States), which could be precisely managed as a "device" by the console.

When preparing to cross the guidewire, the assembling of the balloon or the stent over the wire in a single attempt can spare exchange times; this is particularly important, mainly in the early procedures, as it represents a new competence for the nursing team.

During displacement of the guiding catheter from the coronary ostia during PCI, "pulling the device" while maintaining balloon inflation, as can be done advancing the catheter during manual PCI, can mostly solve inadequate coronary engagement.

After stent deployment, pushing forward the device (stent balloon) to ensure its complete release before its withdrawal can help to avoid deep catheter intubation.

Use high doses of heparin. Catheter tip thrombus occurred in 2 patients during the first trimester of training who received a weight-adjusted heparin dose (100 UI/kg). However, since we systematically administered 10 000 UI, no intracoronary thrombus has been documented and no bleeding complications have appeared so far.

In conclusion, prior evidence indicates that R-PCI is safe and effective,[1,2] and opens new horizons in the field of coronary revascularization. Evidence on its ability to perform complex PCI—including AHA C lesions, chronic total occlusions and left main disease—has been reported, maintaining outstanding clinical and procedural results,[3,5] and real-life data of this robotic technology in daily PCI is currently ongoing. Our early experience is favorable, although limited by selection bias, as it was gained in a single center and we lacked a control cohort to match our findings. Available models so far have certain limitations, as they allow for manipulation of only 1 coronary guidewire or device at a time, are incompatible with intracoronary imaging techniques, and have high costs. These and other technical limitations can be addressed through iteration and innovation.[6] Nevertheless, early experience requires adequate lesion selection, with cumulative experience procedural times tend to shorten, and better skills are acquired to deal with higher levels of complexity (figure 1).

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AUTHORS' CONTRIBUTIONS

All authors meet each and all the characteristics defined by the International Committee of Medical Journal Editors for the criteria for authorship of scientific articles.

CONFLICTS OF INTEREST

Nothing to disclose.

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Mycotic pseudoaneurysm and aortitis caused by *Candida sake*

Seudoaneurisma micótico y aortitis por Candida sake

To the Editor,

A 60-year-old woman was admitted for right-sided lower back pain, with onset 7 days previously, and fever that gradually worsened despite analgesia. The patient was an exsmoker and had elevated cholesterol levels, primary hyperparathyroidism, and chronic femoropopliteal ischemia of the left lower limb. She was receiving treatment with rosuvastatin, aspirin, and calcifediol. On physical examination, the aortic beat was palpable. Blood analysis yielded the following values: C-reactive protein, 96.69 [0-5] mg/L; leukocytes, 9.45 [4.5-11] $\times 10^3$ with neutrophils at 8.18 [2-5] $\times 10^3$, and fibrinogen, 682 [200-450] mg/dL. Urine and blood cultures were performed, and ceftriaxone (2 g/24 h iv) treatment was started.

Contrast-enhanced computed tomography (CT), performed after 2 days of treatment with no appreciable improvement in the patient's condition, showed increased thickness of the infrarenal aortic wall, consistent with a mycotic pseudoaneurysm (figure 1). Daptomycin (850 mg/24 h iv) was added to her treatment; transthoracic echocardiography showed no cardiac vegetations. Findings on single-photon emission tomography

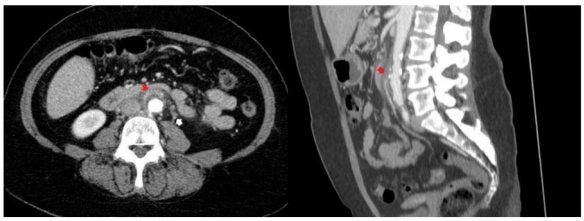

Figure 1. Thickening of the infrarenal aortic wall (red arrow) with luminal irregularities. Associated adjacent inflammatory changes with periaortic fat stranding and reactive lymph nodes (white arrow).

(SPECT)-CT of indium-111-oxine-labeled leukocytes were also consistent with mycotic pseudoaneurysm (figure 2).

Surgical resection involving an aortofemoral bypass and placement of a silver-impregnated bifurcated stent in the right lower limb was performed with in situ revascularization, and 2 aortic wall specimens were obtained for microbiological study. The patient's condition improved markedly after surgery and she was discharged home with intravenous antibiotics.

Urine and blood cultures were negative. The 2 aortic wall specimens were inoculated on chocolate agar, Brucella agar, CAN agar, BD MacConkey agar, and thioglycolate-enriched medium, and incubated at 37 °C. In addition, the enriched medium was used for molecular study to investigate bacterial and fungal pathogens. The *16S* ribosomal RNA gene was studied to rule out bacteria, whereas the internal transcribed spacer (ITS) 1, the *5.8 S* ribosomal RNA gene, and ITS-2 were sequenced to exclude fungi. All cultures and *16S* ribosomal RNA sequencing were negative; however, a sequence was obtained from ITS-1, *5.8 S* and ITS-4. The sequence was analyzed in Blast, and *Candida sake* was identified with 100% sequence alignment.

Based on these results, the patient's antibiotic was changed to caspofungin (50 mg/24 h iv) for 6 weeks. In addition, transesophageal echocardiography was performed, which showed no cardiac vegetations.

Among other causes, pseudoaneurysms can be iatrogenic or result from trauma or inflammation. Mycotic pseudoaneurysms are rare (incidence, 0.7%-3%), and when secondary to endocarditis or osteomyelitis, they are often caused by *Staphylococcus aureus* and streptococci.[1] The genus *Salmonella* is the most common cause of arteritis (half the total cases). Fungal infections are uncommon and mainly occur in patients who are immunosuppressed or have undergone surgery.[2]

C. sake has been isolated from tomato and apple skins. It grows at temperatures of 1 ° to 20 °C, whereas temperatures > 34 °C can impede its growth. This was likely the cause of its failure to grow in our cultures. It has rarely been associated with infection in humans, but is reported to be a cause of endocarditis.[3] This is the first description of *C. sake* causing a mycotic pseudoaneurysm and aortitis. Some strains of this microorganism are resistant to fluconazole, while others are not. Voriconazole, caspofungin, and amphotericin B seem to be viable treatment options for these infections.

The diagnosis of pseudoaneurysms is based on clinical suspicion, radiologic findings, and in the case of mycotic pseudoaneurysms, on microbiological confirmation. It is important to rule out secondary involvement, such as endocarditis or osteomyelitis, as the type of intervention differs in each case. Up to 25% of blood cultures can test negative, while direct cultures may be even less sensitive (40%-50% of cases).[1] Advances in the molecular techniques used have enabled etiological diagnosis in cases of high clinical suspicion with negative cultures.[4] The patient had chronic left lower limb ischemia, which could be related to the development of these conditions or to culture-negative endocarditis. Hence, in the presence of this clinical finding, some of these conditions should be ruled out.

Surgical treatment and constant antibiotic administration are essential. The duration of antibiotic treatment has been estab-

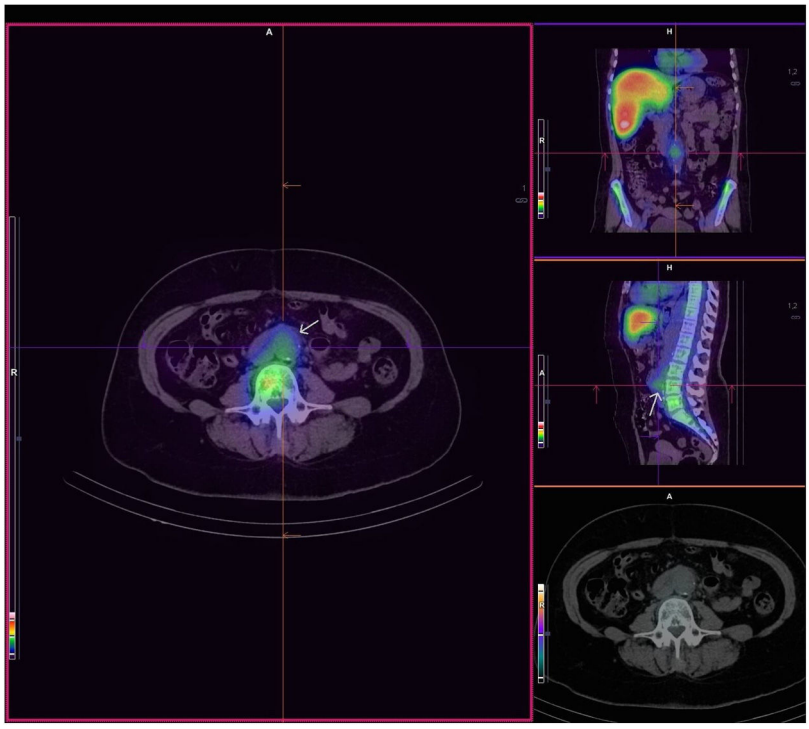

Figure 2. Single photon emission tomography/computed tomography with indium-111-oxine-labeled leukocytes at 24 hours postinjection. Pathological leukocyte accumulation in the posteromedial infrarenal aorta at the level of the L4 lumbar vertebra (arrow).

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