Differences in Handgrip Strength Between Statin Users and Non-users in Older Patients with Heart Failure. An Observational Retrospective Study of the PROFUND-IC Registry

Original Article

Lucía Gandía-Blanquer[1], Paula Hernández-Sanjuán[1], Rebeca Fuerte-Martínez[1], Gonzalo Serralta-San Martin[1], Jorge F. Gómez-Cerezo[1], Fernando Aguilar-Rodriguez[2], Manuel Méndez-Bailón[3] and Llanos Soler-Rangel[1]

[1] *Internal Medicine Service. Infanta Sofia University Hospital, Foundation for Biomedical Research and Innovation of the Infanta Sofia University Hospital and Henares University Hospital (FIIB HUIS HHEN), Madrid, Spain*
[2] *Internal Medicine Service. University Hospital 12 of October. Madrid, Spain*
[3] *Internal Medicine Service. Hospital Clínico San Carlos. Institute of Health Research Hospital Clínico San Carlos (IdISSC). School of Medicine. Complutense University of Madrid, Spain*

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Abstract—Introduction: Heart failure is a chronic disease associated with different cardiovascular diseases. The management of this disease is based on the control of cardiovascular risk factors, in some cases, using different drugs, such as statins in the treatment of dyslipidemia. However, there are not enough studies evaluating the influence of statin use on muscle strength. Since frail elderly people may be more vulnerable to the side effects of statins, it would be of interest to evaluate the possible association between the use of statins and loss of muscle strength in patients with heart failure. **Patients and Methods:** We conducted an observational retrospective study including patients from the PROFUND-IC registry, which studied pluripathological patients hospitalized for acute heart failure. We described the different characteristics between statin users and non-users and compared handgrip strength between both groups. **Results:** A total of 476 patients were included, of whom 53.2% (253) had a median age of 84 years and were on statin therapy. Handgrip strength was pathological in 81% of statin users compared to 84.7% of non-users. Multivariate analysis showed no differences in handgrip strength between statin users and non-users, with an overall OR value of 1.036 (p = 0.94). **Conclusion:** Our study does not show an association between the use of statins and worse handgrip strength in older patients with heart failure. Therefore, frailty or older age should not be a limitation for the prescription of statins. **Rev Med Clin 2025;9(1):e28032509006**

Keywords—Heart failure, Statin, Muscle strength, Frailty, Older adult

Resumen—Diferencias en la Fuerza de Prensión Manual Entre Usuarios y No Usuarios de Estatinas en Pacientes Mayores con Insuficiencia Cardiaca: Un Estudio Observacional Retrospectivo del Registro PROFUND-IC
Introducción: La insuficiencia cardíaca es una enfermedad crónica asociada a diferentes enfermedades cardiovasculares cuyo manejo se basa en el empleo de diferentes fármacos como las estatinas. Sin embargo, no hay suficientes estudios que evalúen la influencia del uso de estatinas en la fuerza muscular. Dado que las personas mayores frágiles pueden ser más vulnerables a los efectos secundarios de las estatinas, es interesante evaluar la asociación entre el uso de estatinas y la pérdida de fuerza muscular en pacientes con insuficiencia cardíaca. **Pacientes y Métodos:** Se realizó un estudio observacional retrospectivo incluyendo pacientes del registro PROFUND-IC, que estudió pacientes pluripatológicos hospitalizados por insuficiencia cardíaca aguda. Describimos las diferentes características entre los usuarios y no usuarios de estatinas y comparamos la fuerza de prensión manual entre ambos grupos. Resultados: Se incluyeron 476 pacientes, de los cuales el 53.2% (253) tenían una mediana de edad de 84 años y consumían estatinas. La fuerza de prensión manual fue patológica en el 81% de los usuarios de estatinas en comparación con el 84.7% de los no usuarios. El análisis multivariante no mostró diferencias en la fuerza de prensión manual entre usuarios y no usuarios de estatinas, con un valor OR general de 1.036 (p = 0.94). **Conclusión:** Nuestro estudio no muestra una asociación entre el uso de estatinas y una peor fuerza de prensión manual en pacientes mayores con insuficiencia cardiaca. Por tanto, la fragilidad o la edad avanzada no deben ser una limitación para la prescripción de estatinas. **Rev Med Clin 2025;9(1):e28032509006**

Palabras clave—Insuficiencia cardaca, Estatina, Fuerza muscular, Fragilidad, Anciano

INTRODUCTION

Heart failure (HF) is a clinical syndrome triggered by a structural or functional cardiac abnormality that results in elevated intracardiac pressures or inadequate cardiac output at rest or during exercise.[1]

It is a chronic disease with an incidence in Europe of 5 per 1,000 person-years in adults, with a prevalence of 1-2%. The prevalence of HF increases progressively with age, from 1% in patients under 55 years old to over 10% in patients over 70 years old. This disease has become one of the main causes of hospital admission in the elderly population, placing a significant burden on the health system.[1]

This clinical syndrome is associated with numerous comorbidities, such as diabetes mellitus, dyslipidemia, or hypertension, and therefore, with underlying cardiovascular diseases.[2] Part of the management of HF involves controlling cardiovascular risk factors, sometimes through the use of different drugs, such as statins in the treatment of dyslipidemia.

Statins have been shown to reduce apoptosis and oxidative stress in myocardial cells, consequently reducing the loss of cardiomyocytes.[3] According to a study by Douglas et al., treatment with statins for one month reduced the production of reactive oxygen species in patients with HF with reduced ejection fraction, improving endothelial function and functional capacity.[4] Another study by Andreou et al. also supports this fact, demonstrating that statins reduce myeloperoxidase levels after one month of treatment in patients with systolic HF.[5]

However, the use of statins in HF patients remains controversial, as it has not independently demonstrated to improve the prognosis of this disease. In fact, while statins have been shown to prevent the development of HF, they do not reduce morbidity and mortality in these patients once the disease is established, where the main cause of death is pump failure and ventricular arrhythmia.[6]

Patients with HF often develop a state of malnutrition and cachexia, and hypocholesterolemia is not uncommon. Low total and LDL cholesterol levels have been consistently associated with increased mortality in patients with established HF.[7] In fact, the latest European Society of Cardiology guidelines on the clinical management of patients with HF do not recommend the regular use of statins in patients with HF without other indications for their use.[8]

Contact data: Lucia Gandia-Blanquer, Hospital Universitario Infanta Sofia Paseo de Europa, 34 28702 San Sebastian de los Reyes (Madrid)., Phone number: (+34) 627816424, lucia.gandia@salud.madrid.org

The use of statins has traditionally been associated with the development of sarcopenia,[9] which may limit their efficacy in HF patients, especially considering that HF and sarcopenia share pathophysiological pathways that lead to the onset of muscle dysfunction. Ahmad et al. found that patients with HF on statins had worse physical performance than those not on statins, suggesting a potential adverse effect of statins on physical performance in HF patients.[10]

However, some newer studies suggest that statins are consistently associated with a lower risk of sarcopenia, as the benefits of statins at the circulatory level would positively affect the neuromuscular system and, consequently, the maintenance of muscle strength.[11] Despite the increasing aging of the population due to improvements in life expectancy, there is an underrepresentation of frail elderly patients in these studies conducted to date. In fact, the scarcity of studies implies a lack of evidence to initiate or continue statins in elderly patients with HF.[12] Similarly, there are not enough studies evaluating the effect of statin consumption on parameters that influence the prognosis of these patients, such as muscle strength, frailty, and malnutrition.[13] We consider whether treatment with statins could lead to sarcopenia, loss of strength, or worse physical performance and how this could affect their potential benefit in HF.

Moreover, since frail elderly individuals may be more vulnerable to the side effects of statins, it is of interest to evaluate the association between the use of statins and loss of strength, especially in fragile HF patients, who are underrepresented in clinical trials.

We conducted this study to describe the characteristics between statin users and nonusers in older adults with heart failure and to determine the differences in handgrip strength.

PATIENTS AND METHODS

Patients

We conducted an observational retrospective multicenter study based on the PROFUND-IC registry of the Spanish Society of Internal Medicine. It included patients admitted for acute heart failure as the main diagnosis in the internal medicine services of 18 hospitals in Spain between September 2020 and May 2022.

The inclusion criteria used in PROFUND-IC registry were age > 18 years, main diagnosis of acute HF, and NT-proBNP on admission > 1500 pg/mL, and multimorbidity defined as two or more chronic diseases: Heart failure (NYHA II in clinical stability), ischaemic heart disease, vasculitis and systemic autoimmune diseases, chronic kidney disease, chronic respiratory disease, chronic inflammatory bowel disease, chronic liver disease, stroke, neurological disease with permanent motor impairment, neurological disease with permanent cognitive impairment, symptomatic peripheral artery disease, diabetes mellitus with proliferative retinopathy or symptomatic neuropathy, chronic anemia, active solid or hematologic neoplasia, chronic osteoarticular disease.

Patients with active COVID-19 infection were excluded.

The study protocol was approved by the Clinical Research Ethics Committee of the Hospital Fundación Alcorcon, and informed consent was obtained from all patients prior to inclusion. The investigators of this study adhered to the recommendations of the Declaration of Helsinki, and its subsequent modifications, for the protection of the participants.

For the present study, we analyzed patients over 50 years who met all the other inclusion criteria (main diagnosis of acute HF, and NT-proBNP on admission > 1500 pg/mL, and multimorbidity defined as two or more chronic diseases) and for whom information about statin use was available. We described the different characteristics between statin users and non-users, as well as comparing handgrip strength levels between both groups.

Variables

The primary outcome variable was the determination of handgrip strength in statin users compared with nonusers in HF patients. A Jamar hydraulic hand-held dynamometer was used to measure grip strength in the non-dominant hand with the arm positioned at a 90° angle. It was measured three times, when the patient was stable before hospital discharge, choosing the best result. As values of grip strength vary depending on different factors, we considered pathological strength values below 27 kg in men and 16 kg in women, based on a study that describes different grip strength values considering age, gender, and multimorbidity.[14]

Demographic variables such as sex and age were recorded, as well as clinical variables such as comorbidities (hypertension, dyslipidemia, diabetes mellitus, chronic obstructive pulmonary disease, obstructive sleep apnea, atrial fibrillation, ischemic heart disease, stroke), toxic habits (smoking, alcohol), HF etiology (hypertensive, ischemic, dilated cardiomyopathy, valvular, or amyloidosis), left ventricular ejection fraction (LVEF), New York Heart Association (NYHA) functional class, date of diagnosis of HF, and number of hospital admissions before the current admission. Fragility and nutritional status were assessed by Barthel index, MNA test (Mini Nutritional Assessment), Short Physical Performance Battery scale (SPPB), and Profund Index. Analytical variables included NT-proBNP, hemoglobin, lymphocyte count, creatinine, serum sodium, serum potassium, total cholesterol, LDL cholesterol, albumin, urinary sodium, and potassium. Treatments received during admission and at discharge, including statins, were collected.

Statistical Analysis

All data were analyzed using IBM® SPSS® Statistics V25.0 for Windows®. For the description of the qualitative variables, absolute and relative frequencies were used for each category. Quantitative variables were expressed as mean and standard deviation if they had a normal distribution, or as median and interquartile intervals (IQI) if the distribution did not meet the criteria for normality. Normality of a distribution was checked by frequency histogram and the Kolmogorov-Smirnov test. Quantitative variables were compared by Student's t-test or Mann-Whitney U test according to whether the sample distribution was normal or not. Qualitative variables were compared by Chi-square test. The strength of association was expressed as odds ratio (OR) with 95% confidence interval (95% CI). For this purpose, a univariate analysis of the outcome variable between statin users and nonusers was performed, followed by a multivariate analysis. The level of statistical significance established was $p<0.05$.

RESULTS

Patients

A total of 476 patients were included in the study, with a median age of 86 years and 59% being women. Table 1 displays the characteristics of statin users and nonusers. Of the total, 53,15% (253) were on statin therapy, with a median age of 84 years. Statin users exhibited more cardiovascular risk factors such as diabetes, hypertension, ischemic heart disease, and stroke. Pathological handgrip strength was observed in 81% of statin users compared to 84.7% of nonusers.

Comparison between participants with normal and pathological handgrip strength

Table 2 presents the characteristics of patients with normal and pathological handgrip strength. We observed that patients with low handgrip strength were older than those with normal handgrip strength ($p<0.001$), with no differences by gender. Furthermore, patients diagnosed with heart failure more than 18 months before the current admission, those classified as NYHA III-IV and individuals who exhibited poorer scores in the various measured tests (SPPB, Barthel, Profund) demonstrated further reductions in handgrip strength.

Univariate analysis

In the univariate analysis (Table 3), factors associated with pathological handgrip included a diagnosis of heart failure more than 18 months before the current admission, number of hospital admissions for HF in the last year, NYHA class III-IV, age, smoking, reduced LVEF, use of beta-blockers, use of corticosteroids, MNA score, Barthel index and Profund index.

Multivariate analysis

Although crude OR indicated a non-significant tendency towards higher handgrip strength in statin users, this finding was not confirmed by multivariate analysis adjusted for these variables, considered as confounding factors (Table 4). Therefore, we concluded that there is no difference in handgrip strength between statin users and nonusers, with an overall OR value of 1.036 ($p=0.94$).

DISCUSSION

Heart failure is a chronic disease characterized by several modifications, including malabsorption, malnutrition,

Variable*	Statins (n=253)	Non-statins (n=223)	p= μ
Age, Md (IQR)	84.5 (77.9 - 88.8)	87.5 (83.2 - 91.6)	<0.001
Sex,n(%)			0.140
Men	111 (43.9)	83 (37.0)	
Women	142 (56.1)	140 (62.8)	
Diabetes mellitus, n(%)	130 (51.4)	76 (34.9)	<0.001
Dyslipidemia, n(%)	213 (84.2)	77 (34.5)	<0.001
Hypertension, n(%)	237 (93.7)	194 (87)	0.013
Smoking, n(%)	17 (6.7)	6 (2.7)	0.024
Cholesterol, Md (IQR)	129 (109 - 155)	145 (123 - 176)	<0.001
LDL Cholesterol Md (IQR)	67 (50 - 85.5)	82 (65 - 103)	<0.001
COPD, n(%)	57 (22.5)	43 (19.3)	0.396
OSA, n(0%)	56 (22.1)	28 (12.6)	0.006
Atrial Fibrillation, n(%)	75 (69.2)	159 (71.3)	0.612
Ischemic heart disease, n(%)	97 (38.3)	32 (14.4)	<0.001
Stroke, n(%)	58 (23)	27 (12.2)	0.002
NYHA, n(%)			0.306
I-II	138 (55)	133 (59.6)	
III-IV	113 (45)	90 (40.4)	
LVEF, n(%)			0.762
<40	39 (15.6)	33 (15)	
40-49	42 (16.8)	32 (114.5)	
$\geq$ 50	169 (67.6)	155 (705)	
Phatological Handgrip Strenght, n(%)	136 (81)	111 (84.7)	0.392
SPPB, n(%)			0.213
0-6 (moderate - severe)	165 (73.7)	153 (78.9)	
7-12 (minumum - medium)	59 (26.3)	41 (21.1)	
Barthel, Md (IQR)	85 (55 - 95)	70 (45 - 95)	0.010
MNA, Md (IQR)	11 (10 - 12)	10 (8 - 12)	0.006
Profund Index, Md (IQR)	5 (3 - 9)	7 (3 - 10)	0.033
IECA, n(%)	64 (25.3)	41 (18.4)	0.070
ARA II, n(%)	68 (26.9)	42 (18.9)	0.040
ARNI, n(%)	30 (11.9)	14 (6.3)	0.035
Beta-Blockers, n(%)	170 (67.2)	129 (57.8)	0.035
Antagonists of Aldosterone, n(%)	97 (38.3)	76 (34)	0.616
Diuretics, n(%)	242 (95.6)	198 (88.8)	0.012
iSGLT2, n(%	96 (38)	69 (31)	0.618
Corticosteroids, n(%) 59 (23.3)	51 (22.9)	0.934	
Intravenous Iron, n(%)	69 (27.5)	69 (31.5)	0.340

Table 1: Baseline Characteristics for Participants (n=476).
*Absolute and relative frequencies were used to describe the qualitative variables (n,%). Quantitative variables were expressed as median (Md) and interquartile range (IQR) since all of them had a non-normal distribution. The normality of a distribution was tested using the Kolmogorov-Smirnov test. μ For the comparison of variables, the Chi-square test and the Mann-Whitney U test were used when the distribution was non-normal. The level of statistical significance established was p<0, 05.

inflammation, oxidative stress, apoptosis, endothelial dysfunction, physical inactivity, and low muscle blood flow, among others. All of these factors may contribute to the development of loss of muscle strength in HF patients.[15]

On the other hand, there has been a traditional belief that the use of statins is associated with the development of muscle weakness, leading to questioning their use in patients with heart failure.[9]

It has been reported that statins cause symptomatic adverse events in up to approximately 50-100 patients per 10,000 treated for 5 years. In a review by Collins et al., they estimated that the treatment of 10,000 patients with statins for 5 years would cause about 5 cases of myopathy. However, randomized placebo-controlled trials have shown that most adverse events attributed to statins in usual practice are not actually caused by them. In our study, it was found that statins also seem to be less frequently prescribed in frail or dependent patients, likely due to concerns about their side effects and the lack of knowledge about the benefit on prognosis in these patients.[16]

In our study, we found no difference in handgrip strength between statin users and nonusers in patients with heart failure (OR=1.036 (IC95% 0.38-2.82); p=0.94). This fact does

Variable*	Patients with normal handgrip strength	Patients with pathological handgrip strength	p=
Age, Md (IQR)	79.6 (73.3 - 85.9)	85.5 (80.3 - 89.9)	<0.001
Sex,n(%)			0.0.309
Men	33 (49.3)	123 (42.4)	
Women	34 (50.7)	167 (57.6)	
COPD, n(%)	15 (22.7)	61 (20.7)	0.772
HF diagnosis > 18 months before current admisions, Md (IQR)	30 (45.5)	200 (68)	<0.001
N° hospital admissions, Md (IQR)	1 (1 - 2)	1 (1 - 2)	0.021
NYHA III-IV, n(%)	22 (33.3)	161 (54.8)	0.002
LVEF, Md (IQR)	50 (39 - 58)	54 (48 - 60)	0.010
SPPB, Md (IQR)	5 (2 - 9)	2 (0 - 5)	<0.001
Barthel, Md (IQR)	95 (80 - 100)	70 (45 - 95)	<0.001
MNA, Md (IQR)	11 (10 - 14)	10 (9 - 12)	0.021
Profund Index, Md (IQR)	3 (2 - 5)	7 (3 - 11)	<0.001
N° drugs, Md (IQR)	10 (8 - 13)	11 (8 - 14)	0.902
Beta-Blockers, n(%)	52 (81.5)	175 (70.8)	0.002
Statins, n(%	32 (61.5)	139 (55.4)	0.415
Corticosteroids, n(%) 9 (13.6)	85 (29.1)	0.010	

Table 2: Descriptive analysis of patients with normal handgrip strength versus pathological handgrip strength.
*Absolute and relative frequencies were used to describe the qualitative variables (n, %). Quantitative variables were expressed as median (MD) and interquartile range (IQR) since all of them had a non-normal distribution. The normality of a distribution was tested using the Kolmogorov-Smirnov test.

Variable	OR (IC95%)	Sig.
Statins	0.77 (0.42-1.141)	0.393
Sex, Mex (ref)	1.32 (0.77-2.25)	0.310
Smoking	0.51 (0.34-0.79)	0.002
COPD	0.89 (0.47-1.69)	0.714
NYHA III-IV	2.39 (1.37-4.320)	0.002
Reduced LVEF	0.46 (0.26-0.80)	0.005
Marthel (Moderate/severe /total dependence)	3.75 (2.16-6.53)	<0.001
Malnutrition/malnutrition risk (MNA)	1.56 (0.72-3.36)	0.256
PROFUND index	1.25 (1.16-1.36)	<0.001
N° drugs	1.00 (0.93-1.07)	0.948
Bleta-blockers	0.35 (0.18-0.68)	0.002
Forticosteroids	2.56 (1.21-5.42)	0.014

Table 3: Univariate Analysis for pathological handgrip strength.

not support the hypothesis that statins are related to lower muscle strength in patients with heart failure. Therefore, it appears that muscle strength is equally affected among heart failure patients, regardless of statin use.

As expected, our results suggest that statins are more frequently prescribed in patients with cardiovascular risk factors, in addition to HF itself. However, the role of statins in heart failure remains controversial.[17,18] Statins prescription is based on the premise that c-LDL levels should be reduced as much as possible in patients with HF at high and very high cardiovascular risk. However, it is not clear that applying such strict c-LDL limits in high-risk patients with established HF will provide much benefit. In fact, there is data suggesting even worse prognosis in HF patients with low total and LDL cholesterol levels (LDL < 100).[7] This is known as the "cholesterol paradox", which means that, in patients with established atherosclerotic cardiovascular disease (but without HF), statins may reduce the risk of incident heart failure, mainly by preventing myocardial infarction. Meanwhile, in patients with established HF, statins do not reduce the risk of cardiovascular death, mostly associated with arrhythmias and cardiac pump failure.[6]

Two large clinical trials that have been carried out in patients with HFrEF (CORONA and GISSI-HF) and some meta-analysis including different trials evaluating the use of statins in patients with HF have not shown to reduce mortality or morbidity in patients with HFrEF.[8,19,20] On the other hand, it has been described that in patients with established HF, the anti-inflammatory effect of statins could improve myocardial fibrosis and cardiac filling abnormalities, producing favorable effects in patients with HFpEF (heart failure with preserved ejection fraction) but not in patients with HFrEF (heart failure with reduced ejection fraction), whose main problem is the loss and stretching of cardiomyocytes.[21,22] Currently, the regular use of statins in patients with HF without other indications for their use is not recommended.[8]

In our study, we found that statins also seem to be less frequently prescribed in frail or dependent patients, likely due to concerns about their side effects and the lack of knowledge about the benefit on prognosis in these patients.[16] We also found that patients with worse scores in strength tests were older, more dependent, and frail, with longer evolution and severity of HF and worse functional class.

	OR (IC95%)	Sig.
Crude OR	0.77 (0.42-1.41)	0.393
Adjusted OR*	1.04 (0.38-2.82)	0.944

Table 4: Analysis of the association of statins therapy and worse handgrip strength.
*Adjustment was made for the following variables: diagnosis of HF >18 months before current admission, nº admissions, NYHA III-IV, age, smoking, LVEF, beta-blockers, corticosteroids, MNA score, Barthel index and Profund index.

Handgrip strength has been considered a valid tool for measuring overall muscle function and predicting functional limitations and mortality. Age and gender have been identified as the strongest influencing factors on grip strength. However, various chronic disorders and certain medications, such as statins in the context of heart failure, may also be associated with muscle weakness strength.[14]

A study of Scott et al. which evaluated muscle performance and falls risk between statin users and nonusers with a mean age of 62 years old, showed that statin therapy may be associated with greater declines in muscle strength and falls risk in older adults. In this study, the sample of patients represented is significantly younger than that of our study, although statin users were significantly more likely to report a previous diagnosis of cardiovascular disease and diabetes, as was the case with our sample. No differences were found in muscle parameters depending on the type of statins, which reinforces the validity of our results.[23] Furthermore, a randomized clinical trial by Golomb et al. was conducted to evaluate whether the use of statins affected energy and exertional fatigue in a healthy population. This study affirms unfavorable statin effects on energy and exertional fatigue in patients with statins.[24]

In contrast, a recent study involving 136 patients with heart failure (of whom 65.4% were statin users) demonstrated a reduced likelihood of muscle weakness among statin users (OR=0.03; IC95% 0.01-0.30). In this study, the median age was 59, which is younger than the median age observed in our sample, and fewer comorbidities were reported. The discrepancy in muscle strength not being associated with NYHA classes in our study compared to theirs may stem from the inclusion of older patients diagnosed with longer evolution and greater severity of heart failure in our study, factors that could significantly influence poorer muscle strength.[12]

Other studies have reported no associations between statin use and muscular decline, in line with our findings. A study of the Hertfordshire cohort showed that the use of statins, among other drugs, was not associated with differences in reduced grip strength in healthy elderly people, although the median age of the population included was 65 years.[25] Furthermore, a randomized, controlled clinical trial designed to evaluate the effects of physical exercise on muscle performance in older adults demonstrated no discernible influence based on the treatment received, including the use of statins.[26] Another study designed to determine the relationship between statin use and proximal muscle strength, cognition, and depression in older adults, showed that statin use was not associated with significant adverse effects in muscle strength. Mean age was 74.5 years old and statin users were more likely to have cardiac, cerebrovascular, and peripheral vascular disease, a sample of patients more similar to that represented in our study. Because statins share a similar pharmacological mechanism of action, statins were grouped together in this study, which supports our results.[9]

In view of the results of our study and the evidence found, we believe that frailty and age should not be limitations for prescribing statins in patients for whom they are indicated.

As for strength of our study, it includes older pluripathological patients, a profile of patients which is not always represented in published studies and in which the risk/benefit balance of drugs that are commonly used for HF is a cause of concern. In addition, the sample was obtained from the PROFUND multicenter study, which investigates the impact of frailty and comorbidity in HF patients and has led to several publications related to this pathology. Therefore, we consider that the results obtained in our study could be extrapolated to clinical practice in most hospitals of our country.

Our study also has some limitations. The type and dose of statin administered were not recorded in the registry, so we cannot know if there is any difference in the effect of different compounds or doses on muscle strength. However, some studies have found no differences between the use of different types of statins, as we mentioned below.[23] We also do not know the influence of statins on muscle strength over time and the prognosis of HF patients because of the lack of follow-up.

Larger studies are required to discern whether there are differences in outcomes for specific statins, although to date, there is no evidence in literature demonstrating the influence of dose or type of statins on muscle strength.

As the use of statins in HF remains controversial, it would be interesting to investigate whether statin use influences the prognosis of heart failure patients through prospective studies in future research.

Conclusions

In conclusion, our study has found no differences in reduced muscular strength between statin users and nonusers. While further evidence is needed to clarify the relationship between statin use and muscle strength, we suggest that this should not limit the prescription of statins in frail elderly patients with heart failure.

Author Contributions

Conceptualization, LSR, LGB, PHS, MMB, FAR; methodology LSR, MMB, FAR, GSS; formal analysis, LSR,

LGB, PHS, SSS; investigation LSR, LGB, PHS, MMB, FAR; writing—original draft preparation, LGB, PHS, RFM; writing—review and editing, LSR, GSS, MMB, FAR, JGC. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

The study protocol was approved by the Clinical Research Ethics Committee of the Hospital Fundación Alcorcon on 11 de junio de 2019 and informed consent was obtained from all patients prior to inclusion. The investigators of this study adhered to the recommendations of the Declaration of Helsinki, and its subsequent modifications, for the protection of the participants.

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Conflicts of Interest

The authors declare no conflicts of interest.

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