critical Illness with Brain Injury

Kertee Goswami[1*], Lata Kumari[2] and Muhammad Maaz[3]

To the Editor,

We are writing in response to your recent article "Acute Skeletal Muscle Wasting Is Associated With Prolonged Hospital Stay in Critical Illness With Brain Injury" by Kangalgil et al. [1]. This study must be complimented for drawing attention to muscle wasting in relation to prolonged hospital stay. This article provides details about acute muscle loss in hospitalized patients with brain injury.

It is remarkable that the study has stressed dietary assessment, including daily energy and protein adequacy. Consequently, we consider that the study should have included the protein turnover in hospitalized patients in critical illness with brain injury. Proteolysis surpasses protein synthesis, leading to a net catabolic effect [2]. There are four crucial systems that play significant roles in protein degradation in skeletal muscle atrophy. These are the ubiquitin protease system, the cysteine aspartate protease system, the autophagy-lysosome system, and the calcium-dependent calpain system [3, 4].

In addition, a physiological principle is applied to skeletal muscles: "use it or lose it". Reduced muscle activity in patients with brain injury leads to muscle wasting and metabolic disturbance [5]. The study did not emphasize the impact of disuse atrophy in hospitalized patients. Classic models of muscle atrophy due to disuse include bed rest, joint immobilization, mechanical ventilation, spaceflight, and denervation [6]. Muscle atrophy is primarily due to protein imbalance; thus, the loss of muscle strength results in a reduction two times its size [7]. For treating disuse atrophy, exercise and amino acid supplements stand out as the best modality of management [5].

Furthermore, the study has not mentioned the correlation of the urea to creatinine ratio (UCR) in evaluation of muscle mass wasting. This ratio can provide a biochemical marker for assessment of muscle breakdown in patients with prolonged hospital stay due to brain injury [8]. Physiologically, the UCR holds a strong base. In a catabolic state, urea synthesis increases, and because of muscle wasting, creatinine levels fall. Hence, both move in opposite directions, leading to an overall increase [9]. Raised levels of the UCR run parallel with muscle atrophy; hence, it can serve crucially beneficial in determination of muscle catabolism [8].

Moreover, this study has emphasized the use ultrasonography for measurement of the cross-sectional area of the rectus femoris, whereas other techniques, such as computerized tomography (CT) and magnetic resonance imaging (MRI), can also be used. Even though ultrasonography is a noninvasive, easy, and quick technique, it only manages to measure muscle area and thickness and not muscle mass. Muscle thickness underestimates muscle loss as compared with a cross-sectional area. CT scans and MRI both measure fat-free skeletal muscle, but CT scans also provide evaluation of deep muscles. The results of both techniques are highly accurate and reliable, thus conclusively serving as a much appreciable diagnostic measure [10].

*Correspondence: kerteegoswami@gmail.com
[1] Liaquat University of Medical and Health Sciences, Bungalow No#2, Doctors Colony, Jamshoro, Pakistan
Full list of author information is available at the end of the article

This letter is in regard to the original article available at https://link.springer.com/article/10.1007/s12028-024-02017-y. The response to this letter is available online at https://link.springer.com/article/10.1007/s12028-024-02104-0.

Springer

Thus, having the aspects addressed in the prior discussion, one can indeed find enhancements to the overall efficacy of the article concerned in this letter.

Author details
[1] Liaquat University of Medical and Health Sciences, Bungalow No#2, Doctors Colony, Jamshoro, Pakistan. [2] Gambat Institute of Medical Sciences, Gambat, Shop No# 7/1 Al-Rahim Villaz, Pakistan. [3] Shaheed Mohtarma Benazir Bhutto Medical College Liyari, Flat No. B-16, Hasham Apartment, Rafa-e-aam, Malir Halt, Karachi, Pakistan.

Author contributions
Kertee Goswami: concept, drafting, and approval of final version. Lata Kumari: literature search and approval of final version. Muhammad Maaz: editing and approval of final version.

Source of support
None.

Conflict of interest
The authors declare no conflicts of interest.

Publisher's Note

Received: 13 July 2024 Accepted: 8 August 2024
Published online: 23 August 2024

References

1. Kangalgil M, Ulusoy H, Ayaz S. Acute skeletal muscle wasting is associated with prolonged hospital stay in critical illness with brain injury. Neurocrit Care. 2024. https://doi.org/10.1007/s12028-024-02017-y.
2. McCarthy JJ, Esser KA. Anabolic and catabolic pathways regulating skeletal muscle mass. Curr Opin Clin Nutr Metab Care. 2010;13(3):230–5.
3. Lad H, Saumur TM, Herridge MS, Dos Santos CC, Mathur S, Batt J, et al. Intensive care unit-acquired weakness: not just another muscle atrophying condition. Int J Mol Sci. 2020;21(21):7840.
4. Pasiakos SM, Carbone JW. Assessment of skeletal muscle proteolysis and the regulatory response to nutrition and exercise. IUBMB Life. 2014;66(7):478–84.
5. Pang X, Zhang P, Chen X, Liu W. Ubiquitin–proteasome pathway in skeletal muscle atrophy. Front Physiol. 2023;14:1,289,537.
6. Bodine SC, Edward F. Adolph Distinguished Lecture. Skeletal muscle atrophy: multiple pathways leading to a common outcome. J Appl Physiol. 2020;129(2):272–82.
7. Sirago G, Pellegrino MA, Bottinelli R, Franchi MV, Narici MV. Loss of neuromuscular junction integrity and muscle atrophy in skeletal muscle disuse. Ageing Res Rev. 2023;83: 101810.
8. Haines RW, Zolfaghari P, Wan Y, Pearse RM, Puthucheary Z, Prowle JR. Elevated urea-to-creatinine ratio provides a biochemical signature of muscle catabolism and persistent critical illness after major trauma. Intensive Care Med. 2019;45(12):1718–31.
9. Gunst J, Kashani KB, Hermans G. The urea-creatinine ratio as a novel biomarker of critical illness-associated catabolism. Intensive Care Med. 2019;45(12):1813–5.
10. Vanhorebeek I, Latronico N, Van den Berghe G. ICU-acquired weakness. Intensive Care Med. 2020;46(4):637–53.