lished at around 5 to 6 weeks depending on the series consulted. The treatment approaches include open surgery with resection and revascularization, endovascular stenting, or endovascular embolization. The less invasive endovascular treatments could be a useful option in complex patients.[5]

In conclusion, clinical suspicion is vital in the diagnosis of pseudoaneurysm. The main factors to avoid fatal consequences are a prompt diagnosis and early initiation of treatment, both surgical and pharmacological.

The patient provided informed consent to undergo the treatment described. The case has the approval of the Basurto Hospital Ethics Committee. The patient's data have been anonymized.

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AUTHORS' CONTRIBUTIONS

D. Fernández Vecilla: molecular diagnosis, conception, and description of the case, and literature review. M.J. Urrutikoechea-Gutiérrez: conception of the case and review of the molecular diagnosis process and scientific letter. E. Ugalde Zárraga: conception of the case and review of the molecular diagnosis process and scientific letter. M. Urizar Gorosarri: imaging diagnosis and review of the case. M.L. Rodríguez Iriarte: imaging diagnosis and review of the case. J.L. Díaz de Tuesta del Arco: review of the literature review process and the scientific letter.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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Lung ultrasound in the follow-up of subclinical pulmonary congestion in outpatients with heart failure

La ecografía pulmonar en el seguimiento de la congestión pulmonar subclínica de pacientes ambulatorios con insuficiencia cardiaca

To the Editor,

Lung ultrasound (LUS) is a simple and rapid scan that provides information on pulmonary congestion by visualizing B lines. It has become a highly useful tool in multiple clinical situations related to heart failure (HF). Although it remains to be fully characterized in patients with chronic HF,[1,2] its considerable prognostic value has been reported.[3] In these patients, a certain degree of subclinical congestion is often detected by LUS, even if the patients are clinically euvolemic, although the long-term implications of this finding are unknown.

Our objective was to use LUS to evaluate changes in pulmonary congestion in a cohort of stable outpatients with HF who attended 2 scheduled follow-up visits at least 2 months apart in a specific HF consultation. All participants gave their written informed consent for the performance of the tests and the publication of the results, and the local ethics committee approved the protocol. LUS was performed with a portable ultrasound using a scanning protocol for 8 chest areas, with masked clinical data at study initiation (LUS1) and after a mean of 4.2 ± 0.4 years (LUS2). LUS1 was performed between July 2016 and October 2017 while LUS2 was performed between October 2020 and December 2021. The B line sum of all areas and the quartiles of this sum were used in the main analysis. Pleural effusion was counted as 10 lines. We excluded patients with clinical decompensation at the time of the visit and those with a history of pulmonary fibrosis.

Of the 577 patients who underwent LUS1, 122 died during follow-up and 287 did not undergo LUS2 for various reasons: appointment rescheduling due to the pandemic, clinical congestion at the visit, or logistical aspects (researcher or ultrasound availability). Finally, 168 patients were included (mean age, 66.6 ± 11.5 years; 73.8% men). The most frequent etiology was ischemia (42.3%), followed by dilated cardiomyopathy (20.8%) and valvular cardiomyopathy (10.7%). Of the patients, 19% were in New York Heart Association functional class I and 69% were in class II. Time since diagnosis of HF was 7.6 ± 5.3 years, left ventricular ejection fraction (LVEF) was 45% ± 11.9%, and median N-terminal pro-B type natriuretic peptide (NT-proBNP) was 568 [interquartile range, 220-1198] ng/L. The number of B lines in the initial lung ultrasound (LUS1) was 2.9 ± 3.8 (median, 2 [0-4]) but was 3.1 ± 3.3 (median, 2 [0-5]; $P = .51$) at follow-up.

The drug therapies at the initial visit and in the follow-up assessment are shown in table 1. The number of patients with loop

Table 1
Changes in drug therapy between LUS1 and LUS2

Treatments	LUS1, n (%)	LUS2, n (%)	*P*
ACEIs/ARBs	149 (88.7)	100 (59.5)	< .001
Beta-blockers	161 (95.8)	160 (95.2)	> .99
ARNIs	2 (1.2)	46 (27.4)	< .001
MRAs	99 (58.9)	101 (60.1)	.84
Loop diuretics	106 (63.1)	91 (54.2)	.02
Digoxin	38 (22.6)	29 (17.3)	.09
SGLT2i	0	20 (11.9)	—
Thiazides	6 (3.6)	10 (6)	.42

ACEIs, angiotensin-converting enzyme inhibitors; ARBs, angiotensin II receptor blockers; ARNIs, angiotensin receptor-neprilysin inhibitors; LUS, lung ultrasound; LUS1, initial LUS; LUS2, follow-up LUS; MRAs, mineralocorticoid receptor antagonists; SGLT2i, sodium-glucose cotransporter-2 inhibitor.

Table 2
Changes in functional class at the 4-year follow-up

	LUS2			
LUS1	NYHA I	NYHA II	NYHA III	Total
NYHA I	14	18	0	32
NYHA II	12	82	22	116
NYHA III	0	6	14	20
Total	26	104	36	168

LUS1, initial lung ultrasound; LUS2, follow-up lung ultrasound; NYHA, New York Heart Association functional class.
McNemar's test, $P = .006$.

diuretics was lower at LUS2 than at LUS1 ($P = .02$), whereas the dose of furosemide or equivalent in patients who were still receiving diuretics was not significantly different ($P = .14$). There were no clinically significant changes in LVEF or NT-proBNP during follow-up, with a mean LVEF of 46.5% ± 12.3% ($P = .045$) and a median NT-proBNP of 458 [177-1158] ng/L ($P = .43$) at the time of LUS2. Most patients (65%) were in the same functional class and, when this changed, more patients worsened (23.8%) than improved (10.7%) (table 2). There were no significant changes in the B line sum between the first (2.3 ± 3.7) and second (3.4 ± 3.9) examinations ($P = .19$) in the 40 patients who deteriorated by at least 1 functional class. There was also no association between the B line sum in LUS1 and functional class deterioration. During follow-up, there were 19 hospitalizations for HF in 12 patients (2.7/100 patient-years of follow-up). Notably, one of these patients experienced 4 admissions for acute pulmonary edema, with no evidence of congestion outside of these episodes.

This study is the first with a long-term follow-up (4 years) to assess the progression of pulmonary congestion in outpatients with chronic HF. Our results reveal that the subgroup of patients with low risk of HF hospitalization according to clinical course, biomarkers, and LVEF also show a low rate of subclinical pulmonary congestion on follow-up LUS. In this context, monitoring with LUS could provide information to the clinician in the outpatient setting, where access to natriuretic peptides and echocardiography is more limited. Indeed, other studies of chronic HF have demonstrated the prognostic value of subclinical pulmonary congestion in stable patients.[3–5] The limitations of our analysis include its single-center design with patients managed in a multidisciplinary HF unit in a tertiary hospital with a structured follow-up and the fact that we cannot rule out a possible selection bias given that it concerns patients who survived until the second visit,[3] which is why our results cannot be extrapolated to all patients with chronic HF.

Our results show that, in a selected group of outpatients with stable chronic HF, there was no increase in subclinical pulmonary congestion on LUS, despite the long-term progressive deterioration in loop diuretics and functional class deterioration experienced by some of the patients.

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M. Domingo: conception, design, data acquisition, interpretation, drafting, critical review of the intellectual content, and final approval of the version for publication. G. Cediel, P. Codina, E. Santiago-Vacas, and A. Bayés-Genís: critical review of the intellectual content and final approval of the version for publication. J. Lupón: conception, conception, design, analysis, interpretation, drafting, critical review of the intellectual content, and final approval of the version for publication.

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