

In response to the article, 'Interrelationship of Sarcopenia and Cardiovascular Diseases: A Review of Potential Mechanisms and Management,' by Rivera, et al., published in JAFES Vol. 39 No. 1.

## Sarcopenia in Patients with Cardiovascular Disease Can Also Be Triggered by Other Risk Factors

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We read with interest the review article by Rivera et al. on the influence of cardiovascular disease on the development of sarcopenia.<sup>1</sup> The aim of the review was to explore the pathophysiological mechanisms that explain the relationship between sarcopenia and cardiovascular disease, as well as its clinical assessment and associated management.<sup>1</sup> Inflammation, oxidative stress, endothelial dysfunction, neuronal and hormonal changes and other metabolic disturbances have been identified as factors responsible for sarcopenia in patients with cardiovascular disease.<sup>1</sup> The overview is impressive, but some points still need to be discussed as several causes of sarcopenia associated with cardiovascular disease were not considered in the review.<sup>1</sup>

The first point is that there is evidence that sarcopenia is associated with low hemoglobin levels, so patients with sarcopenia should also be screened for anemia.<sup>2</sup> Other studies also indicate that anemia is associated with sarcopenia, so it has been suggested that these patients should switch to a diet rich in antioxidants, high-quality proteins and micronutrients, as well as engage in moderate physical activity and maintain a healthy weight.<sup>3</sup> Sarcopenia can also occur in survivors of severe aplastic anemia treated after allogeneic hematopoietic stem cell transplantation.<sup>4</sup> The effects of anemia on skeletal muscle can be exacerbated by heart failure.

The second point is that arterial hypotension or lowering blood pressure with beta-blockers was not mentioned in the review as being associated with sarcopenia.<sup>5</sup> Inadequate blood supply to skeletal muscle due to chronic hypotension

may reduce skeletal muscle mass and lead to progression of sarcopenia, as adequate blood flow is required to maintain skeletal muscle mass.<sup>6</sup> One factor that may contribute to the development of sarcopenia in this context is low capillarization of skeletal muscle, which leads to restricted diffusion of substrates, oxygen, hormones and nutrients.<sup>6</sup>

The third point is that medications that are frequently taken over a long period of time have not been discussed in detail as a cause of sarcopenia. These include statins, which are known to cause myopathy as a side effect in about one percent of cases, sulfonylureas and glinides.<sup>7</sup> There is also evidence that GLP-1 receptor agonists, SGLT2 inhibitors, glucocorticoids, antineoplastics, immune checkpoint inhibitors, antiandrogens, chloroquine/hydroxychloroquine, colchicine, nucleoside analogs, loop diuretics and D-penicillamine can cause sarcopenia.<sup>8</sup>

The fourth point is that hemodialysis, which is often associated with cardiovascular disease, was not mentioned in the review as a recognized cause of sarcopenia.<sup>9</sup> Sarcopenia in patients with end-stage renal disease undergoing maintenance hemodialysis can be explained by accelerated protein depletion, multiple metabolic disturbances and nutrient deficiencies.<sup>10</sup>

Fifth, we disagree with the statement that beta-blockers have a beneficial effect on sarcopenia.<sup>1</sup> On the contrary, several studies have found that beta-blockers are associated with an increased risk of sarcopenia.<sup>5</sup> Another argument in favor of the sarcopenic effect of beta-blockers is that at least some beta-agonists increase muscle mass and strength.

In summary, it can be said that this interesting review has some limitations that relativize the results and their interpretation. Taking these limitations into account could strengthen the conclusions and increase the validity of the review. The causes of sarcopenia in cardiovascular disease are multifactorial and may be exacerbated or caused by genetic influences, immobility or disuse, hematologic diseases, medications and treatments such as dialysis, endocrine factors, inflammation, and nutritional deficiencies.

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