

Detection of spleen, kidney and liver infarcts by abdominal computed tomography does not affect the outcome in patients with left-side infective endocarditis

José A. Parra, MD, PhD^{a,*}, Luis Hernández, MD^b, Patricia Muñoz, MD, PhD^c, Gerardo Blanco, MD^d, Regino Rodríguez-Álvarez, MD^e, Daniel Romeu Vilar, MD^f, Arístides de Alarcón, MD^g, Miguel Angel Goenaga, MD^h, Mar Moreno, MDⁱ, María Carmen Fariñas, MD, PhD^{j,*}, on behalf of the Spanish Collaboration on Endocarditis-Grupo de Apoyo al Manejo de la Endocarditis Infecciosa en España (GAMES)

Abstract

Extra-cardiac abdominal complications are common in left-side infective endocarditis (LS-IE). The aim of this work was to study whether patients with LS-IE presenting splenic, renal, or liver (SRL) involvement seen in abdominal computed tomography (CT) had different clinical features, therapeutic plans, and outcome than those without these findings on CT.

From January 2008 to April 2010, multidisciplinary teams have prospectively collected all consecutive cases of IE, diagnosed according to the Duke criteria, in which abdominal CT was performed.

A total of 147 patients with LS-IE had abdominal CT. Fifty (34%) had SRL lesions: 46 splenic, 15 renal, 1 liver infarct, and 2 liver abscesses. Patients with SRL lesions were mainly men ($P = .01$), had liver disease ($P = .001$) with natural valve ($P = .050$) and mitro-aortic valve involvement ($P = .042$), splenomegaly ($P = .001$), nonabdominal emboli ($P = .001$), and a greater number and larger vegetation (>15 mm, $P = .049$) in the mitro-aortic valves ($P = .051$) than patients with normal abdominal CT. The site of acquisition, clinical characteristics, microbiology, surgical treatment, days of hospitalization, hospital death, and 1-year mortality were similar in patients with and without SRL emboli on CT. In the stepwise logistic regression analysis, male gender (odds ratio [OR] = 3.6, 95% confidence interval [CI] = 1.4–9.1), liver disease (OR = 8.3, 95% CI = 2.1–31.8), and nonabdominal emboli (OR = 5.2, 95% CI = 2.3–11.7) were independently associated with SRL lesions.

Male patients with native LS-IE who had liver disease and nonabdominal emboli had more frequent abdominal lesions seen on CT. The presence of SRL infarcts on abdominal CT scan performed on patients with LS-IE seems to have poor practical implications, and as a consequence, its realization should only be considered when there are symptoms or signs that suggest them.

Abbreviations: CI = confidence interval, CNS = central nervous system, CT = computed tomography, GAMES = The Spanish Collaboration on Endocarditis-Grupo de Apoyo al Manejo de la Endocarditis Infecciosa en España, HACEK = *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*, HIV = human immunodeficiency virus, IE = infective endocarditis, IQR = interquartile range, IDU = intravenous drug users, LS-IE = left-side infective endocarditis, MDCT = multiple detector computed tomography, MRI = magnetic resonance imaging, OR = odds ratio, SD = standard deviation, SRL = splenic, renal, liver, TEE = transesophageal echocardiography, US = ultrasonography.

Keywords: computed tomography, endocarditis, kidney infarcts, liver abscess, liver infarcts, spleen infarcts

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^a Department of Radiology, Hospital Universitario Marqués de Valdecilla, Universidad de Cantabria, IDIVAL, Santander, Cantabria, ^b Department of Radiology, Hospital General Universitario Gregorio Marañón, Madrid, ^c Department of Clinical Microbiology and Infectious Diseases, Hospital General Universitario Gregorio Marañón, Universidad Complutense de Madrid, ^d Department of Radiology, Hospital Universitario Marqués de Valdecilla, Santander, Cantabria, ^e Service of Infectious Diseases, Hospital de Cruces, Bilbao, ^f Department of Radiology, Hospital Universitario A Coruña, Coruña, ^g Infectious Diseases Service, UGC de Enfermedades Infecciosas, Microbiología y Medicina Preventiva Grupo de Investigación en Enfermedades Infecciosas, Instituto de Biomedicina de Sevilla (IBIS)/CSIC/, Hospital Universitario Virgen del Rocío, Sevilla, ^h Service of Infectious Diseases, Hospital Universitario Donosti, San Sebastian, ⁱ Department of Cardiology, Hospital Universitario La Paz, Madrid, ^j Infectious Diseases Unit, Hospital Universitario Marqués de Valdecilla, IDIVAL, Universidad de Cantabria, Santander, Cantabria, Spain.

* Correspondence: José A. Parra, Department of Radiology, University of Cantabria. Hospital Universitario "Marqués de Valdecilla," Av. Valdecilla, S/N, 39008 Santander, Cantabria, Spain (e-mail: jparra@humv.es); María Carmen Fariñas, Infectious Diseases Unit, University of Cantabria, Av. Valdecilla, S/N, 39008 Santander, Cantabria, Spain (e-mail: mcfarinas@humv.es).

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1. Introduction

Despite improvements in the diagnosis and treatment of infective endocarditis (IE), emboli are still a relatively common complication (22%–50%), especially in patients with left-side infective endocarditis (LS-IE).^[1–7] Most of these emboli are produced in the first 2 to 4 weeks of antibiotic treatment and for the most part are in the central nervous system (CNS).^[7–9] They have also been found in the abdomen, vessels, and musculoskeletal system.^[7,10] Two studies have confirmed that the rate of embolic events decreases dramatically during and after the first 2 to 3 weeks after successful antibiotic therapy.^[8,11] The size of the vegetation (>1 cm), its mobility, the affects valve (mitral valve: anterior leaflet), and the causative agent (*Staphylococcus aureus*, *Candida*, and *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella* -HACEK- microorganisms) are factors that seem to predispose their occurrence.^[7,8,12–14] In the abdomen, the most frequent LS-IE complication sites are the spleen and kidneys, where vascular occlusion created by the emboli will produce an infarction that sometimes can evolve into an abscess formation.^[15,16] Ultrasound (US), computed tomography (CT), and more recently magnetic resonance imaging (MRI) are the most commonly used techniques when abdominal involvement is suspected in patients with LS-IE.^[16,17] However, despite the relative frequency of these complications, few studies^[5,18] have reported if patients with emboli seen on abdominal images have different clinical and etiological characteristics than those without them. Although it could be hypothesized that the involvement of these abdominal organs could cause a poorer prognosis, the influence of the awareness of the presence of these lesions in the treatment and prognosis of LS-IE is unknown.

The aim of this study was to study whether patients with LS-IE who presented embolic complications on abdominal CT had different epidemiological and clinical characteristics, treatment or outcome than those who did not, in order to know the usefulness of performing routine abdominal CT.

2. Methods

From May 2008 to April 2010, a prospective multicenter cohort study was carried out in 25 Spanish hospitals. These centers are included in the Spanish Collaboration on Endocarditis-Grupo de Apoyo al Manejo de la Endocarditis Infecciosa en España (GAMES).^[19] Specific data on demographics, medical history, predisposing factors, microbiology, imaging studies, echocardiography, complications, antibiotic therapy, surgery, and outcomes were obtained for each patient. Cases of definite and possible LS-IE were defined according to the modified Duke criteria.^[20]

Of the 25 centers that collected the data, 6 participated in this study. A total of 206 patients had a CT performed and 147 with the abdomen included. Almost one-third (43) of these abdominal CTs were performed as part of a study to identify the frequency of abdominal affectation in IE patients, and the rest because of patient symptoms. Patients with spleen, renal or liver (SRL) infarcts, or abscesses on abdominal CT were compared to those with normal CT scans. Only the first abdominal CT was considered in the analysis. The definitions were described previously.^[19]

Abdominal CT studies were carried out via 2 to 64 multiple detector computed tomography (MDCT) scans after the administration of an intravenous contrast agent. In the spleen, renal, and liver, infarct was defined as a triangular area of low density that did not show enhancement.^[16,21] An abscess was

considered if there was gas within the hypodense lesion or a capsule around the hypodense defect on the CT performed after the administration of an intravenous contrast agent.^[21]

Replacement or repair of the affected valve or correction of anatomical abnormality and/or debridement during the hospitalization for IE was divided in 3 periods: emergency surgery (<8 days), early surgery (8–21 days), and late surgery (>22 days).

2.1. Statistical analysis

Patients with LS-IE and SRL infarcts or abscesses on abdominal CT scan were compared to those with normal CT. Quantitative variables were expressed as mean and standard deviation (SD) or as medians with interquartile range (IQR) as appropriate; qualitative variables were expressed as frequency and percentage. Continuous variables were compared using the *t*-test, and categorical variables were compared using the chi-squared test or Fisher's exact test when the chi-square test was not appropriate. The Mann-Whitney *U* test was used to compare nonparametric variables. Multivariate analysis was performed using a stepwise logistic regression (with a significance level for removal from the model and for addition to the model of 0.2) and adjusted odds ratios (OR) and their 95% confidence intervals (CI) were calculated to analyze the association between different exposure variables and the presence of SRL lesions on CT or in-hospital mortality. A curve of survival was obtained using the Kaplan-Meier method, calculating survival rates for the time elapsed between discharge and 1-year mortality in patients surviving hospitalization. The heterogeneity of the curves was tested using the log-rank test. Cox regression was applied to estimate survival rate over time as a function of several covariates. Hazard ratios (HR) and their 95% CI were calculated.

A two-tailed $P < .05$ was considered statistically significant. The data were analyzed using SPSS (version 19) (SPSS Inc., Chicago, IL) statistical software.

2.2. Ethics

The entire project and the common case report forms were approved by the national and local institutional review boards and ethics committees (E.C. 155 18/07). All patients signed an informed consent according to established standards.

3. Results

Of the 147 patients included in the study, 136 (92.5%) had a CT performed after the administration of an intravenous contrast agent. The median time between hospital admission and abdominal CT was 6 days (IQR 4–14) in the SRL group and 8 days (IQR 4–22) in the patients without SRL. Table 1 shows the demographic characteristics of the entire population, those with SRL emboli and those with normal CT images. As can be seen, 50 (34%) of 147 patients had SRL emboli: 44 splenic infarcts, 15 kidney infarcts, 1 liver infarct, and 2 liver abscesses. Ten patients had more than 1 organ involved, mainly spleen and renal infarcts. We did not find differences in the incidence of SRL infarcts of patients with abdominal CT scan performed routinely ($n=43$; 27.90% of patients had SRL infarcts) or because a clinical indication ($n=104$; 36.53% of patients had SRL lesions) ($P=.475$). There were 42 (84%) men with a median age of 63 years (IQR 50.7–76) in the group of patients with SRL emboli and 62 (63.9%) men with a median age of 68 years (IQR 61–76.5) in the group without emboli on CT images ($P=.013$ and $P=.064$, respectively).

Table 1**Epidemiological and clinical characteristics of 147 patients with left-side infective endocarditis and abdominal CT* scan.**

	All patients (%) N = 147	Patients with SRL [†] lesions (%) N = 50	Patients with normal CT* (%) N = 97	P value [‡]
Mean age (SD) [§]	65.0 (14.3)	62.1 (14.4)	66.5 (14.1)	.076
Male	104 (70.7)	42 (84.0)	62 (63.9)	.013
Affected valve				
Natural valve	109 (74.1)	42 (84.0)	67 (69.1)	
Prosthetic valve	38 (25.9)	8 (16.0)	30 (30.9)	.050
Type of affected valve				
Aortic valve	54 (36.7)	19 (38.8)	35 (36.1)	
Mitral valve	70 (47.6)	18 (36.7)	52 (53.6)	
Mitral-aortic	22 (14.9)	12 (24.5)	10 (10.4)	.042
Underlying conditions				
Active smoker	26 (17.7)	12 (26.1)	14 (15.6)	.20
Lung disease	33 (22.4)	12 (24.0)	21 (21.6)	.67
Coronary disease	28 (19.0)	6 (12.0)	22 (22.7)	.13
Atrial fibrillation	36 (24.5)	8 (16.0)	28 (28.9)	.080
Cardiac device	10 (6.8)	1 (2.0)	9 (9.3)	.17
Heart failure	47 (32.09)	12 (24.0)	35 (36.1)	.14
Diabetes mellitus	32 (21.8)	8 (16.0)	24 (24.7)	.22
Hypertension	87 (59.2)	26 (52.0)	61 (62.9)	.20
Hyperlipidemia	47 (32.0)	14 (28.0)	33 (34.0)	.41
Peripheral vascular disease	15 (10.2)	4 (8.0)	11 (11.3)	.53
Cerebrovascular disease	28 (19.0)	7 (14.0)	21 (21.6)	.26
Neoplasia	35 (23.8)	10 (20.0)	25 (25.8)	.44
Renal insufficiency	38 (22.6)	11 (22.4)	27 (28.1)	.46
Liver disease	15 (10.2)	11 (22.0)	4 (4.2)	.001
Neurological disease	10 (6.8)	2 (4.0)	8 (8.2)	.50
HIV infection	3 (2.0)	3 (6.0)	0 (0.0)	.039
IVDU [¶]	3 (2.0)	3 (6.0)	0 (0.0)	.038
Previous IE [#]	9 (6.1)	2 (4.0)	7 (7.2)	.72
Congenital heart disease	4 (2.7)	0 (0.0)	4 (4.1)	.31
Previous cardiac surgery	40 (27.2)	9 (18.0)	31 (32.0)	.066
Aortic regurgitation	45 (30.6)	10 (20.4)	35 (36.1)	.045
Aortic stenosis	17 (18.3)	8 (16.3)	19 (19.6)	.67
Mitral regurgitation	44 (29.99)	11 (22.4)	33 (34.0)	.15
Mitral stenosis	6 (4.0)	2 (4.1)	4 (4.1)	.98
Site of acquisition				
Nosocomial	29 (19.7)	7 (14.6)	22 (23.9)	
Community	99 (67.3)	38 (79.2)	61 (66.3)	
Health care related	12 (8.1)	3 (6.3)	9 (9.8)	.28
Presentation				
Fever >38°C	126 (85.7)	42 (84.0)	84 (86.6)	.57
Splinter hemorrhage	20 (13.6)	8 (16.0)	12 (12.4)	.53
Nonabdominal emboli	64 (43.5)	32 (64.0)	32 (33.0)	.001
New murmur	65 (44.2)	22 (44.0)	43 (44.3)	.85
Worsening of old murmur	20 (13.6)	5 (10.0)	15 (15.5)	.55
Splenomegaly	31 (21.0)	18 (36.0)	13 (13.4)	.001
Heart failure	55 (37.4)	20 (40.0)	35 (36.1)	.68
Mean ESR ^{**} (SD)	58.4 (38.9)	60.8 (38.0)	57.0 (39.6)	.65
Glomerulonephritis	2 (1.3)	0 (0.0)	2 (2.1)	.55
Elevated rheumatoid factor	17 (11.5)	9 (18.0)	8 (8.2)	.083
Myocardial infarction	4 (2.7)	1 (2.0)	3 (3.1)	1.00
Persistent bacteremia	11 (7.4)	1 (2.0)	10 (10.3)	.10
CNS ^{††} embolisms	37 (25.1)	16 (32.0)	21 (21.6)	.23
New renal insufficiency	56 (38.0)	17 (34.0)	39 (40.2)	.46
Ventricular tachycardia	1 (0.6)	0 (0.0)	1 (1.0)	1.00
Mechanical ventilation	20 (13.6)	8 (16.0)	12 (12.4)	.56
Septic shock	17 (11.5)	3 (6.0)	14 (14.4)	.13

* CT = computed tomography.

† SRL = spleen, renal, liver.

‡ 2-tailed chi-squared test/Fisher's exact test or t-test as corresponding.

§ SD = standard deviation.

|| HIV = human immunodeficiency virus.

¶ IVDU = intravenous drug users.

IE = infective endocarditis.

** ESR = erythrocyte sedimentation rate.

†† CNS = central nervous system.

Table 2**Etiology of 147 patients with left-side infective endocarditis and abdominal CT scan.**

Etiology	All patients (%) N=147	Patients with SRL lesions (%) N=50	Patients with normal CT (%) N=97	P value*
<i>Streptococcus</i> spp	46 (41.2)	20 (40.0)	26 (26.8)	.56
<i>Staphylococcus aureus</i>	30 (20.4)	10 (20.0)	20 (20.6)	
Coagulase negative <i>staphylococci</i>	22 (14.9)	7 (14.0)	15 (15.5)	
<i>Enterococcus</i> spp	18 (12.2)	6 (12.0)	12 (12.4)	
Others†	17 (11.5)	3 (6.0)	14 (14.4)	
Unknown	14 (9.5)	4 (8.0)	10 (10.3)	
Total	147	50	97	

* 2-tailed Fisher's exact test.

† *Abiotrophia* (2 patients), *Scedosporium* (1 patient), *Acinetobacter* (2 patients), *Actinobacillus* (1 patient), *Bacteroides* spp (1 patient), *Brucella* (1 patient), *Coxiella burnetii* (1 patient), *Escherichia coli* (1 patient), *Tropheryma whippelii* (1 patient), *Candida* spp (3 patients), *Proteus mirabilis* (1 patient), *Aggregatibacter aphrophilus* (1 patient), polymicrobial infection (*Enterococcus avium*, *Proteus mirabilis*, and *Pseudomonas aeruginosa*) (1 patient).

Other abbreviations as in Table 1.

Natural valve affectation (84.0% vs 69.1%, $P=.050$) and both mitral and aortic valve involvement were higher in the patients with SRL emboli, 12 (24.5%) vs 10 (10.4%); $P=.042$. The underlying conditions and site of acquisition were similar in patients with and without SRL emboli except for the presence of liver disease ($P=.001$) and number of human immunodeficiency virus (HIV) ($P=.039$) or intravenous drug users (IVDU) ($P=.038$) patients, which were more frequent in the patients with SRL emboli on CT.

Clinical manifestations were also similar, with fever being the most frequent finding in both groups. The only differences were nonabdominal emboli ($P=.001$) as well as splenomegaly ($P=.001$), which were significantly more frequent in the SRL group. In the stepwise logistic regression analysis, male gender (OR 3.6, 95% CI 1.4–9.1; $P=.008$), presence of liver disease (OR 8.3, 95% CI 2.1–31.8; $P=.002$), and nonabdominal emboli (OR 5.2, 95% CI 2.3–11.7; $P=.0001$) were the only predictors of emboli on the CT scan.

Regarding the etiology (Table 2), no statistically differences were seen in the microorganisms isolated in patients with or without abdominal emboli ($P=.56$). *Streptococci*, followed by *S aureus* and coagulase-negative *staphylococci* were the most

frequently isolated microorganisms in the group with SRL emboli. In 10% of patients, the etiology of LS-IE was unknown. In the 2 patients with liver abscesses, the isolated microorganisms were *S aureus* and *Streptococcus oralis*.

Table 3 shows the main echocardiographic findings. 119 out of 147 patients (80.9%) had a transesophageal echocardiography (TEE), 80% in the SRL group and 81.4% in normal CT scan group. Vegetation in the mitral and aortic valves was similar in both groups. However, the presence of vegetation in the mitral and aortic valves predominated in the group of patients with SRL (22% vs 8.2%) ($P=.051$). No differences were seen between the 2 groups regarding intracardiac complications, valve perforation, pseudoaneurysm, abscess, or fistula. Patients with SRL emboli on CT scan had more and larger (86.0%) vegetation than those with normal CT (presence of vegetation longer than 15 mm in 33.3% vs 15.3%; $P=.049$).

Treatment details and outcomes are shown in Table 4. No statistically significant differences were seen between both groups regarding the type and duration of antibiotics and timing or reasons for surgery. Patients with and without SRL emboli on abdominal CT had similar hospitalization (median=47 days, IQR: 29–59.5 vs 48 days, IQR: 29.7–57.5]) and median hospital

Table 3**Echocardiographic findings of 147 patients with left-side infective endocarditis and abdominal CT scans.**

	All patients (%) N=147	Patients with SRL lesions (%) N=50	Patients with normal CT (%) N=97	P value*
Vegetation	114 (77.6)	43 (86.0)	71 (73.2)	.78
Vegetation in TTE†	30 (20.4)	11 (22.0)	19 (19.6)	.73
Vegetation in TEE‡	88 (59.9)	31 (62.0)	57 (58.8)	.70
Vegetation in TTE and TEE	29 (16.0)	8 (16.0)	21 (21.6)	.41
Aortic vegetation	40 (27.2)	15 (30.0)	25 (25.8)	.051
Mitral vegetation	55 (37.4)	17 (34.0)	38 (39.2)	
Mitro-aortic vegetation	19 (12.9)	11 (22.0)	8 (8.2)	
Intracardiac complication	39 (26.5)	9 (18.0)	30 (30.9)	.093
Valve perforation	19 (17.6)	4 (10.5)	14 (20.6)	.18
Pseudoaneurysm	6 (5.6)	1 (2.6)	5 (7.1)	.42
Abscess	16 (15.1)	4 (10.5)	12 (17.6)	.31
Fistula	1 (0.9)	1 (2.6)	0 (0.0)	.36
Vegetation length >10 mm	37 (41.6)	16 (53.3)	21 (35.6)	.11
Vegetation length >15 mm	19 (21.3)	10 (33.3)	9 (15.3)	.049
Mean vegetation length (mm) (SD)	11.3 (10.8)	14.8 (15.7)	9.6 (6.7)	.12

* 2-tailed chi-squared test/Fisher's exact test or *t*-test as corresponding.

† TEE = transesophageal echocardiogram.

‡ TTE = transthoracic echocardiogram.

Other abbreviations as in Table 1.

Table 4
Antibiotics, surgery treatments, and outcomes in 147 patients with left-side infective endocarditis and abdominal CT scans.

	All patients (%) n = 147	Patients with SRL lesions (%) N = 50	Patients with normal CT (%) N = 97	P value*
Medical treatment				
B-lactam [†]	113 (76.8)	42 (44.0)	71 (73.2)	.14
Aminoglycosides [‡]	72 (48.9)	25 (50.0)	47 (48.5)	.85
Vancomycin	48 (32.6)	12 (24.0)	36 (37.1)	.10
Median antibiotic treatment (IQR) [§]	42 (29.7–42.2)	42 (30.0–42.0)	42 (30–42)	.96
Surgical treatment	72 (48.9)	26 (52.0)	46 (47.4)	.59
Emergency surgery (<8 days)	17 (23.6)	4 (15.3)	13 (28.2)	
Early surgery (8–21 days)	22 (30.6)	7 (27.0)	15 (32.6)	.32
Late surgery (>22 days)	33 (45.8)	15 (57.7)	18 (39.2)	
Reasons for surgery				
Severe heart failure	41 (27.8)	15 (34.1)	26 (28.6)	.51
Myocardial invasion	7 (4.7)	3 (6.8)	4 (4.4)	.55
Severe valve regurgitation	30 (20.4)	11 (25.0)	19 (20.9)	.58
Systemic embolism	9 (6.1)	6 (13.6)	3 (3.3)	.024
Outcome				
Median hospital stay (IQR) [§]	48 (29–59)	48 (29.7–57.5)	47 (29–59.5)	.98
In-hospital mortality	34 (23.1) n = 113	13 (26.0) n = 37	21 (21.6) n = 76	.55
One-year mortality [¶]	13 (11.5)	6 (16.2)	7 (9.2)	.35
Mean survival time from discharge (95% CI ^{**})	336.2 (320.6–351.8)	323.4 (290.6–356.3)	342.4 (320.6–351.8)	.26 [#]

* 2-tailed chi-squared test/Fisher's exact test or Mann-Whitney *U* test as corresponding.

[†] β-lactam: penicillin, cloxacillin, ampicillin, ceftriaxone, cefazolin, and cefotaxime.

[‡] aminoglycosides: gentamicin, tobramycin, and amikacin.

[§] IQR: interquartile range.

^{||} patients who were alive after hospital discharge and included in the follow up.

[¶] 1-year mortality in patients surviving hospitalization.

[#] Log-rank test.

^{**} CI: confidence interval.

Other abbreviations as in Table 1.

stay (48 days, IQR 29.7–57.5 vs 47 days, IQR 29–59.5 $P = .98$). There were no statistically significant differences in global hospital deaths or 1-year mortality between both groups (Table 4). A total of 34 patients (23.1%) died during hospitalization: 13 (26%) had abdominal emboli vs 21 (21.6%) with normal CT ($P = .55$). During the 1-year follow-up, there were 13 deaths: 6 (16.2%) patients with abdominal emboli vs 7 (9.2%) without them ($P = .35$) (Table 4). We also did not find statistically significant differences in the incidence of global hospital deaths: 8 out of 43 (18.6%) patients died when routine abdominal CT scan was performed and 26 out of 104 (25.0%) when because of clinical indication ($P = .40$). Similarly, no statistically significant differences were observed in 1-year mortality between the 2 groups: 3 deaths (8.6%; $n = 35$ alive at discharge) when the CT was routinely indicated compared with 10 deaths (12.8%; $n = 78$ alive at discharge) among those performed because a clinical indication ($P = .51$).

The Kaplan–Meier survival curve in the patients with or without SRL lesions on abdominal CT estimates of 1-year overall survival without statistically significant differences between patients with and without abdominal emboli on CT (Fig. 1): mean survival time of 323.4 days vs 342.4 days ($P = .26$). Similarly, in Cox regression analysis, patients with SRL lesions on abdominal CT did not have a significantly higher death hazard than patients without them (HR adjusted by sex 1.85, 95% CI 0.61–5.61, $P = .28$).

The stepwise regression model selected sex as the unique significant predictor of in-hospital mortality (OR 3.03 95% CI 1.23–7.46, $P = .016$). Other factors selected in the model were the presence of vegetation (OR 3.28, 95% CI 0.89–12.09, $P = .074$)

and SLE emboli on CT (OR = 1.92, 95% CI 0.78–4.70, $P = .15$), although there were no statistically significant differences.

4. Discussion

Abdominal CT, US, and MRI have been used to explore abdominal complications in patients with SRL. However, in clinical practice, US and CT are the most frequently used, especially CT, which is considered superior to US for screening due to its ability to detect changes even in patients with normal ultrasound.^[15,18] Almost all CT explorations in this study were performed after the administration of an intravenous contrast agent when infarcts and abscesses were better seen and in the first 2 weeks after hospital admission when the risk of embolic events was higher.^[7,8]

The proportion of patients with SRL (34%) in our study is similar to other series of the postantibiotic era,^[22] although relatively high compared to recent series such as Luaces et al,^[18] Millaire et al,^[5] or Aalaei-Andabili et al,^[23] where SRL was around 10%. It is possible that the method of selection (with a large group of patients who underwent a CT scan systematically) and the time when the CT was performed result in these differences.^[7,24]

The vascular obstruction produced by an emboli in a patient with LS-IE can result in a splenic, renal, or hepatic abscess; however, most lesions correspond to infarcts and only a small proportion (<4.8%) to abscesses.^[16,17,24,25] Most are located in the spleen and kidney and only a small proportion in the liver, due to the double arterial and portal circulation.^[12] In agreement with these data, practically all of the lesions detected in our study

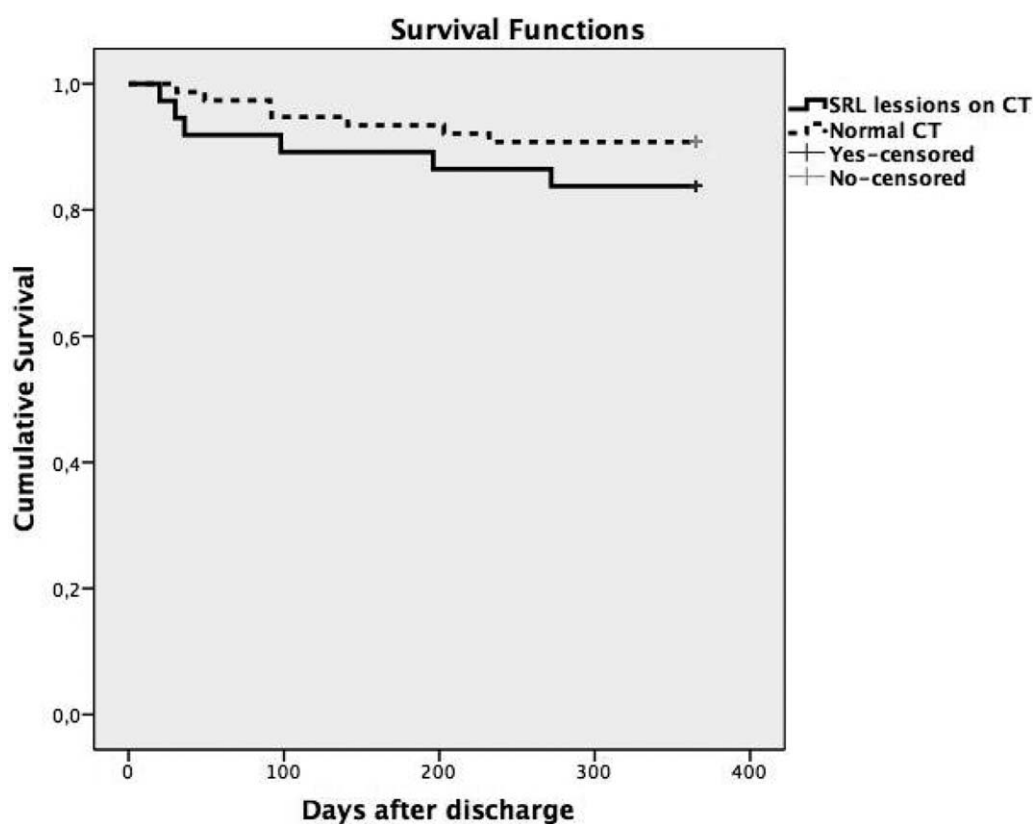


Figure 1. Kaplan–Meier survival curve of patients with left-left side infective endocarditis with and without spleen, liver and renal (SRL) lesions on abdominal computed tomography (CT) scans.

corresponded to infarcts. The majority was in the spleen and kidney, and only 2 patients had liver abscesses that resolved adequately with medical treatment. None of our patients underwent surgical treatment because of splenic, renal, or hepatic involvement during the 1-year of follow up.

Left upper pain or flank pain and splenomegaly or hematuria are the most frequent symptoms and signs in patients with SRL infarcts. However, many of these patients are asymptomatic, and an infarct is an incidental finding on CT, MRI, or US.^[24] Our patients, independent of the presence or absence of SRL emboli had similar characteristics in terms of the type of site of acquisition, clinical background, and causal microorganism. The only significant differences we found compared to other studies^[12,18,24] were the predominance of men, the mitral-aortic valve involvement,^[24] the presence of liver disease,^[26] splenomegaly,^[18] the number of HIV and IVDU patients,^[12] and the number of nonabdominal emboli, which were more frequently found in the patients with SRL lesions. In our study as well as in Luaces et al.^[18] study, patients with SRL infarcts were younger than those without abdominal affection. Our study also shows a trend toward a lower proportion of embolic events in patients with endocarditis in prosthetic valves compared to native valves. Possibly, as Durante Mangoni et al.^[6] pointed out, smaller vegetation size, treatment, or age are important factors.

Most embolic complications have been associated with *S aureus*, *Candida*, and HACEK microorganisms^[7] infections. In our case, however, although overall *Staphylococci* were the most frequent bacteria causing LS-IE (66.6%), *Streptococcus* species were the most prevalent microorganisms (40%) among patients

with SRL embolic events. Geographical reasons and selection bias may explain this finding.^[1,3,27] Mitral vegetation, especially in the anterior valve, has been associated with an increased number of embolic events.^[28,29] In our series, mitro-aortic valve vegetation had more SRL lesions. It seems reasonable to consider as did Ting et al.^[24] that patients with concurrent aortic and mitral valve endocarditis, which may reflect a more virulent infection, appeared to be at a higher risk for embolization. In addition to the valve affected, vegetation length appears to be a strong predictor of embolic events.^[12] Our data support this finding since patients with vegetation longer than 15 mm had more abdominal embolisms.

We did not find a change in the therapeutic approach (medical or surgical) or in the time of surgical treatment between the groups with or without SRL emboli. This is likely due to the fact that most the SRL lesions on abdominal CT scan were infarcts. However, it is important to keep in mind and consider in patients with LS-IE and persistent fever, signs of sepsis or bacteremia, and appropriate antibiotic treatment, the use of an abdominal imaging study seeking SRL abscess that could require specific surgery or percutaneous drainage.^[30] Although cases have been described with splenectomy performed during or after cardiac surgery,^[23,30–32] this approach is generally realized previous to cardiac surgery.^[7,33]

In-hospital and 1-year mortality were similar between patients with or without SRL emboli. Attributable mortality was higher in patients with abdominal involvement but without significant differences. Findings such as those of Lung et al.^[17] and Luaces et al.^[18] in which the presence of abdominal affection in the form

of SRL infarcts has little repercussion on the therapeutic treatment, surgery is necessary or death could result.

Several study limitations should be taken into consideration when extrapolating our results. First, this was not a clinical trial, so the results should be interpreted with caution. Second, this study was performed in most of the patients in whom the multidisciplinary team of experts asked for abdominal CTs due to clinical suspicion of abdominal emboli. This could have led to selection bias because our series may not represent the situation of all patients with LS-IE, but those who may have a worse outcome. Another possible bias could result from the fact that abdominal CT was interpreted in each center, and inter-individual variability was not evaluated. In addition, although it is a multicenter survey with a large number of patients recruited, the number of deaths could be considered relatively small, and this may lack the statistical power to detect some associations, mainly those with little influence. However, there are no other studies that include a concomitant endocarditis and analyze the influence of emboli complications on abdominal CT in patients with LS-IE with this long-term follow-up.

In summary, despite the limitations, our results showed that males with LS-IE liver disease and nonabdominal emboli had more risk of abdominal emboli found on CT scan. The presence of SRL infarcts on abdominal CT scan performed on patients with LS-IE within 2 weeks after hospital admission seems to have poor practical implications. We did not find differences in the incidence of SRL infarcts when CT scan was performed routinely or with clinical indication. New studies should be done to confirm these preliminary results.

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Members of GAMES: Hospital Costa del Sol, (Marbella): Fernando Fernández Sánchez, Marian Noureddine, Gabriel Rosas, Javier de la Torre Lima; Hospital de Cruces, (Bilbao): José Aramendi, Elena Bereciartua, María Victoria Boado, Marta Campaña Lázaro, Josune Goikoetxea, Juan José Goiti, José Luis Hernández, José Ramón Iruetagoiena, Josu IrurzunZuazabal, Inés Martínez, Miguel Montejo, Pedro María Pérez, Regino Rodríguez, Roberto Voces; Hospital Clínico Virgen de la Victoria, (Málaga): M Victoria García López, RadkaIvanova-Georgieva, Manuel Márquez Solero, Isabel Rodríguez Bailón, Josefa Ruiz Morales; Hospital Donostia-Policlínica Gipuzkoa, (San Sebastián): Ana María Cuende, Miguel Ángel Goenaga, Pedro Idígoras, José Antonio Iribarren, Alberto Izaguirre Yarza, Carlos Reviejo, Tomás Echeverría, Eduardo Gaminde, Ana Fuertes; Hospital General Universitario de Alicante, (Alicante): Rafael Carrasco, Vicente Climent, Patricio Llamas, Esperanza Merino, Joaquín Plazas, Sergio Reus; Hospital Juan Canalejo, (Coruña): Nemesio Álvarez, José María Bravo-Ferrer, María del Mar Carmona, Laura Castelo, José Cuenca, Pedro Llinares, Enrique Miguez Rey, Dolores Sousa, M Carmen Zúñiga; Hospital Juan Ramón Jiménez, (Huelva): José Manuel Lomas, Francisco Javier Martínez; Hospital Universitario de Canarias, (Canarias): M del Mar Alonso, Beatriz Castro, Dácil García Marrero, M del Carmen Durán, M Antonia Miguel Gómez, Juan La Calzada, Ibrahim Nassar; Hospital Regional Universitario Carlos Haya, (Málaga): Antonio Plata Ciezar, José M Reguera Iglesias; Hospital Universitario Central Asturias, (Oviedo): Víctor Asensi Álvarez, Carlos Costas, Jesús de la Hera, Jonnathan Fernández Suárez, José Manuel García Ruiz, Lisardo

Iglesias Fraile, José López Menéndez, Pilar Mencía Bajo, Carlos Morales, Alfonso Moreno Torrico, Carmen Palomo, Begonia Paya Martínez, Francisco Pérez, Ángeles Rodríguez, Mauricio Telenti; Hospital Universitario Clínic de Barcelona, (Barcelona): Manuel Almela, Yolanda Armero, Manuel Azqueta, Ximena Castañeda, Carlos Cervera, Carlos Falces, Cristina García de la María, José M. Gatell, Magda Heras, Jaume Llopis Pérez, Francesc Marco, Carlos A. Mestres, José M Miró, Asunción Moreno, Salvador Ninot, José Ramírez, Ana del Río, Marta Sitges, Carlos Paré, Juan Manuel Pericás; Hospital General Universitario Gregorio Marañón, (Madrid): Javier Bermejo, Emilio Bouza, AliaEworo, Ana Fernández Cruz, Marcela González del Vecchio, Víctor González Ramallo, Martha Kestler Hernández, Mercedes Marín, Manuel Martínez-Sellés, M Cruz Menárguez, Patricia Muñoz, Hugo Rodríguez-Abella, Marta Rodríguez-Créixems, Jorge Rodríguez Roda, Marisol Salas, Antonio Segado, Jorge Solis, Blanca Pinilla, Ángel Pinto, Maricela Valerio, Eduardo Verde; Hospital Universitario La Paz, (Madrid): Isabel Antorrena, José M Fraile Vicente, Carlos García Cerrada, Luis García Guereta, Alicia Lorenao Hernández, Alejandro Martín Quirós, Mar Moreno, José Ramón Paño, M Angustias Quesada Simón, Mikel Rico, M Ángeles Rodríguez Dávila, María Romero, Sandra Rosillo, Alicia Rico Nieto, Mikel Rico, María Romero; Hospital Universitario Marqués de Valdecilla, (Santander): Jesús Agüero Balbín, Cristina Amado, Carlos Armiñanzas Castillo, Ana Arnaiz García, Manuel Cobo Belaustegui, María Carmen Fariñas, Concepción Fariñas-Álvarez, Rubén Gómez Izquierdo, Iván García, Claudia González Rico, Manuel Gutiérrez-Cuadra, José Gutiérrez Díez, Marcos Pajarón, José Antonio Parra, Aurelio Sarralde, Ramón Teira, Jesús Zarauza; Hospital Universitario Puerta de Hierro, (Madrid): Pablo García Pavía, Jesús González, Beatriz Orden, Antonio Ramos, Elena Rodríguez González; Hospital Universitario Ramón y Cajal, (Madrid): Tomasa Centella, José Hermida, José Moya, Pilar Martínez, Enrique Navas, Enrique Oliva, Alejandro del Río, Soledad Ruiz; Complejo Hospitalario Universitario de Vigo, (Pontevedra): César Martínez, Andrés Nodar, Roberto Pérez, Francisco José Vasallo; Hospital Universitario Virgen de las Nieves, (Granada): Carmen Hidalgo Tenorio; Hospital Universitario Virgen Macarena, (Sevilla): Antonio de Castro, Marina de Cueto, Pastora Gallego, Juan Gálvez Acebal; Hospital Universitario Virgen del Rocío, (Sevilla): Aristides de Alarcón, Emilio García, Juan Luis Haro, José Antonio Lepe, Francisco López, Rafael Luque; Hospital San Pedro, (Logroño): Luis Javier Alonso, José Ramón Blanco, Lara García, José Antonio Oteo; Hospital de la Santa Creu i Sant Pau, (Barcelona): Natividad de Benito, MercèGurguí, Cristina Pacho, Roser Pericas, Guillem Pons; Hospital Santiago de Compostela, (A Coruña): M. Álvarez, A. L. Fernández, Amparo Martínez, A. Prieto, Benito Regueiro, E. Tijeira, Marino Vega; Hospital Santiago Apóstol, (Vitoria): Andrés Canut Blasco, José Cordo Mollar, Juan Carlos Gainzarain Arana, Oscar García Uriarte, Alejandro Martín López, Zuriñe Ortiz de Zárate, José Antonio Urturi Matos; Hospital SAS Línea, (Cádiz): M Belén Nacle, Antonio Sánchez, Luis Vallejo; Hospital Clínico Universitario Virgen de la Arrixaca (Murcia): José M Arribas Leal, Elisa García Vázquez, Alicia Hernández Torres, Joaquín Ruiz Gómez, Gonzalo de la Morena Valenzuela; Hospital de Pontevedra, (Pontevedra): Nicolás Bayón, M Dolores Díaz, Darío Durán, Juan Carlos Rodríguez, Antonio Moreno; Hospital de Txagorritxu, (Vitoria): Ángel Alonso, Javier Aramburu, Felicitas Elena Calvo, Anai Moreno Rodríguez, Paola Tarabini-Castellani.

Author contributions

Conceptualization: Maria Carmen Fariñas, Gerardo Blanco, Arístides de Alarcón, Jose Antonio Parra.

Data curation: Luis Hernandez, Gerardo Blanco, Regino Rodriguez-Alvarez, Daniel Romeu Vilar, Arístides de Alarcón, Miguel Angel Goenaga, Mar Moreno.

Formal analysis: Maria Carmen Fariñas, Jose Antonio Parra.

Investigation: Patricia Muñoz, Gerardo Blanco, Regino Rodriguez-Alvarez, Daniel Romeu Vilar, Arístides de Alarcón, Jose Antonio Parra.

Methodology: Jose Antonio Parra.

Project administration: Jose Antonio Parra.

Supervision: Maria Carmen Fariñas, Jose Antonio Parra.

Writing – original draft: Jose Antonio Parra.

Writing – review & editing: Maria Carmen Fariñas, Jose Antonio Parra.

José A. Parra: 0000-0002-9259-2730.

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