KLİNİKİ MÜŞAHİDƏ AÇIQ GİRİŞ (OPEN ACCESS)

LUPUS MYOCARDITIS PRESENTING AS NEW ONSET HEART FAILURE: THE TWO-CASE REPORTS

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ABSTRACT

Background. Lupus myocarditis is a rare but potentially life-threatening manifestation of systemic lupus erythematosus (SLE). Because its clinical presentation is nonspecific, early diagnosis remains challenging and requires a high index of suspicion. Cardiac magnetic resonance imaging (CMR) has become an important non-invasive modality for detecting myocardial inflammation.

Case presentation. We report two female patients with lupus myocarditis who presented with new-onset heart failure. The first patient was a 21-year-old woman admitted with progressive dyspnea, lower extremity edema, and fever. The second patient was a 65-year-old woman presenting with worsening dyspnea, generalized weakness, edema, and oliguria.

Transthoracic echocardiography demonstrated reduced left ventricular ejection fraction and global hypokinesia in both patients. CMR revealed diffuse myocardial edema with nonischemic late gadolinium enhancement, confirming the diagnosis of lupus myocarditis. Both patients received immunosuppressive therapy together with guideline-directed heart failure treatment. Three months later, both patients demonstrated significant clinical improvement with recovery of left ventricular systolic function.

Conclusion. These cases emphasize the importance of considering lupus myocarditis in SLE patients presenting with new-onset heart failure and highlight the diagnostic value of CMR for early diagnosis and treatment monitoring.

Keywords: Systemic lupus erythematosus, lupus myocarditis, cardiac magnetic resonance imaging, heart failure, SLE

Case Report

Case 1

A 21-year-old woman was admitted to our clinic with a one-month history of progressive fatigue, generalized weakness, exertional dyspnea, lower extremity edema, cough, and intermittent fever (37–38°C). Her medical history was significant for hypothyroidism. On admission, blood pressure was 120/78 mmHg, heart rate 114 beats/min, respiratory rate 28 breaths/min, and body temperature 36.5°C. Physical examination revealed muffled heart sounds and diminished vesicular breath sounds on lung auscultation.

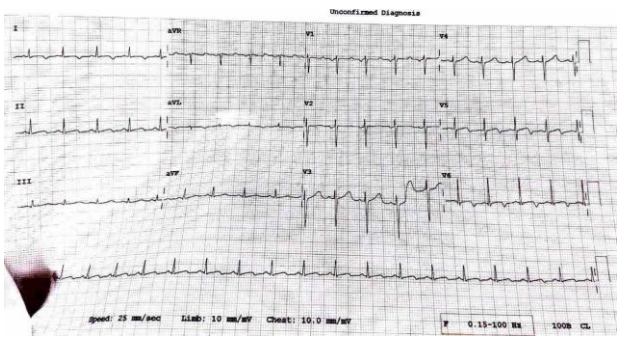
Laboratory investigations demonstrated serum creatinine of 1.02 mg/dL, hyperkalemia (6.32 mmol/L), positive antinuclear antibodies, elevated anti-double stranded DNA antibodies (466 IU/mL), decreased complement C3 and C4 levels, and markedly elevated NT-proBNP (20,840 pg/mL). Cardiac troponin was negative.

Electrocardiography showed sinus tachycardia (Figure 1).

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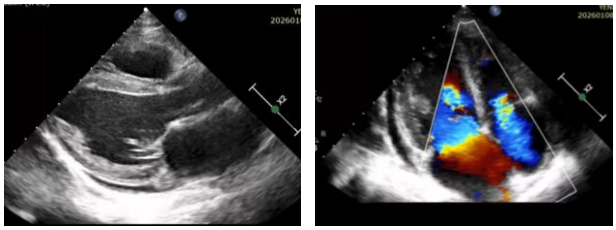
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Figure 1. Electrocardiogram.



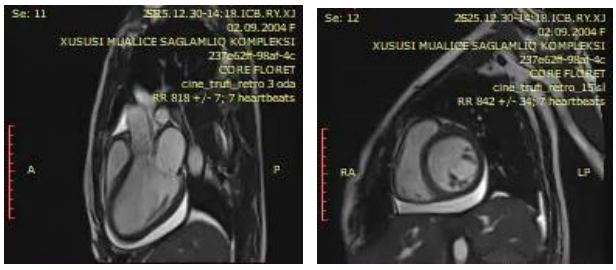
Transthoracic echocardiography demonstrated a left ventricular ejection fraction (LVEF) of 45% with global hypokinesia, moderate mitral and tricuspid regurgitation, pulmonary artery systolic pressure of 65 mmHg, and minimal pericardial effusion (Figure 2).

Figure 2. Transthoracic echocardiogram



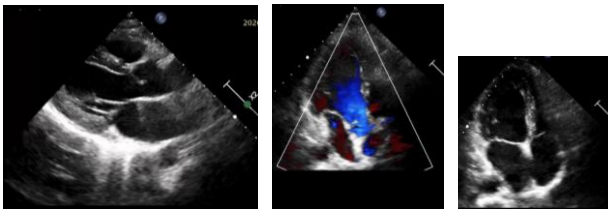
Cardiac magnetic resonance imaging revealed mildly reduced left ventricular systolic function (LVEF 46%), diffuse myocardial edema, non-ischemic late gadolinium enhancement, minimal pericardial effusion, and right pleural effusion, consistent with acute lupus peri-myocarditis (Figure 3).

Figure 3. Cardiac magnetic resonans imaging.



The patient received intravenous methylprednisolone followed by gradual dose reduction, together with guideline-directed heart failure therapy including sacubitril/valsartan, bisoprolol, spironolactone, and diuretics. At three-month follow-up, symptoms resolved completely. Echocardiography demonstrated recovery of left ventricular systolic function (LVEF 55%), normalization of pulmonary artery pressure, and marked reduction of valvular regurgitation (Figure 4).

Figure 4. Echocardiographic imaging after treatment



Case 2

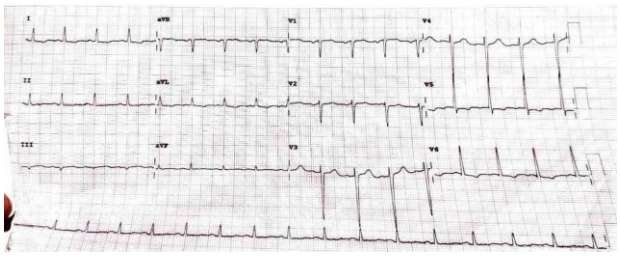
A 65-year-old woman presented with generalized weakness, worsening dyspnea, fever, oliguria, dysuria, and bilateral lower limb edema. Symptoms had started approximately six months earlier and had markedly worsened during the previous five days. Her medical history included arterial hypertension and hypothyroidism.

On admission, blood pressure was 200/118 mmHg, heart rate 105 beats/min, respiratory rate 20 breaths/min, and oxygen saturation was 100% on room air. Physical examination revealed muffled heart sounds and diminished breath sounds bilaterally.

Laboratory testing demonstrated positive ANA, elevated anti-dsDNA antibodies (79.6 IU/mL), positive direct and indirect Coombs tests, elevated NT-proBNP (34,118 pg/mL), and positive cardiac troponin.

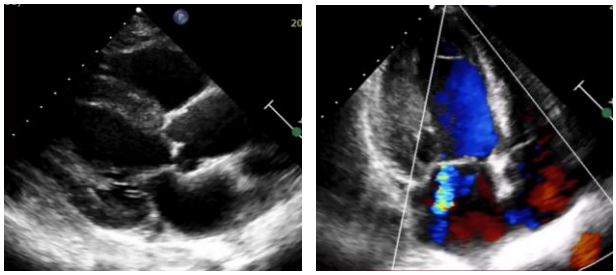
Electrocardiography revealed sinus tachycardia (Figure 5).

Figure 5. Electrocardiogram



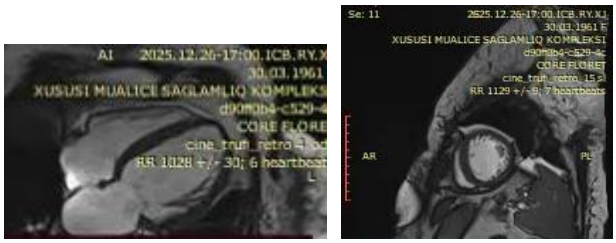
Echocardiography demonstrated global hypokinesia with LVEF of 45%, left ventricular dilatation, moderate pulmonary hypertension (SPAP 50 mmHg), moderate-to-severe mitral regurgitation, mild aortic regurgitation, moderate tricuspid regurgitation, and minimal pericardial effusion (Figure 6).

Figure 6. Transthoracic echocardiogram



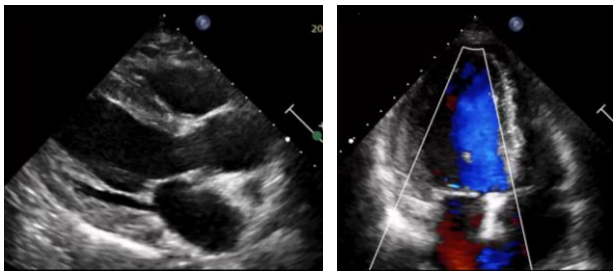
Cardiac magnetic resonance imaging showed diffuse myocardial edema, impaired left ventricular systolic function, biatrial enlargement, severe mitral regurgitation, and nonischemic late gadolinium enhancement compatible with active lupus myocarditis (Figure 7).

Figure 7. Cardiac magnetic resonance imaging.



The patient was treated with pulse-dose methylprednisolone followed by oral corticosteroids, cyclophosphamide, hydroxychloroquine, and optimized heart failure therapy, including sacubitril/valsartan, empagliflozin, bisoprolol, and antihypertensive medication. At the three-month follow-up, the patient's symptoms had significantly improved, LVEF increased to 55%, pulmonary artery pressure decreased to 30 mmHg, and valvular regurgitation was substantially reduced (Firuge 8).

Figure 8. Echocardiographic imaging after treatment



Discussion

Lupus myocarditis is an uncommon but serious manifestation of systemic lupus erythematosus (SLE), with clinically apparent disease reported in approximately 3–9% of patients, although autopsy studies suggest a substantially higher prevalence due to frequent subclinical myocardial involvement [6,8].

Early diagnosis remains challenging because symptoms are nonspecific and may mimic other causes of heart failure, including viral myocarditis, ischemic cardiomyopathy, hypertensive heart disease, and stress-induced cardiomyopathy [1,6].

Both patients presented in this report developed new-onset heart failure accompanied by impaired left ventricular systolic function and global hypokinesia on transthoracic echocardiography. These findings raised suspicion of myocarditis; however, echocardiography alone cannot reliably distinguish lupus myocarditis from other causes of non-ischemic cardiomyopathy [3,7]. Cardiac magnetic resonance imaging (CMR) therefore played a decisive role in confirming the diagnosis by demonstrating myocardial edema and nonischemic late gadolinium enhancement, findings consistent with active myocardial inflammation.

An interesting finding in our cases was the difference in cardiac biomarker profile. The younger patient had markedly elevated NT-proBNP despite negative troponin levels, whereas the older patient demonstrated elevation of both NT-proBNP and cardiac troponin. Previous studies have shown that cardiac troponin may remain normal in lupus myocarditis despite active myocardial inflammation, whereas NT-proBNP more consistently reflects ventricular dysfunction and heart failure severity. Therefore, a normal troponin level should not exclude the diagnosis in patients with compatible clinical findings.

Both patients responded favorably to high-dose corticosteroid therapy combined with guideline-directed medical therapy for heart failure. Recovery of left ventricular systolic function and improvement of clinical symptoms were observed within three months. These findings support previous reports demonstrating that early recognition and prompt immunosuppressive treatment may reverse myocardial dysfunction and improve prognosis [4].

Our case series highlights the complementary role of multimodality imaging in lupus

myocarditis. Echocardiography remains the first-line imaging modality for evaluating ventricular function, valvular abnormalities, pulmonary hypertension, and pericardial effusion. However, CMR provides superior tissue characterization, allowing detection of myocardial edema and fibrosis without the need for invasive procedures. Endomyocardial biopsy remains the histopathological gold standard but is rarely performed because of its invasive nature, limited sensitivity due to patchy myocardial involvement, and potential complications [2,5].

These two cases emphasize that lupus myocarditis should always be considered in patients with SLE presenting with unexplained heart failure, even when cardiac biomarkers are not uniformly elevated. Early CMR evaluation together with prompt initiation of immunosuppressive and heart failure therapy may result in complete or near-complete recovery of cardiac function

Conclusions

Lupus myocarditis is a rare but potentially life-threatening manifestation of systemic lupus erythematosus that requires early recognition and prompt treatment. In patients presenting with new-onset heart failure, especially in the presence of active autoimmune disease, lupus myocarditis should be included in the differential diagnosis. Cardiac magnetic resonance imaging represents a valuable non-invasive diagnostic tool for confirming myocardial inflammation and guiding clinical management. Early immunosuppressive therapy combined with guideline-directed heart failure treatment may lead to significant recovery of left ventricular function and favorable clinical outcomes.

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