

Evolutionary Aspects of Electrocardiographic and Echocardiographic Manifestations in Systemic Lupus Erythematosus: Descriptive Longitudinal Study about 25 Cases in 18-Month Monitoring

Ngaïdé AA^{1*}, Mbaye A¹, Ad Kane², Thiam Al¹, Lèye M³, Dioum M³, Sarr SA², FAW², Ka MM¹, Ndiaye M⁴, Gaye ND¹, Babaka K¹, Ndiaye M¹, Ndao CT², Cissé AF¹, Kouamé I¹, Thiombiano LP², Bah MB², Bodian M², Ndiaye MB², Diao M², Sarr M² and Kane A¹, Bâ SA²

¹Cardiology Clinic of General Hospital Grand Yoff Dakar, Senegal

²Cardiology Clinic of Hospital Aristide Le Dantec Dakar, Senegal

³Cardiology Clinic of National Hospital Dakar, Senegal

⁴Dermatology service of Hospital Aristide Le Dantec Dakar, Senegal

*Corresponding author: Ngaïdé AA, Cardiology Clinic of General Hospital Grand Yoff Dakar, Senegal, Tel: 00221775549233; E-mail: ngaideaa@hotmail.fr

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Abstract

Introduction: Systemic lupus erythematosus is a major systemic autoimmune disease of unknown cause. It can affect all organs and especially the heart with different proportions. The aim of this study was to determine the evolving electrocardiographic and echocardiographic features in systemic lupus erythematosus.

Methodology: This study was carried out at the cardiology and dermatology Aristide Le Dantec Hospital. This is a descriptive longitudinal study conducted from January 2013 to October 2014. We included SLE patients who had at least two echocardiograms and electrocardiograms performed at least 3 months apart. Anamnestic information, electrocardiographic and echocardiographic features were reported on a pre-established survey form. Data were entered using Epi-info software version 3.5.1.

Results: We included 25 patients. It was mentioned a female predominance (88%). The average age of patients was 35.5 years. All patients had dermatological signs of systemic lupus erythematosus. Cardiac events were found on clinical examination in 20% of cases. Sixty four percent (64%) of patients had an abnormal electrocardiogram dominated by left ventricular hypertrophy (24%). During 18 months of follow-up, we noted some electrocardiographic changes in 16% of cases. Ultrasound abnormalities were found in 40% of patients: 8% presented a slight expansion of the OG, a patient had impaired left ventricular function with the presence of a spontaneous contrast intra VG and increased filling pressures. Right ventricular function was impaired in one patient. Other abnormalities were found: 12% of aneurysm of the interatrial septum, 4% of atrial septal defect and 4% of pericardial detachment.

Conclusion: Cardiovascular violations are fairly common in SLE patients. A study on a large sample and long-term could better assess cardiovascular complications in SLE patients and how they occurred.

Keywords: Lupus; Cardiovascular disease; Electrocardiogram; Cardiac doppler ultrasound

Introduction

Systemic lupus erythematosus (SLE) is a major systemic autoimmune disease the reason of which is unknown [1]. Almost every organ can be affected. Cardiovascular complications are variable and are found in different extent depending on the type of damage: myocardial, pericardial or endocardial. Early mortality is still linked to the lupus activity, but the most significant late mortality is of cardiovascular origin [2]. It also affects women with predilection in ovulatory activity period (sex ratio 9 women to 1 man) [3]. It is characterized biologically by the production of antinuclear antibodies directed specifically against native DNA [4].

SLE affects three layers of the heart with the pericardium as favourite field. The diagnostic difficulties to assert the myocardial or endocardial damage explain the inconsistencies in the literature [5,6]. Cardiovascular damage are under-diagnosed because they are most

often asymptomatic [7]. Systemic lupus erythematosus represents an independent risk factor for a first myocardial infarction [8,9]. Visceral involvement, including cardiovascular damage, determine prognosis, hence the need for early diagnosis [10,11]. Given the cardiovascular complications thereof, monitoring of SLE patients on electrocardiographic and echocardiographic plan could be of great diagnostic value.

In the United States of America, the prevalence of systemic lupus is three to four times higher among African Americans than Caucasians [12]. In sub-Saharan Africa, systemic lupus is not uncommon [13]. However, the data reported above relate Senegal and South Africa. It's about Rather than hospital data, so definitely underestimated. In Dakar, the internal medicine department of the University Hospital Aristide Le Dantec, 142 cases of systemic lupus erythematosus have been collected over a period of 10 years [14]. In 2010, the Dermatology Service of the Institute of Social Hygiene Dakar, 33 new cases of lupus were collected of which 18 cases of systemic lupus erythematosus total number of patients about 17000 [14].

Studies on the evolutionary aspect of heart disease in SLE are rare and the interest could be to early diagnose cardiac disease in this condition. The study aimed at determining the evolutionary electrocardiographic and echocardiographic aspects in systemic lupus erythematosus in people with lupus.

Methodology

Data were entered into Epi Info software (version 3.5.1) and using the SPSS 16.0 software. A descriptive study was conducted; the quantitative variables of non-Gaussian distribution are described in median qualitative variables in count, percentage and frequency. An analytical study was also carried out: a comparison of percentages and proportions was conducted using the Chideux test or the Yates correction test when the numbers were lower than 5 with a significance of $p < 0.05$.

Mode TM	Mode 2D	Mode Doppler
NORMES		
Ao: 22-37 mm OAO: 16-25 mm OG: 18-40 mm VDd: 9-26 mm VG: SIVd: 6-11 mm DTD: 38-56 mm PPD: 6-11 mm DTS: 34 ± 7,1 mm FR: 28-44 % FE ≥ 55 %. IMVG: <110 g/m ² (femme)<134 g/m ² (homme)	AnAo: 18-26 mm sous-ao: 17-23 mm An.Pulm:10-22 mm TAP: 12-23 mm VD: STS: 5-20 cm ² STD: 11-35 cm ² VG: STS: 8-32 cm ² STD: 18-47 cm ² VTS: 18-32 ml/m ² VTD: 50-90 ml/m ² FE: >55% OG: Surface: <20 cm ² Volume: <29 ml/m ² OD: Surface <17 cm ²	<p style="text-align: center;">Mitrale</p> Em: 60-130 cm/s Am: 30-73 cm/s ITVm: 15,6 ± 2,5 cm TdEm: 150-220 ms dAm: 133 ± 15 ms TRIV: 60-90 ms MAPSE: >14 mm Ea: 14,4 ± 2,7 cm/s Aa: 11,8 ± 1,3 cm/s Sa: 9,4 ± 1,4 cm/s <p style="text-align: center;">Tricuspide</p> Et: 34-68 cm/s At: 19-35 cm/s ITVt: 12,6 ± 1,9 cm TRIV: 60-80 ms TAPSE: >15 mm <p style="text-align: center;">Aortique</p> V max: 100-170 cm/s ITV ao: 18,7 ± 3,1 cm <p style="text-align: center;">Pulmonaire</p> V max: 60-90 cm/s ITVp: 16,1 ± 2,7 cm

Table 1: Normal echocardiographic values as recommended by the American Society of echocardiography

Results

25 patients were involved. We noted female predominance (88%) with a 0.14 female/male sex ratio. The average age of patients was 35.5 years with extremes of 19 and 58 years. The mean disease length was 3.2 years. All patients had dermatological manifestations as a result of SLE. Sixteen (16%) of the patients had a history of pericarditis and 12% a thromboembolism (pulmonary embolism 8% and vein thrombosis 4%). The most represented cardiovascular risk factors (Figure 1) were

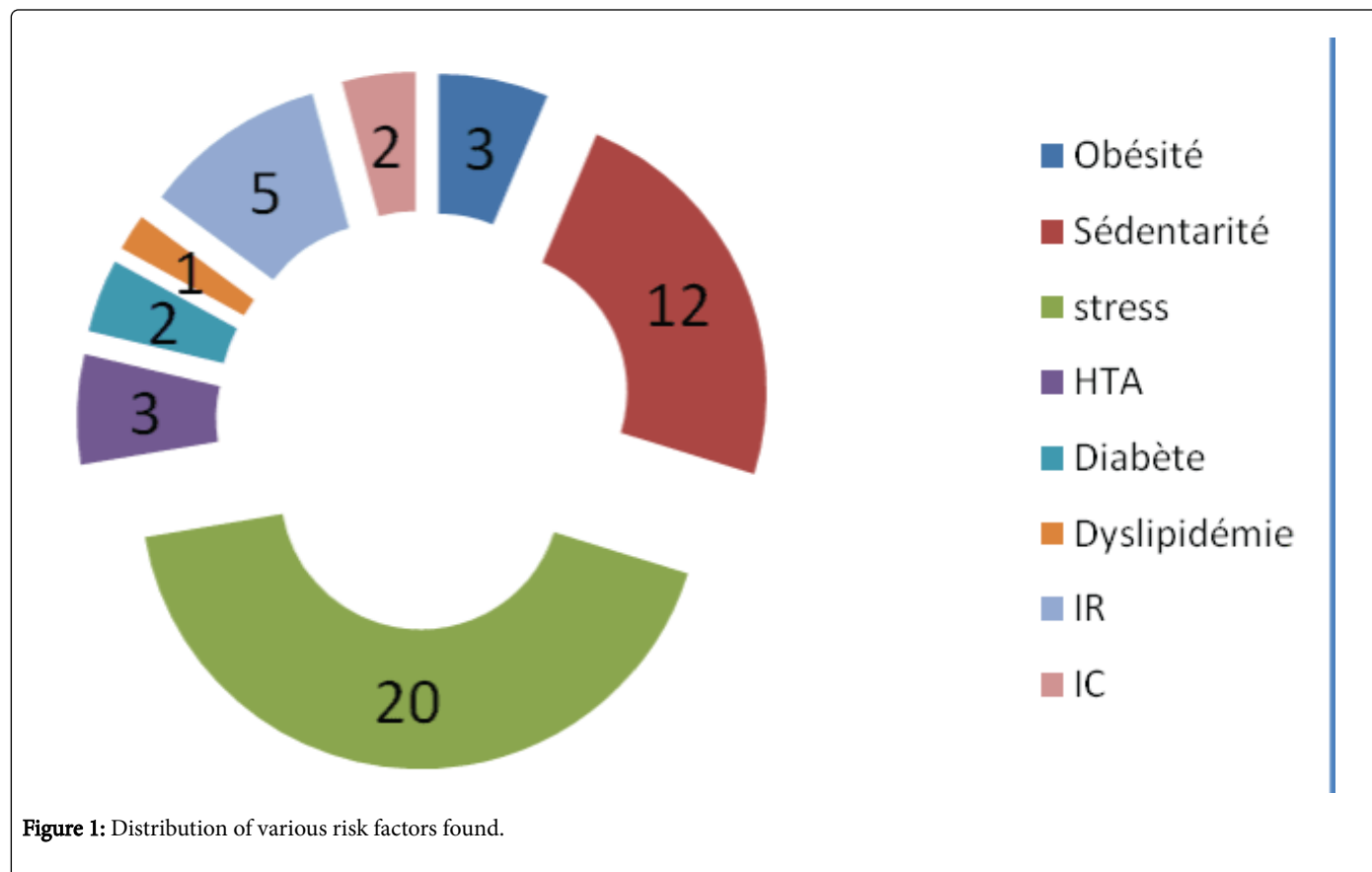
stress (43%), physical inactivity (23%), renal impairment (11%), high blood pressure, obesity (6%) and diabetes (4%). Cardiac events were found on clinical examination in 20% of cases, dominated by dyspnea.

Sixty four percent (64%) of patients had an abnormal electrocardiogram (Figure 2), and was dominated mainly by left ventricular hypertrophy (24%), abnormal repolarization (16%), 8% of primary disorders and 8% secondary to subepicardial ischemia and stage III Holzmans (Figure 3). Moreover, a Q3T3 pattern was found in

16% of the population. In 18 months follow-up, 16% found their electrocardiogram changed. Thus, a PQ sub-segment became isoelectric once again. A first-degree atrioventricular block had disappeared during the following checks. We also noted the appearance of negative T waves diffusely on a path that was normal three months earlier, this without particular clinical setting.

Doppler echocardiography facilitated the finding of anomalies (Figure 4) in 40% of patients. Pericardial detachment observed in 4% (Figure 5) of the patients disappeared during the various controls. Eight percent (8%) of patients had a slight expansion of the OG, a

patient had impaired left ventricular function (30% LVEF using the biplane Simpson's method) with the presence of intra LV spontaneous contrast and increased filling pressures. This myocardial damage was evidenced in the acute phase of SLE and had improved under treatment after three months follow-up without going back to normal. Right ventricular function was impaired in a patient (TAPSE: 11mm, Tricuspid (SA): 6 cm / s). Pulmonary artery systolic pressure (PASP) of 38 mmHg was found in a patient after 6-month monitoring without apparent cause.



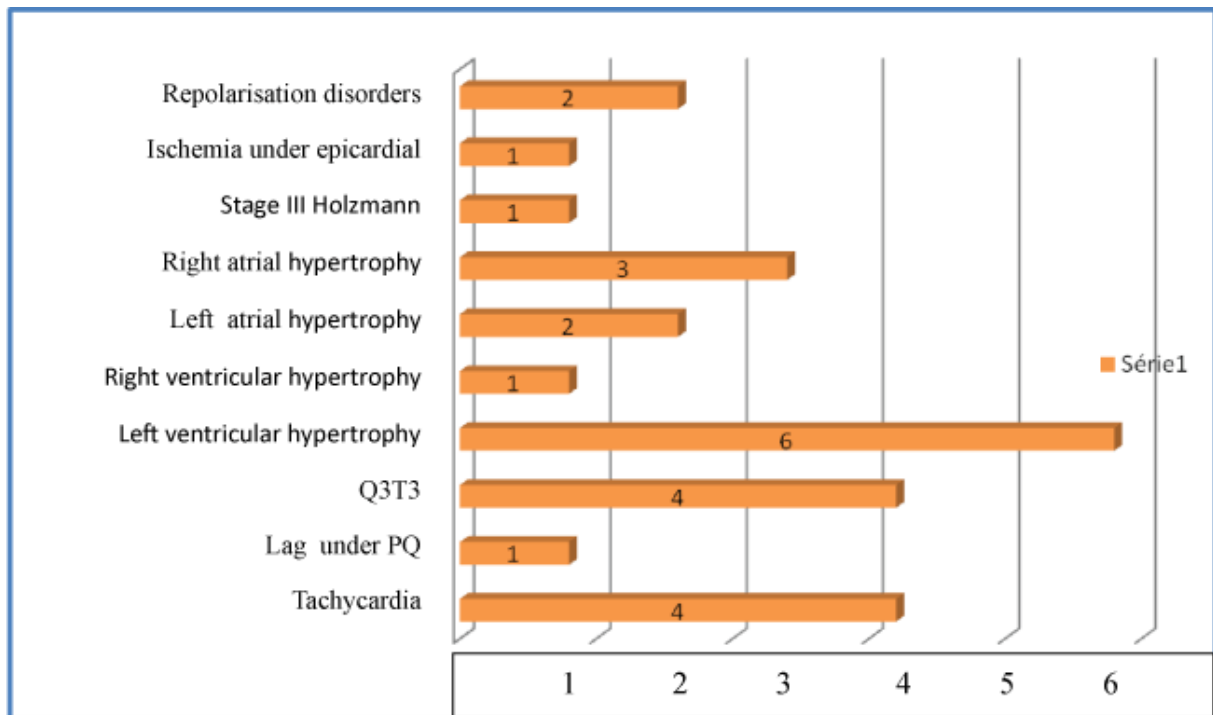


Figure 2: Electrocardiographic abnormalities.

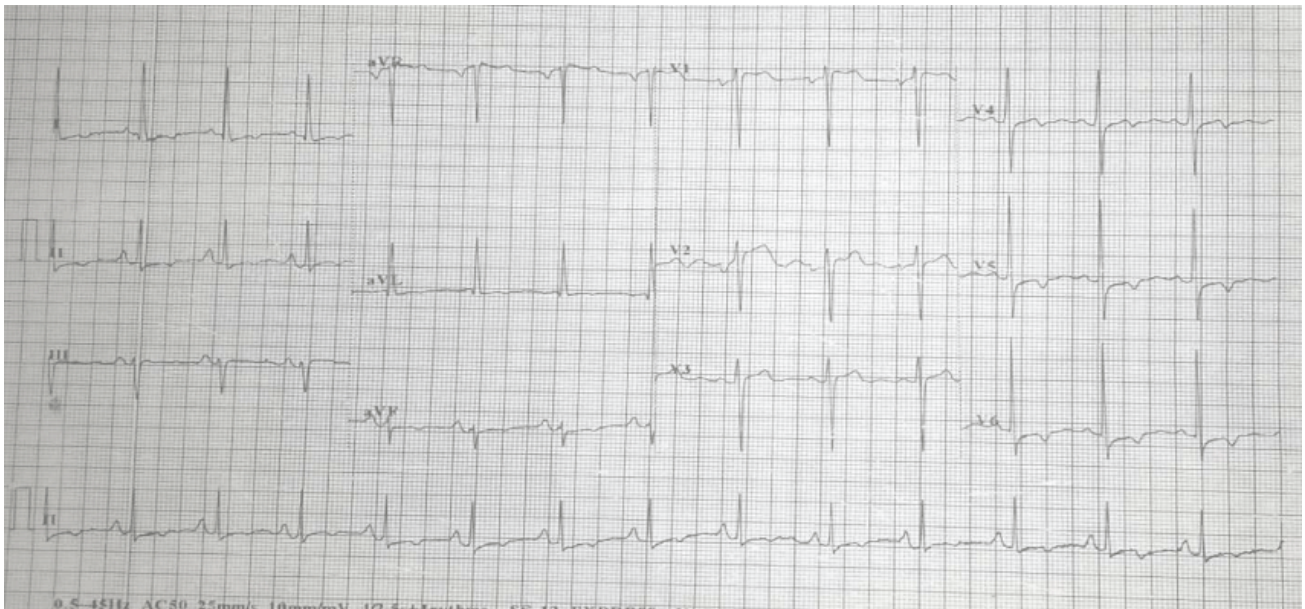


Figure 3: Electrocardiogram 12-lead coupled with a long DII, find a systolic left ventricular hypertrophy, left atrial hypertrophy and right atrial hypertrophy.

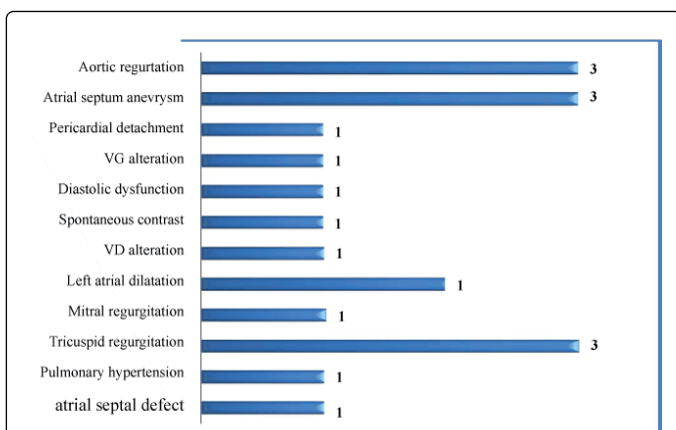


Figure 4: Echocardiographic abnormalities.

No patient presented Libman-Sacks endocarditis and valvular abnormality at disease onset or during follow-up. However minor leaks were noted (aortic regurgitation 12%, tricuspid insufficiency 12%, and mitral regurgitation 4%). Other abnormalities were encountered, such as atrial septal aneurysm (12%) and atrial septal defect (4%).

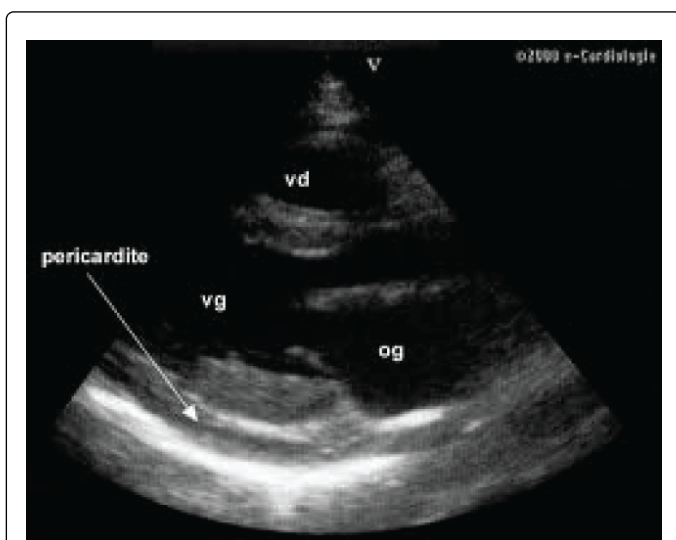


Figure 5: Echocardiography showing pericardial effusion abundance of small longitudinal section major axis.

Discussion

The study involved a small single-center sample. This had led to the systematic selection of mucocutaneous lupus and prevented from getting a more substantial sample. A sizeable population would have helped detect other abnormalities. The follow-up was relatively short (18 months) and patients were almost all under treatment. The average age of the patients (35.5 years) was similar to that observed in the study by Tazi (31 years) [15]. This is in compliance with the fact that SLE is a young woman's disease.

Vasculitis can affect the vessels of almost every caliber. Venous or repeated arterial thrombosis are observed in 20-30% of lupus [16]. The

usual cardiovascular risk factors widely prevalent in lupus population as reflected by this international multicenter study that showed these patients up to 58% of high blood pressure, high cholesterol 60%, 5% of diabetes [17]. These rates are higher than those found in our study except for diabetes. The very small sample could be the reason. Two factors may play a role in the development of atherosclerosis in SLE: inflammation and the use of corticosteroids [18].

The prevalence of electrocardiographic and echocardiographic abnormalities in our study (64%) is much higher than that described in the literature review (1.4%) [1]. This high rate could be explained by the relatively long follow-up. Our prevalence of ventricular hypertrophy is different from those found in the studies by Klinkhoff [19] and Sturfelt [4], where it ranged from 0 to 2%. Regular sinus tachycardia found in 16% of cases is different from the rate found by Hejtmancik [20] which is 50% of cases. Regarding disorders of repolarization, we found a prevalence of 21%, which was very close to that of Klinkhoff (19%) [19]. Evolutionarily speaking, during the 18-month monitoring, there was a change in electrocardiogram in 16% of the patients. We had noted the occurrence of diffusely negative T waves on normal departure route, which reverted to positive during inspections.

Pericarditis is a common manifestation of SLE which can reveal the disease in 10-40% of cases according to the groups studied [21]. In the literature, it is reported that pericarditis occurs at a rate of 11 to 54% [11]. These figures are higher than those found in our study sample (4%). In our study, the prevalence of cardiomyopathy (4%) is lower than the findings in the different studies by Apte [22] and Wijetunga [9] which are 8 to 25%. It is clear that accurate diagnosis becomes significant particularly in massive clinical settings especially in cases of major damage of cardiac function [5]. Let us stress on the difficulty in affirming the direct responsibility of lupus in heart failure [23]. In this study, no patient presented Libman-Sacks endocarditis. This could be due to the fact that valvular damage are observed in asymptomatic patients [24]. The frequency of Libman-Sacks endocarditis is varied depending on the study. Khamashta [25] reported Libman-Sacks vegetations in 7% of cases, Cervera: 27% [26], Nihoyannopoulos: 20% [24], and Sturfelt: 19% [6].

The natural course of pulmonary arterial hypertension (PAH) associated with SLE appears to be variable. Some authors reported stabilization or a spontaneous decrease in the SPSP in some patients [22]. In the study by Winslow [23], the prevalence of PAH was initially 14%, and then 43% in the same patients assessed by echocardiography 5 years later. It seems to occur more frequently after several years of SLE evolution. However, it can also reveal the disease [15] or even precede it, sometimes several years in advance: an average of 7.4 ± 6.5 years in the Shen series [24].

Conclusion

Studies on the evolutionary aspect of heart disease in SLE are rare. Cardiovascular disorders are fairly common in connective tissue diseases in general, and particularly in systemic lupus. The different phases of remission and flare-ups, not to mention the associated pathologies, can explain these electrocardiographic changes. The relationship between lupus and various cardiac abnormalities noted cannot be made from these observations, hence the need to perform cardiovascular check-up, when there is systemic failure, in order to make an early diagnosis of heart defects.

References

1. Meyer O, Khan M, Peltier A (2000) *Maladies et syndrome systémiques*. Paris, Ed. Flammarion: 131-289.
2. Nikpour M, Gladman DD, Ibanez D, Bruce IN, Burns RJ, et al. (2009) Myocardial perfusion imaging in assessing risk of coronary events in patients with systemic lupus erythematosus. *J Rheumatol* 36: 288-294.
3. Task Force for Diagnosis and Treatment of Pulmonary Hypertension of European Society of Cardiology (ESC); European Respiratory Society (ERS); International Society of Heart and Lung Transplantation (ISHLT), Galie N, Hoeper MM, Humbert M, Torbicki A, et al. (2009) Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Respir J* 34: 1219-1263.
4. Chevalier X, Flipo R, Goupille P (2000) *Système immunitaire et l'inflammation Abrégé de Rhumatologie Paris Edition Masson* 81: 337-349.
5. Cacciapuoti F, Galzerno D, Capogrosso P (2005) Impairment of left ventricular function in systemic lupus erythematosus evaluated by measuring myocardial performance index with tissue Doppler echocardiography. *Echocardiography* 22: 315-319.
6. Sturfelt G, Eskilsson J, Nived O, Truedsson L, Valind S (1992) Cardiovascular disease in systemic lupus erythematosus. A study of 75 patients from a defined population. *Medicine (Baltimore)* 71: 216-223.
7. Laraki R, Blétry O, Godeau P (1992) [Lupus pericarditis]. *Ann Med Interne (Paris)* 143: 233-237.
8. Urowitz MB, Gladman D, Ibañez D, Fortin P, Sanchez-Guerrero J, et al. (2008) Accumulation of coronary artery disease risk factors over three years: data from an international inception cohort. *Arthritis Rheum* 59: 176-180.
9. Wijetunga M, Rockson S (2002) Myocarditis in systemic lupus erythematosus. *Am J Med* 113: 419-423.
10. Rosenbaum E, Krebs E, Cohen M (2009) The spectrum of clinical manifestations, outcome and treatment of pericardial tamponade in patients with systemic lupus erythematosus: a retrospective study and literature review. *Lupus* 18: 608.
11. Smiti M, Salem TB, Larbi T, Sfaxi AB, Ghorbel IB, et al. (2009) [Pericarditis in systemic lupus erythematosus: prevalence and clinical and immunologic characteristics]. *Presse Med* 38: 362-365.
12. Francès C, Bécherel P et Piette J (2000) Manifestations dermatologiques du Lupus. *Encycl Méd Chir* 10: 495-498.
13. Danchenko N, Satia JA, Anthony MS (2006) Epidemiology of systemic lupus erythematosus: a comparison of worldwide disease burden. *Lupus* 15: 308-318.
14. Dioum A, Ndiaye FS, Ndongo S (2010) La prévalence des manifestations hémato-immunologiques et leurs implications pronostiques au cours de la maladie lupique. A propos de 140 observations et une revue de la littérature. *Dakar Med* 57: 59-62.
15. Tazi-mezaiek Z, Harmouch H, Adnoui M (2000) Particularités du lupus érythémateux disséminé au Maroc. A propos de 166 observations. *Rev Méd Interne* 1: 465-466.
16. Basset A, Hocquet P, Sow A.M (1960) Description du 1er cas de lupus chez le Noir Africain au Sénégal. *Bull Soc Méd Afr Noire Lgue Frse* 5: 17.
17. Elliott JR, Manzi S (2009) Cardiovascular risk assessment and treatment in systemic lupus erythematosus. *Best Pract Res Clin Rheumatol* 23: 481-494.
18. Bruce IN, Urowitz MB, Gladman DD, Ibañez D, Steiner G (2003) Risk factors for coronary heart disease in women with systemic lupus erythematosus: the Toronto Risk Factor Study. *Arthritis Rheum* 48: 3159-3167.
19. Klinkhoff AV, Thompson CR, Reid GD, Tomlinson CW (1985) M-mode and two-dimensional echocardiographic abnormalities in systemic lupus erythematosus. *JAMA* 253: 3273-3277.
20. hejtmancik Mr, Wright Jc, Quint R, Jennings Fl (1964) The Cardiovascular Manifestations Of Systemic Lupus Erythematosus. *Am Heart J* 68: 119-130.
21. Asanuma Y, Oeser A, Shintani AK, Turner E, Olsen N, et al. (2003) Premature coronary-artery atherosclerosis in systemic lupus erythematosus. *N Engl J Med* 349: 2407-2415.
22. Apte M, McGwin G Jr, Vilá LM, Kaslow RA, Alarcón GS, et al. (2008) Associated factors and impact of myocarditis in patients with SLE from LUMINA, a multiethnic US cohort (LV). [corrected]. *Rheumatology (Oxford)* 47: 362-367.
23. Gottenberg J, Roux S, Assayag P (2004) Cardiomyopathies spécifiques au cours du lupus érythémateux systémique : à propos de 3 cas. *Rev Rhum* 78: 25-28.
24. Nihoyannopoulos P, Gomez PM, Joshi J, Loizou S, Walport MJ, et al. (1990) Cardiac abnormalities in systemic lupus erythematosus. Association with raised anticardiolipin antibodies. *Circulation* 82: 369-375.
25. Khamashta MA, Cervera R, Asherson RA, Font J, Gil A, et al. (1990) Association of antibodies against phospholipids with heart valve disease in systemic lupus erythematosus. *Lancet* 335: 1541-1544.
26. Cervera R, Font J, Paré C, Azqueta M, Pérez-Villa F, et al. (1992) Cardiac disease in systemic lupus erythematosus: prospective study of 70 patients. *Ann Rheum Dis* 51: 156-159.
27. Lameira D, Lejeune S, Mourad JJ (2008) [Metabolic syndrome: epidemiology and its risks]. *Ann Dermatol Venereol* 135 Suppl 4: S249-253.
28. Winslow TM, Ossipov MA, Fazio GP, Simonson JS, Redberg RF, et al. (1995) Five-year follow-up study of the prevalence and progression of pulmonary hypertension in systemic lupus erythematosus. *Am Heart J* 129: 510-515.
29. Shen JY, Chen SL, Wu YX, Tao RQ, Gu YY, et al. (1999) Pulmonary hypertension in systemic lupus erythematosus. *Rheumatol Int* 18: 147-151.