LETTER

Serum 25-hydroxy vitamin D and exercise capacity in COPD

Janssens and colleagues\(^1\) have recently reported that vitamin D deficiency is very common in patients affected by chronic obstructive pulmonary disease (COPD) and that vitamin D status correlates with lung function. In the same issue of Thorax, Quint and Wedzicha\(^2\) discuss potential effects of vitamin D deficiency and supplementation in COPD with special focus on immunomodulatory function. However, they do not consider a potential impact of the hormone on muscle mass and function, and consequently on exercise capacity in these patients.\(^3\)

Since exercise limitation is a very common complaint and a significant contributor to the poor quality of life in COPD,\(^4\) we studied the relationship between maximal aerobic capacity (VO\(_2\) peak) evaluated by an incremental bicycle ergometry until exhaustion, circulating levels of 25(OH) vitamin D and respiratory function (forced expiratory volume in 1 s, FEV\(_1\); carbon monoxide transfer factor in a single breath method, TLCO) in a cohort of 79 stable male COPD patients (table 1).

Serum 25 (OH)D levels below the lower limit of the normal range (50 ng/ml) were found in 50 patients (63.3%), values between 30 and 12 ng/ml in 24 (30.4%) and values below 12 ng/ml in 26 patients (32.9%). In agreement with Janssens et al\(^1\), we report a correlation between 25(OH)D levels and FEV\(_1\) (r=0.504, p<0.001), taking into account the differences in age, body weight and height. Further, 25(OH)D correlated with TLCO (r=0.496, p<0.001) and VO\(_2\) peak (r=0.247 p<0.05). A stepwise linear regression analysis, using VO\(_2\) peak as outcome measure and 25(OH)D levels, FEV\(_1\) (% of predicted), TLCO (% of predicted), age, weight, and height as possible determinants, revealed 25(OH)D, TLCO and age and body weight to be significantly and independently associated with exercise capacity (R\(^2\)=0.567, SEE=0.418 l/min, p<0.001). In other words, our data indicate that low serum 25(OH)D levels are associated with poor exercise capacity in COPD while the degree of resting airflow limitation does not significantly add to the accuracy of the prediction of VO\(_2\) peak in this model.

In conclusion, we agree with Quint and Wedzicha that attention should be paid to the futility of D hormone supplementation in COPD patients. However, a more holistic approach claims to consider muscle health and exercise capacity as further potential targets of D hormone treatment in this multidimensional, disabling and progressive disease.

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