



Case Report

# Perimyocarditis as First Manifestation of Systemic Lupus Erythematosus Successfully Treated with Heart Failure and Immunosuppressive Therapy

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**Abstract:** Systemic lupus erythematosus (SLE) myocarditis is presumed to be rare, but associated with adverse outcomes. If SLE diagnosis has not previously been established, its clinical presentation is often unspecific and difficult to recognize. Furthermore, there is a lack of data in the scientific literature regarding myocarditis and its treatment in systemic immune-mediated diseases, leading to its late recognition and undertreatment. We present the case of a young woman whose first lupus manifestations included acute perimyocarditis, among other symptoms and signs that provided clues to the diagnosis of SLE. Transthoracic and speckle tracking echocardiography were helpful in detecting early abnormalities in the myocardial wall thickness and contractility while waiting for cardiac magnetic resonance. Since the patient presented with acute decompensated heart failure (HF), HF treatment was promptly started in parallel with immunosuppressive therapy, with a good response. In the treatment of myocarditis with heart failure, we were guided by the clinical signs, echocardiographic findings, biomarkers of myocardial stress, necrosis, and systemic inflammation, as well as markers of SLE disease activity.

**Keywords:** systemic lupus erythematosus; heart failure; myocarditis; pericarditis; speckle tracking echocardiography; transthoracic echocardiography; immunosuppressants; glucocorticoids; therapy; biomarkers



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## 1. Introduction

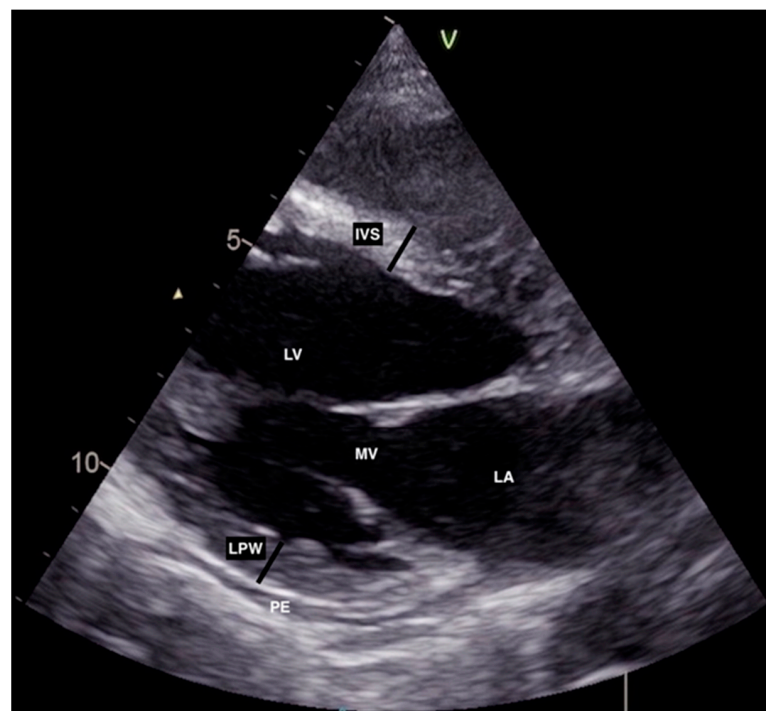
Systemic lupus erythematosus (SLE) is an autoimmune disease more common in women of childbearing age and can affect any organ. Cardiac disease is frequent among SLE patients, and can manifest as conduction abnormalities, valvular, pericardial, myocardial, endocardial, and coronary artery disease, with cardiovascular events being the major cause of premature death [1]. Despite a general decline in SLE mortality over the past decades, cardiovascular mortality remains unchanged [2]. Pericarditis is the most common echocardiographic finding, occurring in more than half of SLE patients [3]. Lupus myocarditis has a prevalence of 0.4–16%, and since it can be life-threatening, we must suspect myocarditis in every SLE patient with HF [4]. Cardiac magnetic resonance (CMR) appears to be the most precise non-invasive diagnostic tool, although not widely available, while right heart catheterization with endomyocardial biopsy (EMB) should be considered when it is not entirely clear that SLE is the cause of myocarditis. Transthoracic echocardiography (TTE) may reveal abnormalities of the left ventricle (LV) systolic and diastolic function, and both global and segmental areas of hypokinesia can be detected. Depending on the degree of myocardial inflammation, chamber size and myocardial wall thickness reflecting interstitial edema can vary [5]. In addition, speckle tracking echocardiography (STE) can detect

subclinical abnormalities in myocardial contractility, and could be helpful in diagnosing myocarditis, especially in its initial stages [6]. Moreover, it was shown that the localizations of myocarditis area that showed in CMR correspond to areas with depressed longitudinal strain in echocardiography [6]. Acute myocarditis in lupus is often accompanied by pericarditis, and treatment of both has not been assessed in controlled trials and is so far mostly based on expert opinions and case reports published in the literature. Here, we present a patient in whom acute heart failure (HF) due to acute perimyocarditis was the first SLE manifestation and who was successfully treated with high doses of methylprednisolone (MP) and hydroxychloroquine (HCQ) in parallel with standard HF therapy.

## 2. Case Description

A 30-year-old woman was hospitalized at the internal medicine department due to recurrent biliary colic and suspicion of the development of acute calculous cholecystitis with a mildly elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Treatment with analgesics, spasmolytics, and antibiotics was started. Despite the treatment, three days later, the patient became febrile ( $>38.5^{\circ}\text{C}$ ), developed a maculopapular rash on the arms and torso, a malar rash, and had dyspnea with elevated N-terminal pro b-type natriuretic peptide (NT-proBNP) (9373 ng/L, normal range:  $<208$  ng/L) and without other clinical signs of heart failure. Additionally, our patient reported chest pain with elevated hs-troponin I (293 ng/L, normal range:  $<39$  ng/L), with sinus tachycardia in the ECG but without signs of ischemia in the serially repeated ECGs (Table 1). The patient also had a low level of albumin, an elevated level of fibrinogen, and a normal value of ferritin, as markers of acute inflammation.

Furthermore, abdominal ultrasound at that time showed no signs of acute cholecystitis, while on TTE, there was hypo-contraction in the basal segment of the anterior, septal, and lateral walls with preserved wall thickness and LV diameter, mildly reduced LV ejection fraction (EF) (45%), reduced global longitudinal peak strain (GLPS) -15.1%, moderate mitral regurgitation (MR), diastolic dysfunction (DD) grade 3, and a small pericardial effusion (2–3 mm) (Figures 1 and 2).



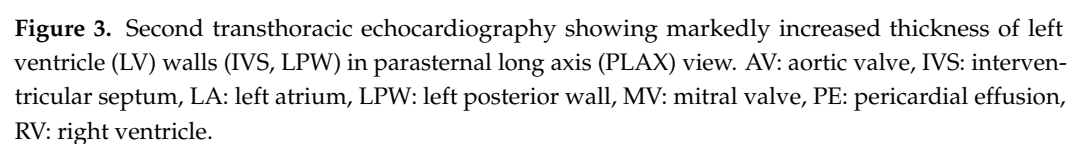
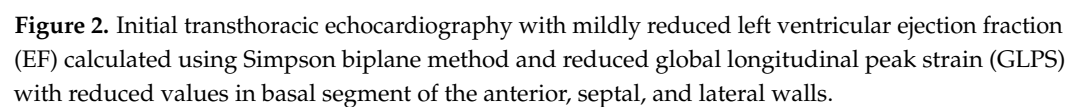
**Figure 1.** Initial transthoracic echocardiography showing left ventricular wall thickness (IVS, LPW) within normal range in parasternal long axis (PLAX) view. IVS: interventricular septum, LA: left atrium, LV: left ventricle, LPW: left posterior wall, MV: mitral valve, PE: pericardial effusion.

**Table 1.** Laboratory data upon admission and 24 months after initiation of heart failure and immuno-suppressive therapy.

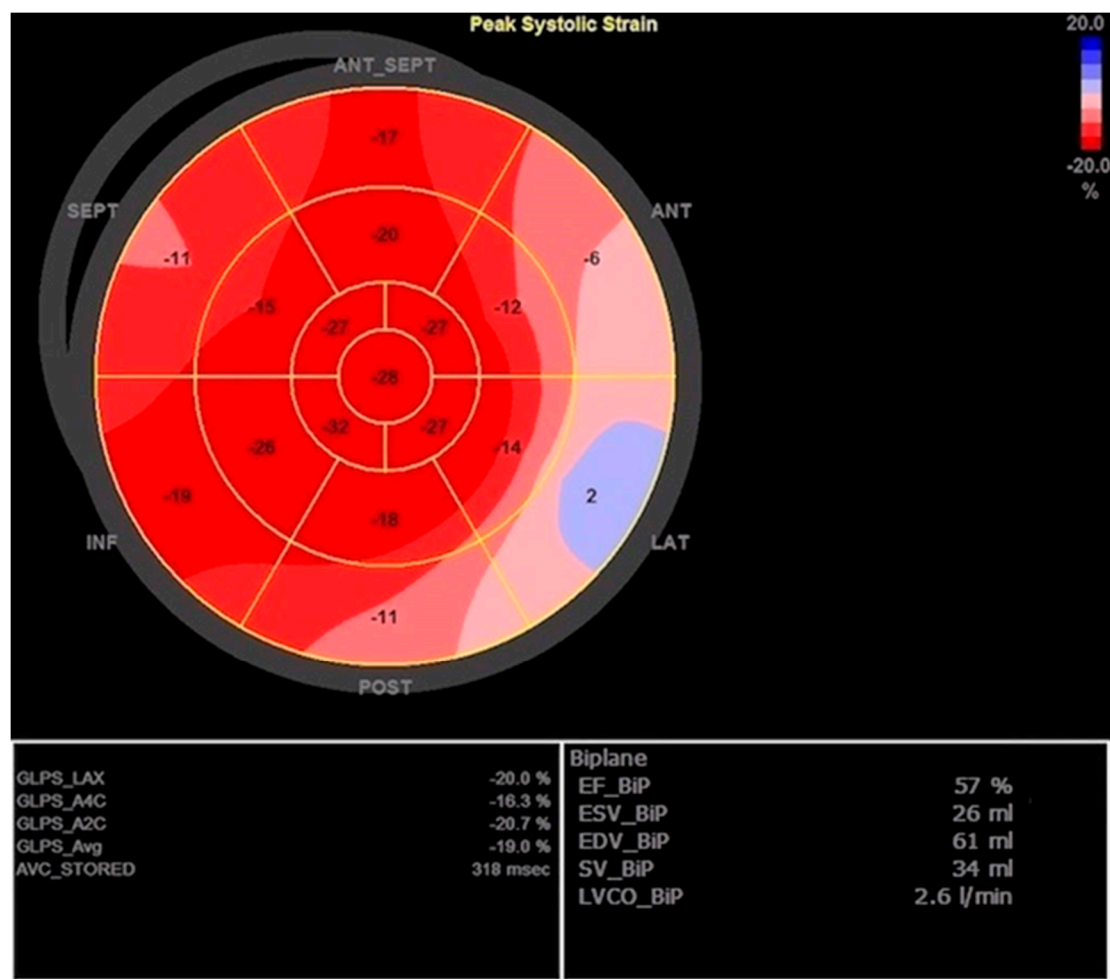
	Reference Range	Admission	After 24 Months
White blood cell count ( $10^9/L$ )	3.4–9.7	3.1	6.9
Hemoglobin (g/L)	138–175	116	137
Platelets ( $10^9/L$ )	158–424	132	193
CRP (mg/dL)	<5.0	26.5	3.5
ESR (mm/3.6 ks)	4–24	28	11
Albumin (g/L)	41–51	36	44
Ferritin ( $\mu g/L$ )	10–158	61	12
Fibrinogen (g/L)	1.8–3.5	3.9	2.2
Procalcitonin ( $\mu g/L$ )	0.5–2	1.30	<0.5
Creatinine ( $\mu mol/L$ )	49–90	68	51
24 h urine protein (mg/24 h)	<150	54	145
hs-troponin I (ng/L)	<39	293	36
NT-proBNP (ng/L)	<208.4	9373	135
Direct antiglobulin test		Negative	
C3 (g/L)	0.89–1.87	0.72	1.0
C4 (g/L)	0.17–0.38	0.17	0.28
Antinuclear antibody *	Positive > 1	>55	Not performed
Anti-dsDNA antibody * (IU/mL)	Positive > 15	<0.7	1.2
Anti-Smith (Sm) antibody		Not performed	Not performed
Anticardiolipin antibody IgM * (MPL-U/mL)	Positive > 40	1.1	0.7
Anticardiolipin antibody IgG * (GPL-U/mL)	Positive > 40	1.2	0.9
Beta2-Glycoprotein I IgM * (EliA U/mL)	Positive > 10	<2.4	<2.4
Beta2-Glycoprotein I IgG * (EliA U/mL)	Positive > 10	<0.8	<0.8
Lupus anticoagulant		Negative	Not performed

CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, NT-proBNP: N-terminal pro b-type natriuretic peptide, C: complement, \* FEIA Phadia 200.

Since there was concern about a possible associated allergic reaction (rash) during the treatment with antibiotics, the patient immediately received a low dose of corticosteroids and antihistamines while extensive evaluation was initiated in parallel with guideline-directed treatment for HF. Blood and urine cultures were negative, as were viral panels for cardiotropic infectious agents. All tumor markers and the interferon-gamma release assay for latent tuberculosis infection were in the reference range. A clinical immunologist examined the patient and recommended diagnostic workup under suspicion of SLE. Coronary angiography did not show obstructive coronary artery disease. Since the patient manifested symptomatic HF with a slightly reduced EF but with the possibility of fast deterioration, we introduced diuretics, beta-blockers, aldosterone antagonists, and sacubitril/valsartan, as well as ibuprofen due to signs of pericarditis. Interestingly, a few days later, the TTE showed a markedly increased thickness of the LV walls with normal EF (57%), GLPS of −19%, mild MR, DD grade 1, and progression of pericardial effusion to 10 mm. The GLPS abnormalities were again found in the basal segments of the anterior, septal, and lateral walls (Figures 3 and 4).

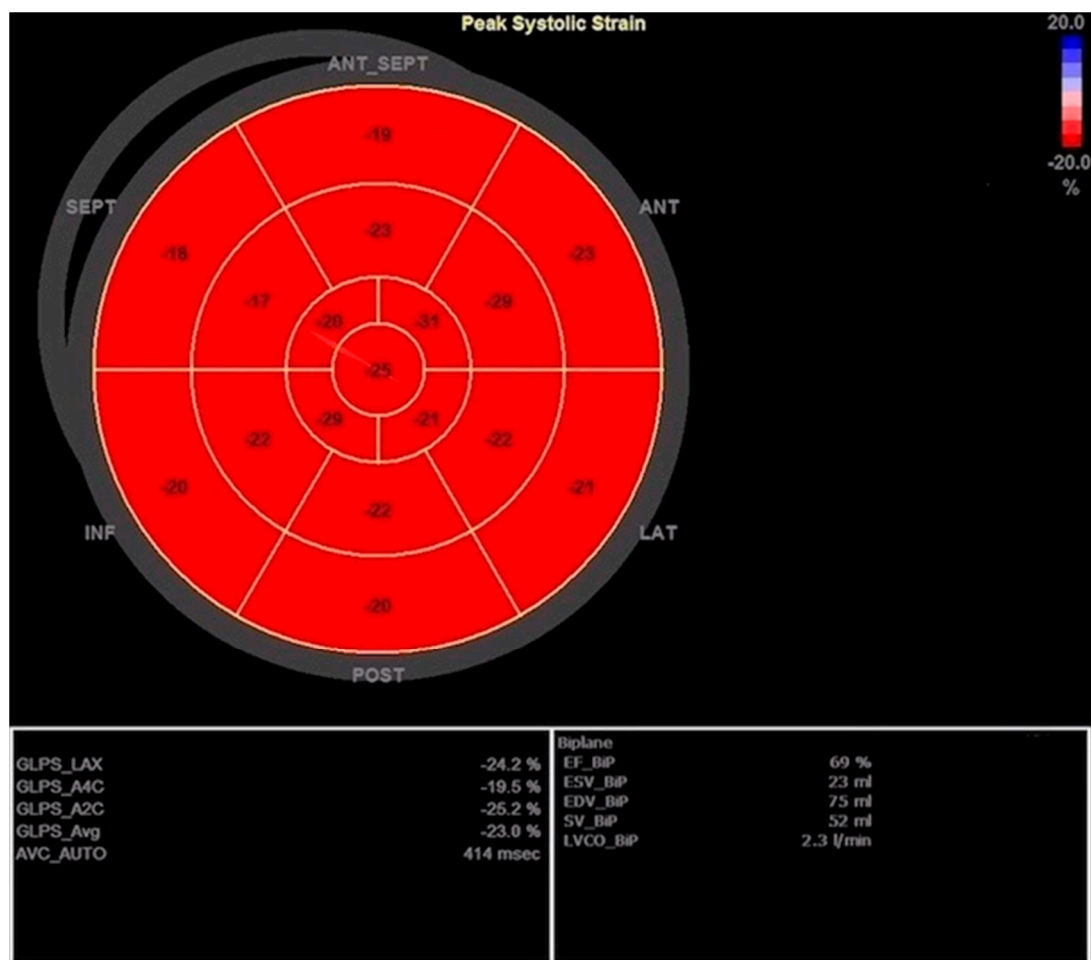






**Figure 4.** Second transthoracic echocardiography with improved left ventricle ejection fraction (EF) and improved global longitudinal peak strain (GLPS) but still noticeable reduced regional strain values.

This increased thickness of myocardial walls in the second TTE likely reflected interstitial edema due to suspected inflammation, and the rapid EF improvement was explained by better loading conditions due to diuretics and HF therapy. Nevertheless, depressed GLPS was still noted within several segments of the LV, raising suspicion of myocarditis. After immunological workup, the diagnosis of SLE was made; the 2019 EULAR/ACR criteria for SLE were fulfilled: leukopenia presented on several occasions before according to available medical documentation, strongly positive antinuclear antibodies, complement (C) consumption low C3, pericarditis, myocarditis, fever, and acute cutaneous lupus (Table 1). Additionally, the patient reported photosensitivity and recurrent cervical lymphadenopathy, and had persistently low lymphocyte count with occasional thrombocytopenia, but not less than  $100 \times 10^9/L$ . Therefore, in addition to HF therapy, HCQ 200 mg and MP 2 mg/kg/day were introduced, and ibuprofen was ceased. Soon after initiating the above-mentioned therapy, the patient reported regression of all symptoms, and there was a significant decrease in NT-proBNP, normalization of leukocyte and platelet count, troponin, ESR, CRP, and fibrinogen, and the MP was reduced to 0.5 mg/kg/day. Meanwhile, we obtained CMR that confirmed the myocarditis diagnosis, and the results were comparable with abnormalities detected in the TTE. More precisely, cardiac magnetic resonance performed 2 weeks after the first heart failure manifestation described focal tissue edema, especially in the anterior region and the interventricular septum, in the area that was most thickened in the TTE. After only 3 weeks of therapy, TTE was completely normal (Figure 5) with an EF of 69%, and we tapered MP to 0.3 mg/kg/day.



**Figure 5.** Completely recovered left ventricle ejection fraction (EF) and global longitudinal peak strain (GLPS) with no signs of hypokinesia 3 weeks after initiating guideline-directed heart failure therapy concomitantly with immunosuppressive therapy.

Sacubitril/valsartan and aldosterone antagonists were completely discontinued after 6 months, and the dose of MP was gradually decreased with total discontinuation after 12 months (after 6 months, MP dose was 8 mg daily). Now, 24 months after the first manifestation of SLE, the patient is still in complete clinical and immunological disease remission and is continuously taking bisoprolol at a minimal dosage and 200 mg of HCQ (Table 1).

### 3. Discussion

Clinicians must be aware of unusual SLE presentations such as acute HF due to SLE-myocarditis, with only 0.37% of patients presenting as such, but with potentially fatal outcomes: cardiogenic shock or malignant arrhythmias [7]. The diagnosis of SLE itself, especially SLE-myocarditis, is challenging in clinical practice if the patient has not had a previous diagnosis of lupus or lupus signs and symptoms. Although EMB is considered the gold standard for the diagnosis of myocarditis, it is not a routine procedure because it is invasive and has a risk of sampling error. Consequently, to establish the correct diagnosis of SLE-myocarditis, we must combine the patient's previous medical history, current signs and symptoms, laboratory tests, TTE with STE, CMR, and EMB if available, and exclude other common causes of myocarditis. In our case, we established a diagnosis of SLE according to the 2019 EULAR/ACR classification criteria and after the exclusion of other causes of myocarditis. In the treatment of myocarditis in SLE, we were guided by the 2016 ESC guidelines for HF valid at that time, the position statement of the ESC Working Group on

Myocardial and Pericardial Disease, EULAR recommendations for the management of SLE, previously reported case reports, expert opinions, and reviews [8–15]. Most of the treatment evidence is derived from case reports, case-control, and cohort studies. According to the literature, the treatment of lupus myocarditis requires an initial period of intensive immunosuppression to decrease disease activity, followed by a longer period of less intensive therapy to consolidate the response and prevent relapses [9,12]. The standard of care for patients with severe myocarditis includes high-dose glucocorticoids plus intravenous cyclophosphamide or another immunosuppressant such as rituximab, and azathioprine is usually used as steroid-sparing maintenance therapy [9,10,12,15]. There are few data in the literature regarding the optimal immunosuppressive regimen in the treatment of SLE myocarditis with mildly reduced ejection fraction, and some data suggest that patients with less severe disease exhibited good left ventricular ejection fraction recovery without cyclophosphamide or other immunosuppressants [15,16]. Our patient was initially treated with a high dose of MP: 2 mg/kg/day (with the possible implication of early-started low-dose corticosteroids due to rash and concern of an allergic reaction), and the high dose was reduced to a moderate dose (0.3 mg/kg/day) after TTE and laboratory confirmation of myocardial recovery with further gradual dose reduction. Ibuprofen was stopped since non-steroidal anti-inflammatory drugs (NSAIDs) could enhance inflammation and increase mortality, which has been shown in animal models of myocarditis, although recent findings suggest that treatment with NSAIDs does not affect outcome in patients with acute myocarditis or myopericarditis [10,11,17]. In our opinion, patients with HF due to SLE should be treated in parallel with HF guideline-directed therapy to reduce the risk of further myocardial damage and potential complications, although the optimal duration of the treatment is not yet well established. Additionally, the optimal duration of glucocorticoid therapy is not known in lupus myocarditis, but we were guided by the patient's clinical signs and symptoms and laboratory and TTE findings. The treatment should be started as soon as possible to prevent further damage by suppressing the inflammation that leads to heart remodeling and fibrosis. Furthermore, in relation to changes detected in TTE with STE, a combination of widely available laboratory parameters/biomarkers such as troponin and NT-proBNP (biomarkers of myocardial injury), anti-dsDNA, C3 and C4 (biomarkers of SLE disease activity), and ESR, CRP, albumin, and fibrinogen (biomarkers of systemic inflammation) could be used not only for early diagnosis but also for SLE-myocarditis follow-up and therapy management [18,19]. In the absence of a rapid response to glucocorticoids, another immunosuppressant such as cyclophosphamide, mycophenolate mofetil, azathioprine, or rituximab should be introduced into the therapy [14,15,20]. Additionally, we must bear in mind that the treatment of myocarditis in systemic autoimmune or autoinflammatory disease should be tailored to each individual patient depending on which organs are simultaneously affected by the disease. We believe that timely initiated immunosuppressive glucocorticoid therapy in parallel with HF therapy with neurohumoral blockade was the key to successful treatment in the case of our patient.

#### 4. Conclusions

Despite its low prevalence, lupus myocarditis should be considered as a differential diagnosis in patients with new-onset heart failure. Early diagnosis, treatment with immunosuppressants according to the severity of myocarditis, and heart failure therapy, as well as close monitoring of the response to therapy, may lead to a favorable outcome. This case demonstrates a rare presentation of SLE myocarditis with rapid clinical recovery on glucocorticoid and heart failure therapy. Of course, an optimal treatment strategy cannot be made on the basis of a single case report. Randomized trials with larger numbers of patients conducted by cardiologists and clinical immunologists/rheumatologists are necessary to provide guidelines and recommendations for diagnosing and managing myocarditis in autoimmune and autoinflammatory systemic diseases.

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**Institutional Review Board Statement:** Since only normal clinical practice is described in this case report, formal ethical approval by the Independent Review Board was not required in accordance with the policy of our institution.

**Informed Consent Statement:** Written informed consent has been obtained from the patient to publish this paper.

**Data Availability Statement:** The original data generated and analyzed for this study are included in the published article. Further inquiries can be directed to the corresponding author.

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