WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Lupic Myocarditis Epidemiological Aspects and the Role of Magnetic Resonance Imaging in Positive Diagnosis

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DOI : https://doi.org/10.55248/gengpi.6.0325.1195

ABSTRACT:

Lupic myocarditis is a rare but serious cardiac manifestation of systemic lupus erythematosus (SLE). It results from autoimmune inflammation of the myocardium, leading to potential complications such as arrhythmias, heart failure, and sudden cardiac death. Cardiac magnetic resonance imaging (CMR) is a key non-invasive tool for diagnosing lupic myocarditis. Early diagnosis using CMR improves prognosis by enabling timely immunosuppressive therapy, preventing long-term cardiac dysfunction. Regular cardiac monitoring remains essential in SLE patients at risk.

Objective:
The objective of our study is to highlight the involvement of the left ventricle in cases of SLE, assess its morphology and function, and emphasize the contribution of MRI in the evaluation and diagnostic guidance.

Methods:
This is a descriptive retrospective study of a series of 5 patients with SLE, referred to our MRI unit from various departments, particularly internal medicine, for suspected lupic myocarditis.

Results:
The study and the review of literature demonstrate that cardiac MRI is an indispensable tool in the diagnosis, management, and follow-up of lupic myocarditis, offering superior sensitivity and specificity compared to other imaging modalities.

Conclusion:
Lupic myocarditis is a serious condition with pancarditis, often affecting ventricular function. Diagnosis relies on clinical suspicion and echocardiography, with cardiac MRI being the most effective non-invasive tool. The prognosis can be poor in severe cases, and treatment remains unclear.

Keywords: Lupic Myocarditis, MRI, Myocardial biopsy,

Consent: The authors confirm that written consent for the submission and publication of this case,

Publication Ethics (COPE) guidance.

Conflict of interest: None declared.

Funding: None declared.

Consent: The authors confirm that written consent for the submission and publication of this case, including images, has been obtained from the patients in line with the Committee on Publication Ethics (COPE) guidance.

Availability of Data and Materials: Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Conflict of interest: None declared.

Funding: None declared.

Figure Legend:

1. **Distribution of patients by sexe**
2. **Distribution of patients by age**

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INTRODUCTION

Acute myocarditis is defined as myocardial inflammation caused by a wide range of infectious agents (viruses, bacteria, and others), systemic diseases, medications, and toxins. Clinical presentation can vary from asymptomatic forms to acute fulminant cases, as well as sub-acute or chronic presentations.

The European Society of Cardiology's expert group emphasizes the importance of myocardial biopsy for a definitive diagnosis, though imaging methods are often secondary. However, in practice, biopsy is rarely performed due to associated risks.

Histologically, acute myocarditis is characterized by inflammatory infiltrates in the myocardium with necrosis and/or myocyte damage, explaining the myocardial dysfunction seen with imaging methods.

Echocardiography is typically the first-line exam, but findings such as left ventricular dilation and segmental hypokinesis/akinesis are non-specific.

Cardiac MRI is the key diagnostic tool for confirming acute myocarditis, revealing inflammatory lesions with contrast uptake, often in the subepicardial region. T2-weighted imaging sequences help identify edematous segments that appear as hyperintense areas in the myocardium. Dynamic sequences assess left ventricular systolic function, segmental motion, and heart cavity sizes, thicknesses, and volumes. After gadolinium injection, late gadolinium enhancement (LGE) highlights myocardial damage, typically subepicardial or mid-ventricular in location, distinguishing it from ischemic processes (which typically show endocardial involvement).

This work will focus on myocarditis in systemic lupus erythematosus (SLE). We will present literature data on the primary pathophysiological mechanisms, the role of cardiac MRI in diagnosis and management, through 5 patient cases with suspected acute myocarditis based on clinical, biological, and/or echocardiographic findings.

MATERIALS AND METHODS

The objective of our study is to highlight left ventricular involvement in systemic lupus erythematous (SLE), assess its morphology and function, and emphasize the role of MRI in evaluation and diagnostic orientation.

Through five cases of patients with lupus erythematous referred to our MRI unit from various departments, notably internal medicine for suspected lupus myocarditis, we investigated:

- The confirmed diagnosis of disseminated lupus erythematous in all patients
- The presence of a clinical context suggestive of myocarditis
- Suspected diastolic or systolic dysfunction of the left ventricle on echocardiography

All patients meeting these criteria were included and underwent cardiac MRI.

The data collected from the study population are as follows:

a) **Epidemiological Data:**

Demographic data:

- Name
- Sex
- Age
- Circumstances of discovery
- Signs of heart failure

Complementary examination data:

- ECG abnormalities

- Echocardiographic results

b) **Imaging Data (Cross-sectional Imaging):**

Cardiac MRI:

- Cardiac chambers: analysis of the ventricles (end-diastolic volume, end-systolic volume, ejection fraction), ventricular and atrial diameters.
- Tagging: assessment of intrinsic contractility abnormalities
- Detection of myocardial edema
- Early enhancement (hyperemia)
- Late enhancement following gadolinium injection

Regarding MRI characteristics, an examination protocol was established: all examinations were performed on a Philips 3T MRI system, synchronized with the electrocardiogram and retrospective acquisition. Sequences were acquired during apnea.

We used Gadovist® as the gadolinium chelate.

As for the acquisition protocol, as in any cardiac MRI examination, we began with a series of topograms and localization of the three reference cardiac planes (long axis, short axis, and four-chamber view).

For CINE sequences, a classic multiplanar CINE MRI study was conducted using balanced steady-state free precession (Balanced FFE®) sequences, with slices acquired from the base to the apex along the short axis covering the entire heart, as well as 2-chamber views of the left and right ventricles, and 3- and 4-chamber views.

For T2-weighted sequences, we used specific sequences to evaluate myocardial edema, which appears as hyperintense, particularly the STIR sequence (rapid spin-echo sequence with triple inversion recovery to suppress signals from fat and circulating blood, thereby enhancing the contrast between edema, healthy myocardium, and the left ventricular cavity).

Additionally, a first-pass perfusion study was performed to assess early enhancement, followed by the study of late enhancement 5 to 10 minutes after gadolinium injection.

Regarding MRI data analysis, it was performed using Philips-provided software. The measured covariates included the size of both atria, end-diastolic and end-systolic diameters, end-diastolic and end-systolic volumes, and the ejection fraction of both ventricles. Myocardial wall thickness and cardiac mass were also measured. For each patient, any abnormalities in global or segmental cardiac kinetics were specified, along with the analysis of intrinsic contractility of the ventricular walls, ventricular chamber geometry, presence or absence of myocardial edema, hyperemia (early enhancement), and late enhancement, with detailed description of the segmental location and extent.

RESULTS:

A. Clinical, Electrical, and Echocardiographic Characteristics:

1. **Age and Sex**

We present 5 cases. The reference characteristics of the study groups are shown in Table 3.

The mean age of our patients was 37.6 years. The youngest patient was 20 years old, and the oldest patient was 55 years old. There is a notable female predominance, with 80% of patients being female (4 out of 5).

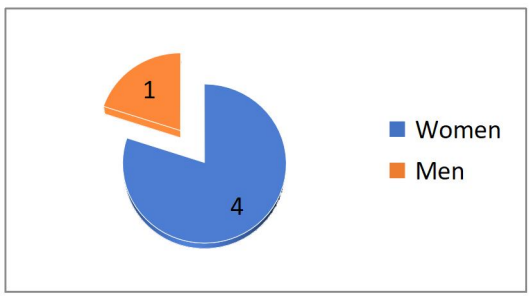

Figure 1: Distribution of patients by sexe

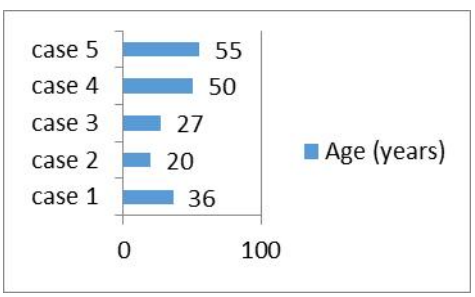

Figure 2: Distribution of patients by age

Clinical Data

All our patients had a confirmed diagnosis of disseminated lupus erythematous, with two patients having mixed connective tissue diseases. One case (Patient No. 3) presented with lupus erythematous associated with thrombocytopenic purpura, while another case (Patient No. 4) had lupus erythematous associated with rheumatoid arthritis.

Clinically, the main symptom was dyspnea in 3 cases, followed by chest pain and palpitations. All our patients had coronary artery disease excluded as a criterion.

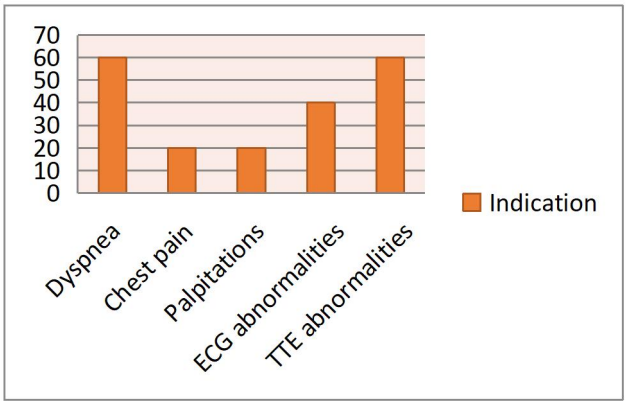

Figure 3: Indication for MRI

An electrocardiogram was performed on all cases and was abnormal in 2 cases. It showed repolarization disturbances in Patient No. 1 and ventricular and supraventricular hyper excitability with numerous premature ventricular beats and supraventricular premature beats in Patient No. 3.
All our patients underwent transthoracic echocardiography, which was abnormal in 3 patients. It revealed left ventricular dysfunction, severe pulmonary hypertension, and in Patient No. 5, mobile elements at the level of the aortic valve, suggestive of Libman-Sacks endocarditis in the context of SLE.

Parameters	Case n°1	Case n°2	Case n°3	Case n°4	Case n°5
Age	36	20	27	50	55
Sexe	Female	Female	Female	Female	Masculine
Lupus	Confirmed	Confirmed	Confirmed + thrombocytopenic purpura	Confirmed + RA	Confirmed
Clinique	atypical chest pain	Dyspnea	Palpitation	Dyspnea	Dyspnea
ECG	Repolarization Abnormalities	Normal	Ventricular and supraventricular hyperexcitability	Not provided	Normal
ETT	Not provided	LV Dysfunction	Normal	Severe PAH	Element in the aortic valve

Figure 4: Summary of epidemiological, clinical, and echocardiographic parameters

B.MRI Data

The cardiac MRI data of our patients are detailed in the tables below.

Case N° 1			
Left Cavities		**Right Cavities**	
EDD	44 mm	**EDD**	24mm
ESD	27 mm	**RA**	34 mm
EDV	63 ml	**Segmental kinetics**	Normal
ESV	24ml	**Late enhancement 10 minutes after**	Absent
Ejection Fraction	62%		

LA	27mm	**Gadolinium**	
Segmental kinetics	Normal		
Edema: PD	Absent		
Late enhancement	Inferior anterior medial-parietal inferoseptal antero-lateral		
Case N° 2			
Left Cavities		Right Cavities	
EDD	56 mm	**EDD**	29 mm
ESD	40 mm	**RA**	36 mm
EDV	149 ml	**Segmental kinetics**	Normal
ESV	79 ml	**Late enhancement 10 minutes after Gadolinium**	Absent
Ejection Fraction	45%		
LA	35 mm		
Segmental Kinetics	Global hypokinesia		
Perfusion Delay	Absent		
Late enhancement	Absent		
Cas N° 3			
Cavités gauche		**Cavités droites**	
EDD	51 mm	**EDD**	26 mm
ESD	31 mm	**RA**	35 mm
EDV	105 ml	**Segmental kinetics**	Normal
ESV	42 ml	**Late enhancement 10 minutes after Gadolinium**	Absent
Ejection fraction	60 %		
LA	31 mm		
Segmental kinetics	Normal		
Perfusion Delay	Absent		
Late enhancement	Absent		
Case N° 4			
Left Cavities		**Right Cavities**	
EDD	42 mm	**EDD**	51 mm
ESD	24 mm	**RA**	55 mm
EDV	108 ml	**Segmental kinetics**	Normal
ESV	39 ml	**Late enhancement 10 minutes after Gadolinium**	Absent
Ejection Fraction	63%		
LA	31 mm		
Segmental Kinetics	Paradoxical septum with altered tagging		

Perfusion delay	Absent		
Late enhancement	Absent		
Case N° 5			
Left Cavities		**Right Cavities**	
EDD	53 mm	**EDD**	29 mm
ESD	35 mm	**RA**	38 mm
EDV	122 ml	**Segmental kinetics**	Normal
ESV	43 ml	**Late enhancement 10 minutes after Gadolinium**	Absent
Ejection Fraction	65%		
LA	36 mm		
Segmental kinetics	Normal		
Perfusion Delay	Absent		
Late enhancement	Absent		

1. **Analysis of Left Cavities:**

Cardiac MRI in myocarditis primarily assesses left ventricular function. It looks for systolic dysfunction and impaired contractility, along with possible myocardial enhancement. Myocardial edema is rarely found due to late imaging. The mean end-diastolic diameter was 49.2 mm. The mean end-systolic diameter was 31.4 mm. The mean left ventricular end-diastolic volume (VTDVG) was 109.4 ml. The mean end-systolic volume (VTSVG) was 45.4 ml.

Four patients had left ventricular diameters and volumes within normal limits. Case No. 2 showed a dilated left ventricle with a DTDVG of 56 mm, DTSVG of 40 mm, VTDVG of 149 ml, and VTSVG of 79 ml.

The ejection fraction was preserved in four cases (>60%) with normal wall kinetics. Case No. 2 had an impaired ejection fraction of 45% with global hypokinesis.

The left atrium was of normal size in all patients.

2. **Analysis of Right Cavities:**

The analysis of right cavities assesses myocarditis extension and associated pathologies.

The mean right ventricular end-diastolic diameter (DTDVD) was 31.8 mm. The mean right atrial diameter was 39.6 mm.

The right ventricle was normal in size and volume in four cases. In Case No. 3, the right atrium was dilated at 55 mm, with a DTDVD of 51 mm.
The septum showed a paradoxical movement, and intrinsic contractility was impaired in the septum. A massive tricuspid regurgitation was observed in this patient. These findings suggest severe pulmonary hypertension, likely due to mixed connective tissue disease.

3. **Study of Enhancement:**

Late enhancement was present only in Case No. 1. It showed diffuse myocardial fibrosis in the inferior, anterior, and anterolateral walls, indicative of lupus myocarditis.

DISCUSSION

A. <u>Epidemiological Data on Age and Sex:</u>

Few studies have focused on lupus myocarditis, with most research addressing cardiac involvement in the three cardiac layers in cases of systemic lupus erythematosus (SLE), predominantly highlighting pericardial involvement.

The most recent Moroccan study was conducted in 2016 by Harouna H. et al. [1], which included 31 patients with cardiac involvement out of 121 SLE cases. Cardiac involvement was predominantly pericarditis of low abundance in 27 cases, while myocarditis was present in only 4 cases. Another Moroccan study conducted in 2015 by Oubelkacem et al. [2], on 165 lupus patients, found that 11 had myocardial involvement. Ten of these patients were female, with an average age of 35 years, ranging from 16 to 49 years.

These national data align with our findings, showing a clear female predominance (80%) and a young age at the time of diagnosis.
Internationally, the largest series in the literature was conducted by the Mayo Clinic, published in 2012 by Zawadowski et al. [3], which included

24 cases with inclusion criteria of a confirmed diagnosis of SLE associated with clinical symptoms and abnormalities on echocardiography (ETT) or cardiac MRI, excluding coronary artery disease. The study showed a female predominance of 79%, with a mean age of 47.6 years.

Another Chinese study, conducted between 2001 and 2012 by Li Zhang et al. [4], published in 2015, and focused on 25 patients with lupus myocarditis. Twenty-two of the patients were female (88%), and the average age was 28.0 ± 12.28 years.

B. Positive Diagnosis

1. Clinical Manifestations:

Clinical signs of acute myocarditis are numerous and nonspecific. The diagnosis should be considered in the presence of recent signs of heart failure or supraventricular or ventricular arrhythmias in the absence of coronary or valvular disease. At the time of diagnosis, patients may present with chest pain, arthralgia, fever, or a general feeling of malaise. The signs of heart failure can be subtle or, conversely, quite prominent [5, 6].

2. Physical Examination:

The clinical examination may be normal, and the patient's hemodynamic status is usually stable, though signs of instability and shock can be present. Heart failure signs may also be found, including tachycardia, a gallop rhythm, and crackles. Signs of right heart failure may include jugular venous distension and peripheral edema. Furthermore, patients with ventricular dilation may have a mitral regurgitation murmur, typically described as a holosystolic apical murmur. A pericardial friction rub may also be appreciated, especially in cases of concomitant pericarditis [5, 6].

3. Electrocardiogram (ECG):

The ECG can show arrhythmias at all levels, intraventricular or atrioventricular conduction disturbances. Sometimes, systematic repolarization disturbances are observed, which are compatible with a myocardial infarction diagnosis and can lead to unnecessary urgent revascularization discussions [5, 6].

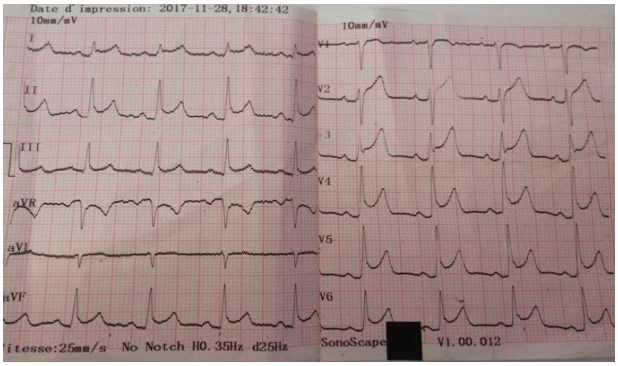

Figure 5: ECG shows upward concave ST-segment elevation

4. Biological Assessment

The serum levels of cardiac enzymes, particularly Troponin Ic, are frequently elevated and are proportional to the severity of the disease [7, 8]. Occasionally, leukocytosis (or leukopenia), elevated serum C-reactive protein (CRP), and an increased erythrocyte sedimentation rate (ESR) may be observed.

5. Role of Echocardiography

Conventional transthoracic echocardiography (TTE) is often the first imaging test. It can reveal left ventricular dilation to varying degrees, alterations in segmental kinetics, such as hypokinesis or akinesis, although it's diagnostic utility remains limited in cases of preserved left ventricular ejection fraction. Additionally, echocardiography can show an associated pericardial effusion and provides an estimation of systolic pulmonary pressure and diastolic function [9].The analysis of myocardial deformation (strain) through automated pixel tracking (speckle tracking) offers a more detailed exploration of myocardial systolic function [10, 11]. Two-dimensional strain (2D strain), using adjacent myocardium as a reference point, allows for angle-independent quantification along three orthogonal axes (circumferential, radial, and longitudinal). Several studies [12, 13] have shown that global and regional myocardial deformation is decreased in myocarditis. Strain longitudinal grading (SLG) is effective in identifying myocardial abnormalities in young populations with suspected myocarditis and preserved left ventricular ejection fraction.

6. Cardiac MRI

a) Indications for Cardiac MRI

b)

Recent or Persistent	Recent Signs of Myocardial Injury	Suspected Viral Etiology

Suggestive Symptoms		
Dyspnea	Ventricular dysfunction on imaging	Recent history of viral infection or recent myocarditis
Or orthopnea	Or recent or persistent ECG repolarization abnormalities	Or absence of vascular risk factors
Or palpitations	Or Troponin elevation	Or age under 35 years
Or exertional malaise		Or symptoms unexplained by documented coronary artery disease
Or chest pain		Or normal myocardial ischemia detection tests

Cardiac MRI should only be performed in symptomatic patients when there is sufficient clinical evidence of myocarditis and if the MRI findings are expected to optimize diagnostic and therapeutic management [14]. It is particularly indicated in patients with recent or persistent symptoms suggestive of myocardial injury, especially in cases of chest pain with elevated troponin levels and normal coronary arteries. Studies have shown that myocarditis is identified in over 30% of such patients [15].

Relative indications may extend to individuals suspected of having myocarditis who are engaged in intense physical activity, particularly athletes, or to patients with unexplained ECG abnormalities suggestive of myocarditis, even in the absence of symptoms [14].

b) MRI Diagnostic Criteria for Myocarditis ("Lake Louise Criteria")

Due to the lack of large-scale multicenter data, current recommendations reflect the best possible expert consensus. The **Lake Louise Criteria** combine three tissue markers, and in cases of clinically suspected myocarditis, MRI findings are considered indicative of myocardial inflammation if at least **two** of the following criteria are present:

- **Increased myocardial T2 signal (regional or global):**
  - Myocardial edema
  - Reversible myocardial injury
- **Early gadolinium enhancement (T1-weighted imaging):**
  - Marker of myocardial vasodilation
- **Late gadolinium enhancement (T1-weighted imaging):**
  - At least one focal lesion with a non-ischemic regional distribution
  - Irreversible myocardial injury

➢ **Additional supportive findings:**

- Left ventricular dysfunction
- Pericardial effusion

When all imaging sequences are performed, the presence of **two or all three** tissue criteria allows myocarditis to be diagnosed or excluded with a **diagnostic accuracy of 78%**. If only late gadolinium enhancement is observed, the diagnostic accuracy is reduced to **68%.**

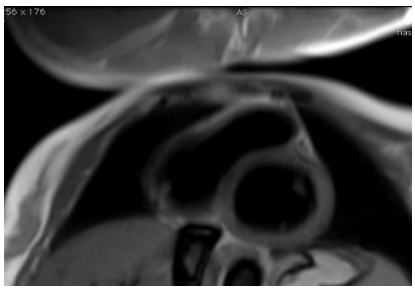

Figure 6: Short-axis T2-weighted sequence: No hyper intensity observed, indicating the absence of edema.

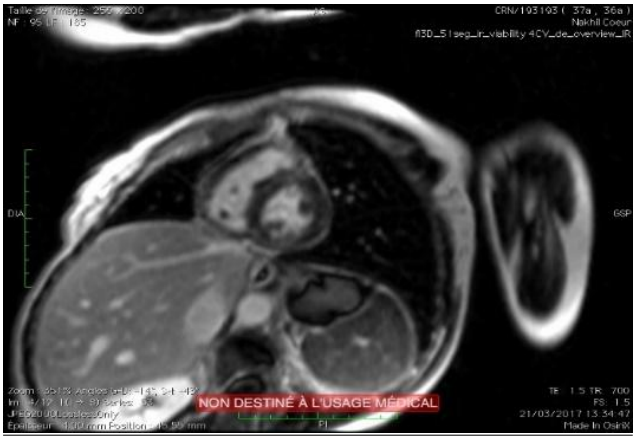

Figure 7: Short-axis view in late gadolinium enhancement sequence: reveals sub epicardial contrast uptake in the inferior wall and patchy intramyocardial nodular enhancement in the inferoseptal wall.

A follow-up MRI between one and two weeks is recommended when only one criterion is present or when all criteria are absent, especially if the clinical presentation is highly suggestive of myocarditis [14]. In our study, only one patient had a single positive Lake Louise criterion, which was mid-wall late gadolinium enhancement.

7. Myocardial Biopsy

A myocardial biopsy is recommended in cases of acute heart failure persisting for more than two weeks, the recent onset of arrhythmias or conduction disorders, and/or suspicion of a specific disease (connective tissue disease, immunoallergic reaction, sarcoidosis) [16].

Myocardial biopsy is particularly useful in the assessment of unexplained cardiomyopathy that cannot be diagnosed through conventional cardiac examinations. In a series of 845 patients, a definitive diagnosis was established in 75% of cases [17]. However, the sensitivity and specificity of the procedure are limited due to the often focal and subendocardial nature of the histological lesions [18]. Furthermore, the severity of histological involvement was not correlated with prognosis in one of the largest published series to date [19].

8. Diagnostic Criteria in the Literature

In the two Moroccan series by Haroun [1] and Oubelkacem [2], the diagnosis was established based on clinical criteria, particularly chest pain in the absence of coronary artery disease, biological markers such as elevated cardiac enzymes (especially troponin), and echocardiographic findings showing segmental wall motion abnormalities or left ventricular dysfunction. Notably, no patient in these studies underwent cardiac MRI or myocardial biopsy to confirm the diagnosis.

Zawadowski [3] identified the diagnosis based on echocardiography, which demonstrated left ventricular dysfunction and segmental wall motion abnormalities. Cardiac MRI was performed in only six patients, with late gadolinium enhancement observed in just two cases. Myocardial biopsy was performed postmortem in a single patient who died of cardiogenic shock, revealing histological findings consistent with myocarditis.

In the Chinese study by Li Zhang [4], the diagnosis was based solely on echocardiographic criteria, with no patient undergoing cardiac MRI.
Beyond these series, a few case reports in the literature describe lupus myocarditis, with or without complications [20-21].

C. Treatment

Myocarditis is a rare but potentially fatal complication of systemic lupus erythematosus (SLE). Few controlled trials have been conducted, and most reports are based on isolated case studies. High-dose corticosteroid therapy remains the most commonly used treatment for lupus myocarditis [4, 22]. It appears to be an effective approach, leading to improved left ventricular function in hemodynamically stable patients. However, cases of lupus myocarditis presenting with cardiogenic shock requiring mechanical support are rare.

Immunosuppressive agents such as azathioprine, cyclophosphamide, and Mizoribine have shown benefits in both acute and chronic lupus myocarditis, with significant improvement in systolic function observed within six months of treatment initiation [23, 24]. Biologic therapies, including Rituximab and Belimumab, may also be beneficial in SLE management [25, 26]. Belimumab was approved by the U.S. Food and Drug Administration (FDA) in 2011 for SLE patients receiving optimal standard therapy, including non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, synthetic anti malarials, and immunosuppressants.

Studies have demonstrated that this biologic therapy reduces disease severity and the recurrence of severe disease flares [27, 28].

However, these agents have not been specifically tested in lupus myocarditis, and no cases in the literature support their role in its treatment. Some anecdotal cases have reported the use of intravenous immunoglobulins (IVIG) and plasmapheresis [29, 30].

Although treatment options for lupus myocarditis remain limited, biologic agents have shown promising results and may be valuable for managing refractory cases, particularly those unresponsive to high-dose corticosteroids.

D. Cardiac MRI Follow-up

The decision regarding the follow-up of myocarditis patients is not well standardized and is made on a case-by-case basis. Myocardial assessment in the early stages of myocarditis may be less sensitive compared to evaluations conducted seven days after symptom onset [31]. This may be due to the focal nature of inflammatory lesions in the early stages of the disease.

Thus, in cases where there is strong clinical suspicion of myocarditis but initial cardiac MRI findings are negative, repeat imaging is justified to establish the diagnosis.

Follow-up imaging at least four weeks after disease onset may be beneficial, as tissue inflammation should not persist beyond 2 to 3 weeks. Persistent inflammatory markers on MRI four weeks after symptom onset are considered a relevant prognostic factor [18].

E. Prognosis and Evolution

Myocarditis in SLE patients is a rare but potentially life-threatening complication.

The relative rarity of this condition limits the feasibility of controlled trials to determine optimal management and treatment strategies. Current clinical decisions are largely based on anecdotal case reports. Additionally, long-term outcomes in patients who initially respond to treatment remain poorly understood.

Life-threatening complications include ventricular arrhythmias leading to cardiac arrest or sudden death [20]. Other significant complications involve the rapid onset of heart failure due to severe left ventricular dysfunction, as highlighted in multiple case reports. This condition can further deteriorate into cardiogenic shock [32, 33, 34].

CONCLUSION

Lupus myocarditis, unlike viral myocarditis, is characterized by pancarditis with pericardial and valvular involvement. It frequently affects ventricular function and can be a potentially life-threatening complication.

Diagnosis is primarily based on clinical suspicion, after excluding other etiologies, particularly coronary artery disease. Echocardiographic findings confirming myocardial involvement include left ventricular systolic dysfunction, segmental wall motion abnormalities, and alterations in global longitudinal strain.

The most specific and relevant diagnostic tool remains histology; however, myocardial biopsy is not commonly performed in routine practice. Instead, cardiac MRI has emerged as the most effective imaging modality for myocarditis diagnosis, with well-established criteria.

The prognosis can be poor in the presence of ventricular arrhythmias or severe heart failure. Furthermore, an optimal treatment strategy for lupus myocarditis has yet to be clearly defined.

BIBLIOGRAPHIE

1- Harouna H., Aboudib F., Bouissar W., Echchilali K., Moudatir M., Alaoui F.-Z., El Kabli H. Manifestations cardiaques au cours dulupus érythémateux systémique.La Revue de médecine interne 37 (2016): A192.

2- Oubelkacem N., Khammar Z., Atassi M. , Berrady R., Rabhi S., Bono W.L'atteinte hématologique au cours dulupus érythémateux systémique. La Revue de médecine interne 36S (2015) : A091.

3- Zawadowski GM., Klarich KW., Moder KG. et al.A contemporary case series of lupus myocarditis. Lupus (2012) 21, 1378–1384.

4- Zhang L, Zhu YL, Li MT, Gao N, You X, Wu QJ, Su JM, Shen M, Zhao LD, Liu JJ, Zhang FC, Zhao Y, Zeng XF. Lupus Myocarditis: A Case–Control Study from China. Chin Med J 2015; 128:2588-94

5- Sarda L, Colin P, Boccara F, Daou D, Lebtahi R, Faraggi M, Nguyen C, Cohen A, Slama M.S, Steg P.G, Le Guludec D. Myocarditis in patients with clinical presentation of myocardial infarction and normal coronary angiograms. J Am Coll Cardiol 37(2001): 786-792.

6- Narula J., Khaw B.A., Dec G.W., Jr., Palacios I.F., Southern J.F., Fallon J.T., Strauss H.W., Haber E., Yasuda T. Brief report: recognition of acute myocarditis masquerading as acute myocardial infarction. N Engl J Med 328 (1993).: 100-104.

7- Smith S.C., Ladenson J.H., Mason J.W., Jaffe A.S. (1997). Elevations of cardiac troponin I associated with myocarditis. Experimental and clinical correlates. Circulation 95 : 163-168.

8- Mirabel M, Luyt C.E, Leprince P, Trouillet J.L, Leger P, Pavie A, Chastre J, Combes A. Outcomes, Long-term Quality-of-Life and Psychological Assessment of Fulminant Myocarditis(2011) ; Crit Care Med.39(5):1029-35.

9- Fourcade. C. Analyse de la déformation myocardique longitudinale dans le diagnostic de myocardite aiguë à fraction d'éjection ventriculaire gauche normale. 21 Avril 2017.code thèse : TOU 31540-Page 19.

10- Ha SJ, Woo JS, Kwon SH, Oh CH, Kim KS, Bae JH, Kim WS. Acute regional myocarditis with normal ventricular wall motion diagnosed by two-dimensional speckle tracking imaging. Korean J Intern Med. 2013 Nov;28(6):732-5.

11- Brown J, Jenkins C, Marwick TH. Use of myocardial strain to assess global left ventricular function: a comparison with cardiac magnetic resonance and 3-dimensional echocardiography. Am Heart J. 2009 Jan;157(1):102–105.

12- Uppu SC, Shah A, Weigand J, Nielsen JC, Ko HH, Parness IA, Srivastava S. Two-dimensional speckle-tracking-derived segmental peak systolic longitudinal strain identifies regional myocardial involvement in patients with myocarditis and normal global left ventricular systolic function. Pediatr Cardiol. 2015 Jun; 36(5):950-9.

13- Di Bella G, Gaeta M, Pingitore A, Oreto G, Zito C, Minutoli F, Anfuso C, Dattilo G, Lamari A, Coglitore S, Carerj S. Myocardial deformation in acute myocarditis with normal left ventricular wall motion A cardiac magnetic resonance. Circ J. 2010 Jun;74(6):1205-13.

14- Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, et al. Cardiovascular Magnetic Resonance in Myocarditis: A JACC White Paper. Journal of the American College of Cardiology. 2009 Apr;53(17):1475– 87.

15- Assomull RG, Lyne JC, Keenan N, et al. The role of cardiovascular magnetic resonance in patients presenting with chest pain, raised troponin, and unobstructed coronary arteries. Eur Heart J 2007; 28:1242-9.

16- Cooper LT, Baughman KL, Feldman AM, Frustaci A, Jessup M, Kuhl U, et al. The Role of Endomyocardial Biopsy in the Management of Cardiovascular Disease: A Scientific Statement From the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. Circulation. 2007 Nov 6; 116(19):2216–33.

17- Ardehali H, Qasim A, Cappola T, Howard D, Hruban R, Hare J.M, Baughman K.L, Kasper E.K. Endomyocardial biopsy plays a role in diagnosing patients with unexplained cardiomyopathy. Am Heart J (2004); 147: 919-923.

18- Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation 2004 ; 109 :1250–8.

19- McCarthy R.E., 3rd, Boehmer J.P., Hruban R.H., Hutchins G.M., Kasper E.K., HareJ.M., Baughman K.L. Long-term outcome of fulminant myocarditis as compared with acute (nonfulminant) myocarditis. N Engl J Med 342 (2000): 690-695.

20- -Picque J.B., Stamboul K., Bielefeld P., Turcu A., Muller G., H. Devilliers, Besancenot J.F. Arrêt cardiaque récupéré révélant unemyocardite lupique La Revue de médecine interne (2015) 36S A76–A185.

21- Ben Abdellah et al. Les manifestations cardiaques en cours du lupus érythémateux systémique. La Revue de médecine interne (2009) ; 30 : page 90.

22- Barnado A, Kamen DL. Myocarditis successfully treated with intravenous immunoglobulin in a patient with systemic lupus erythematous and myositis. Am J Med Sci 2014; 347: 256-7.

23- Naarendorp M, Kerr LD, Khan AS, et al. Dramatic improvement of left ventricular function after cytotoxic therapy in lupus patients with acute cardiomyopathy: report of 6 cases. J Rheumatol. 1999; 26:2257–2260.

24- Akazawa S, Ichinose K, Origuchi T, et al. Successful treatment of chronic lupus myocarditis with prednisolone and mizoribine. Mod Rheumatol. 2010; 20:606–610.

25- Merrill JT, Neuwelt CM, Wallace DJ, et al. Efficacy and safety of rituximab in moderately-to-severely active systemic lupus erythematosus: the randomized, doubleblind, phase II/III systemic lupus erythematosus evaluation of rituximab trial. Arthritis Rheum. 2010; 62:222–233.

26- Wallace DJ, Stohl W, Furie RA, et al. A phase II, randomized, double-blind, placebo-controlled, dose-ranging study of belimumab in patients with active systemic lupus erythematosus. Arthritis Rheum. 2009 ;61 :1168–1178.

27- Furie R, Petri M, Zamani O, et al. A phase III, randomized, placebo-controlled study of belimumab, a monoclonal antibody that inhibits B lymphocyte stimulator, in patients with systemic lupus erythematosus. Arthritis Rheum. 2011 ;63 :3918–3930.

28- Navarra SV, Guzmán RM, Gallacher AE, et al. Efficacy and safety of belimumab in patients with active systemic lupus erythematosus: a randomised, placebo-controlled, phase 3 trial. Lancet. 2011; 26:721–73

29- Sherer Y, Levy Y, Shoenfeld Y. Marked improvement of severe cardiac dysfunction after one course of intravenous immunoglobulin in a patient with systemic lupus erythematosus. Clin Rheumatol. 1999 ;18:238–240.

30- Tamburino C, Fiore CE, Foti R, et al. Endomyocardial biopsy in diagnosis and management of cardiovascular manifestations of systemic lupus erythematosus (SLE). Clin Rheumatol. 1989; 8:108–112

31- Friedrich MG, Strohm O, Schulz-Menger J, Marciniak H, Luft FC, Dietz R. Noninvasive diagnosis of acute myocarditis by contrastenhanced magnetic resonance imaging—response to the author. Circulation 1999; 99:459-460.

32- Appenzeller S, Pineau CA and Clarke AE. Acute lupus myocarditis: Clinical features and outcome.Lupus Aug;20(9) 2011: 981-8

33- Gurveen M, Serafin C, Vamsi K et al.Rare Presentation of Lupus Myocarditis With Acute Heart Failure-A Case Report. American Journal of Therapeutics (2015) : 1-4.

34- Gottenberg JE, Roux S, Assayag P, Clerc D, Mariette X; Specific cadiomyopathy in lupus patients: report of 3 cases. Revue du Rhumatisme (2004); 71: 78–81.

Acknowledgments:

Not applicable.

Author contribution:

MB: Study concept, Data collection, Data analysis, writing the paper.

RL: Study concept, Data collection, Data analysis.

RF: Study concept, Data analysis, writing the paper.

NM: Supervision and data validation

IA: Supervision and data validation

AB: Supervision and data validation

All authors reviewed the final manuscript.

Funding Sources:

There are no funding sources to declare.

Availability of supporting data

Not applicable.

Ethics approval and consent to participate:

Not applicable.

Consent for publication:

Written informed consent was obtained from the patients for publication of this cases report.

Competing interests:

None.

Provenance and peer review

Not commissioned, externally peer-reviewed

References :

[1] Monpère C, Sellier Ph, and Meurin Ph et al.

Recommandations de la Société française de cardiologie concernant la pratique de la réadaptation cardiovasculaire chez l'adulte version 2. Archive des maladies du coeur et

des vaisseaux, 2002 ; 95: 963-997.

[2] Pavy B, IIIou MC, vergès-PatoIs B et al.

French Society of Cardiology guidelines for cardiac rehabilitation in adults.ArchCardiovas Dis, 2012;105:3

[3] Lesniak, « Performance Measures for Short-Term Cardiac Rehabilitation in Patients of Working Age: Results of the Prospective Observational Multicenter Registry OutCaRe »

[4] Rosa, « Cardiac rehabilitation after acute coronary syndrome: Do all patients derive

the same benet? »

[5] Popovic, « Improvements in Key Cardiopulmonary Exercise Testing Variables Following Cardiac Rehabilitation in Patients With Coronary Artery Disease ».

[6] Adghar D. ,Bougherbal R , Hanifi R et al.

La réadaptation cardiaque du coronarien : première expérience en Algérie- Annales de Cardiologie et d'Angéiologie 2008 ; 57 : 44–47

[7] Gotzmann M, et al.

One-year results of transcatheter aortic valve implantation in severe symptomatic aortic valve stenosis. Am J Cardio 2011;107(11):1687–9

[8] Tariq MI, Khan AA, Khalid Z, Farheen H, Siddiqi FA, Amjad I.

Effect of Early ≤ 3 Mets (Metabolic Equivalent of Tasks) of Physical Activity on Patient's Outcome after Cardiac Surgery. J Coll Physicians Surg Pak. 2017 Aug;27(8):490-494.

[9] Baudin B, Cohen A.

Données épidémiologiques des maladies cardio-vasculaires et prise en charge des accidents cardio-vasculaires. Revfrdes labo 2009 ; 409 : 27 – 39.

[10] Halm M, Penque S, Doll N, Beahrs M.

Women and cardiac rehabilitation: referral and compliance patterns. J Cardiovasc Nurs. 1999 Apr;13(3):83-92.

[11] Baumgatner H., Falk V., Bax J.J., et al.

2017 ESC/EACTS Guidlines for the managment of valvular heart disease. Eur Heart J. 2017;38 :2739-2791.

[12] Fiorina C, Vizzardi E, Lorusso R, Maggio M, De Cicco G, Nodari S et al.

The 6-min walking test early after cardiac surgery. Reference values and the effects of rehabilitation programme. European Journal of Cardio-Thoracic Surgery. 2007;32(5):724-729.

[13] Gaalema DE, Cutler AY, Higgins ST, Ades PA

Smoking and cardiac rehabilitation participation: associations with referral, attendance and adherence. Prev Med. 2015;80:67–74.

[14] Samadoulougou A, Millogo G, Yameogo NV et al.

Aspects épidémiologiques, cliniques et évolutifs des cardiopathies ischémiques dans le service de cardiologie de CHU de Yalgado OUEDRAGO. MedAf Noire 2011 ;5801 : 14-8.)

[15] Le Breton PH.

Prise en charge de l'infarctus du myocarde : les délais. Presse Med 2011 ; 40 :600-5.

[16] Vanhees L, Geladas N, Hansen D, Kouidi E, Niebauer J, Reiner Ž et al.

Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular risk factors: recommendations from the EACPR (Part II). European Journal of Preventive Cardiology. 2011;19(5):1005-1033.

[17] Marcassa C, Giordano A, Corrà U, Giannuzzi P.

Greater functional improvement in patients with diabetes after rehabilitation following cardiac surgery. Diabet Med. 2016 Aug;33(8):1067-75.

[18] Vanhees L, Stevens A, Schepers D, et al.

Determinants of the effects of physical training and of the complications requiring resuscitation during exercise in patients with cardiovascular disease. Eur J Cardiovasc Prev Rehabil 2004; 11: 304– 312.

[19] Bellmann, B., Lin, T., Greissinger, K. et al.

The Beneficial Effects of Cardiac Rehabilitation. Cardiol Ther 9, 35–44 (2020). 129

[20] Belardinelli R., Lacalaprice F., Faccenda E., and al.

Effects of short-term moderate exercise training on sexual function in male patients with chronic stable heart failure Int J Cardiol 2005 ; 101 : 83-90 [cross-ref]

[21] ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories.

Atsstatement: guidelines for the six-minute walk test 2002. Am J Respir Crit Care Med 2002; 166: 111–117.

[22] Butchart EG, Gohlke-Bärwolf C, Antunes MJ, Tornos P, De Caterina R, Cormier B, Prendergast B, Iung B, Bjornstad H, Leport C, Hall RJ, Vahanian A;

Working Groups on Valvular Heart Disease, Thrombosis, and Cardiac Rehabilitation and Exercise Physiology, European Society of Cardiology. Recommendations for the management of patients after heart valve surgery. Eur Heart J. 2005 Nov;26(22):2463-71.

[23] Shanmugasegaram S., Gagliese L., Oh P., et al.

Psychometric validation of the cardiac rehabilitation barriers scale. Clin Rehabil. 2012;26 :152-164

[24] Zhang L, Sobolev M, Piña IL, Prince DZ, Taub CC.

Predictors of Cardiac Rehabilitation Initiation and Adherence in a Multiracial Urban Population. J Cardiopulm Rehabil Prev. 2017 Jan;37(1):30-38