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Short Notes

Therapeutic Response of Patients followed for Systemic Lupus Erythematosus in the Rheumatology Department of the University Hospital of Bogodogo

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Abstract

Objective: To analyze the therapeutic response of patients followed for systemic lupus erythematosus in the rheumatology department of the university hospital of Bogodogo from January 2006 to December 2020.

Method: This was a cross-sectional and analytical study with retrospective data collection conducted in the rheumatology department of the university hospital of Bogodogo in Burkina Faso, from January 1, 2006 to December 31, 2020. The diagnosis of SLE was based on the existence of at least four of the ACR/EULAR criteria, with quantitative or qualitative evidence of immunopositivity to at least anti-native DNA and anti-Sm autoantibodies.

Results: Hydroxychloroquine was prescribed as background treatment in 27 patients (93.10%). Under treatment, the evolution was marked by clinical remission in 15 patients (55.17%), and a mean SELENA-SLEDAI score of 4.2 ± 3.97 with extremes of 0 to 13 compared to 9.07 ± 6.64 with extremes of 0 to 23 of mean SELENA-SLEDAI score (p=0.001), over one year of follow-up. Immunologically, the mean anti-dsDNA antibody level was 93.08 ± 117.43 IU at diagnosis versus 20.08 ± 13.48 IU at control (p=0.041), with a mean time interval of 5.25 ± 4.01 years with extremes of 0.41 and 15 years.

Conclusion: A clinical improvement and an immunological response were observed under treatment.

INTRODUCTION

Systemic lupus erythematosus (SLE), is a chronic systemic autoimmune disease with mainly osteoarticular and dermatological expression, associated with the production of multiple autoantibodies, the most characteristic of which are directed against certain components of the nucleus such as native DNA [1]. Epidemiologically, it is the most common connective tissue disease [2]. It is a serious disease with a multifactorial cause involving environmental, hormonal and genetic factors, leading to a dysfunction of the immune system, hormonal and cellular hyperactivity, responsible for the production of numerous autoantibodies and various tissue lesions [1]. Therapeutically, the treatment of SLE is based on synthetic antimalarials, in particular hydroxychloroquine, corticosteroid therapy, nonsteroidal anti-inflammatory drugs, immunosuppressants depending on the severity of the disease, and biotherapy, which has brought a new dimension to the therapeutic management of autoimmune diseases [1]. From an evolutionary point of view, lupus disease evolves spontaneously through successive relapses, interspersed with remissions of varying duration and quality under treatment. The "activity" of the disease is assessed by activity scores, the most important of which are the SLEDAI (Systemic lupus erythematosus disease activity index), and the BILAG (British Isles Lupus Activity Group). These scores are

based on the presence or absence of clinical and biological signs, with different weightings according to the severity of the organ damage observed [3]. These scores allow practitioners to define whether the patient is in remission or not. Many studies have been conducted on the evolution of treatment in practice, both clinically and immunologically in Western countries, but very few in Africa, hence the purpose of our study.

PATIENTS AND METHODS

The rheumatology department of the Bogodogo University Hospital in Burkina Faso served as the setting for our study. It was a descriptive cross-sectional and analytical study with retrospective data collection covering the period from January 2006 to December 2020. All patients with a diagnosis of SLE based on the existence of at least 04 ACR/EULAR criteria with quantitative or qualitative evidence of immunopositivity to at least anti-native DNA and anti-Sm autoantibodies were included. Data were entered and analyzed on a microcomputer using SPSS version 2.0 software. Tables and graphs were made with Microsoft Excel 2019 software.

Categorical variables were expressed as frequency. Quantitative variables were expressed as mean and standard deviation or median and interquartile range. Comparison of quantitative variables was performed using a Pearson correlation test. The comparison of qualitative variables was done by calculating the Odd ratio. The risks are defined by an Odd Ratio > 1 if the test is significant. A significance threshold of 0.05 was used.

RESULTS

Twenty-nine (29), patients were included in our study. The mean age of the patients was 32 ± 8.60 years with extremes of 08 and 46 years. 96.55% were female, i.e. a sex ratio of 0.04. The average duration of treatment was 5.25 years ± 4.01 years with extremes of 0.41 and 15 years. The cutaneous and rheumatological manifestations were represented by 44.86% of photosensitivities, 75.86% of lupus skin lesions, 79.31% of arthralgias and 27.59% of arthritides were observed at diagnosis. The average 24-hour proteinuria was 0.93 ± 1.7 g/24h at the time of diagnosis with extremes of 0.01 and 1.85 g/24h. It was positive in 13 patients. Table 1 compares the distribution of patients according to organ involvement at diagnosis and control. Concerning the immunological profile, anti-native DNA and anti-Sm antibodies were positive in 68.97% of patients. The mean value of anti-native DNA antibodies was 93.08 ± 117.43 IU with extremes from 0 to 380 IU at diagnosis versus 20.08 ± 13.38 IU with extremes from 0 to 47 IU at control (p=0.041). Table 2 compares the autoantibody levels of patients at diagnosis and control. Hydroxychloroquine was administered in 93.10% of patients. The mean SELENA-SLEDAI 2 score was 4.62 ± 3.97 with extremes of 2 to 13 compared to 9.07 ± 6.64 with extremes of 0 to 23 mean SELENA-SLEDAI 1 score (p=0.001). Table 3 shows the comparison of the SELENA-SLEDAI 1 and 2 scores. Clinical remission was observed in 55.17% of patients at the time of control.

DISCUSSION

We noted a significant clinical improvement in rheumatological manifestations (P=0.004). Our results were comparable to those of Aouhab in Illinois, USA [4]. This result shows the therapeutic effectiveness of the molecules indicated for the management of SLE [1]. Regarding the renal manifestations, in our sample the renal function was disturbed in 13 patients at diagnosis, with a positive mean 24-hour proteinuria of 0.93 ± 1.7g/24h. Our results are superior to those of Diop in Senegal in whom renal function was disturbed in 8 (11.1%) patients [5]. On the other hand, Shariati-Sarabi in Iran did not note any cases of renal impairment in his sample of 41 patients [6]. The average 24-hour proteinuria at control was 0.44 ± 0.51g/24h. Our result could be explained by the frequency of renal involvement at diagnosis and during the course of SLE as described in the literature [7], or by the existence of a urogenital tract infection that should have been ruled out by a uroculture examination. Hydroxychloroquine was the most commonly administered drug, accounting for 93.10% of cases. In general, in West Africa, the diagnosis of SLE remains

Table 1: Distribution of patients according to organ damage at diagnosis and control.

	Diagnosis	Control	*p*-value
	staff		
Rheumatological manifestations			
Arthralgia	23	11	0.0003
Arthritis	8	3	
Myalgia	2	3	
Cutaneous manifestations			
Lupus skin lesions	22	4	0.2
Alopecia	13	2	
Photosensitivity	13	0	
Oral mucosal lesions	1	1	
Neurological manifestations			
Polyradiculoneuropathy	1	0	0.045
Headache	0	3	
Respiratory Manifestations			
Pleurisy	3	2	0.35
Bronchitis	1	0	
Bronchopneumopathy	0	1	
Cardiovascular Manifestations			
PAH	1	0	-
OD dilatation	1	0	
OG dilatation	1	0	
Pericardial effusion	1	0	
Digestive Manifestations			
Gastroenteritis	3	1	0.171
Hepatomegaly	0	1	
Ocular manifestations			
Decreased visual acuity	1	0	-
Renal Manifestations			
Elevated 24-hour proteinuria	13	14	-
Hematological Manifestations			
Leukopenia	8	9	0.65
Anemia	7	8	
Thrombocytopenia	0	1	

P< 0.05: significant difference t: Pearson test

Table 2: Correlation between patients' antibody levels at diagnosis and control.

	Average	Standard deviation	*t*	*p*-value
Native DNA antibodies				
Diagnosis	93.08	117.43	2.292	0.041
Control	20.08	13.38		
Anti-Sm antibodies				
Diagnosis	135.08	173.79	0.999	0.338
Control	97.38	152.65		

P<0.05: significant difference t: Pearson test

Table 3: Comparison between the average SELENA-SLEDAI scores over one year of follow-up.

	Average	Standard deviation	*t*	*p*-value
SELENA-SLEDAI 1	9.07	6.64	3.636	0.001
SELENA-SLEDAI 2	4.62	3.97		

P<0.05: significant difference t: Pearson correlation test

difficult because of the clinical polymorphism. However, the disease seems to be benign in the sub-region with a management that remains dominated by corticosteroid therapy and synthetic antimalarials; immunosuppressants and biotherapy being of difficult use and accessibility in our countries [8].

The immunological profile of the patients at the beginning was dominated by anti-DNA and anti-Sm antibodies in 51.72% of patients. Toumin in Ivory Coast also noted immunological disorders in 85.19% of patients [9]. At the immunological control, we noted a significant decrease in the average level of anti-DNA antibodies (P=0.04), under hydroxychloroquine alone or associated with cyclophosphamide or azathioprine) over a mean treatment duration of 5.25 ± 4.01 years. Shariati-Sarabi in Iran and Gheita in Egypt, had also reported a significant decrease in anti-DNA (p<0.001)[6], (P=0.013)[10], respectively under hydroxychloroquine and cyclophosphamide. In our sample, we found two cases of auto-antibody negativation in two patients aged 20 and 08 years at control, after respectively eight and two years of background treatment with hydroxychloroquine 400mg/day. This result is lower than that of Hickman in the United Kingdom [11]. Several other authors in the literature have reported similar results with other molecules [12-15]. These results could be explained by the mechanism of action of these different molecules: hydroxychloroquine [6], cyclophosphamide [16], rituximab [17,18]. Disease activity over one year of follow-up, there was a significant decrease in SELENA SLEDAI scores from an average of 9.07 at the first appointment of the year to 4.62 at the last appointment, (p=0.001). The majority of patients had mild disease activity at the end of 2020. Our results were comparable to those of Shariati-Sarabi (p<0.001)[6], and Dario Roccatello also found an improvement in SLEDAI score from a mean of 17.3 before treatment to 3.1 after one year of rituximab and cyclophosphamide therapy [13]. Clinical remission was observed in 16 patients. This result could be explained by an effective choice of treatment by the physician, best suited to the clinical picture, as well as good compliance by the patient.

CONCLUSION

At the end of our study, we noted an evolution under treatment, marked by an improvement on the clinical level and a response on the immunological level.

REFERENCES

1. Protocole National de Diagnostic et de Soins. Lupus systémique. Paris : Filière FAI2R. 2020; 1-89.
2. Bertsias G, Ioannidis JP, Boletis J, Bombardieri S, Cervera R, Dostal C, et al. EULAR recommendations for the management of systemic lupus erythematosus. Report of a task force of the eular standing committee for international clinical studies including therapeutics. Ann Rheum Dis. 2008; 67:195-205.
3. Seror R. Évaluation thérapeutique dans le lupus érythémateuxsystémique. In : Lupus érythémateux. 1re éd. Strasbourg : Elsevier Masson. 2013; 209-21.
4. Aouhab Z, Hong H, Felicelli C, Tarplin S, Ostrowski RA. Outcomes of Systemic Lupus Erythematosus in Patients who Discontinue Hydroxychloroquine. ACR Open Rheumatol. 2019; 1: 593-599.
5. Diop MM, Guèye YA, Leye A, Touré PS, Berthe A, Ka MM. Les modes de révélation du lupus érythémateux systémique à Dakar (Sénégal): à propos d'une série de 161 cas. Rev Afr Med Int. 2014; 1:12-15.
6. Shariati-Sarabi Z, Tavakol Afshari J, Mahmoudi M, Dormanesh B, Jahandoost F, Khoshdel Ar, et al. Efficacy analysis of hydroxychloroquine therapy in systemic lupus erythematosus: a study on disease activity and immunological biomarkers. Inflammopharmacol. 2018; 26: 1175-1182.
7. Meyer O. Lupus érythémateux systémique. EMC - Rhum Orthop. 2005; 2: 1-32.
8. Zomalheto Z, Assogba M, Agbodande A, Atadokpede F, Gounongbe M, Avimadje M. Lupus érythémateux systémique : particularités au Bénin et en Afrique de l'Ouest. Tunis Med. 2014; 92: 707-710.
9. Toumin C, Mamady D, Mamoudou C, Michel K, Bakari T, Lamine K, et al. Présentation clinique, traitement et évolution du lupus systémique au CHU de Treichville à Abidjan. Health Sci Dis. 2021; 22: 88-91.
10. Gheita TA, Abaza NM, Hammam N, Mohamed AA, El-Gazzar II, Eissa AH. Anti-dsDNA titre in female systemic lupus erythematosus patients: relation to disease manifestations, damage and antiphospholipid antibodies. Lupus. 2018; 27:1081- 1087.
11. Hickman RA, Hira-Kazal R, Yee CS, Toescu V, Gordon C. The efficacy and safety of rituximab in a chart review study of 15 patients with systemic lupus erythematosus. Clin Rheumatol. 2015; 34: 263-271.

12. Tew GW, Rabbee N, Wolslegel K, Hsieh H-J, Monroe JG, Behrens TW, et al. Baseline autoantibody profiles predict normalization of complement and anti-dsDNA autoantibody levels following rituximab treatment in systemic lupus erythematosus. Lupus. 2010; 19: 146-157.

13. Roccatello D, Sciascia S, Rossi D, Alpa M, Naretto C, Baldovino S, et al. Intensive short-term treatment with rituximab, cyclophosphamide and methylprednisolone pulses induces remission in severe cases of SLE with nephritis and avoids further immunosuppressive maintenance therapy. Nephrol Dial Transplant. 2011; 26: 3987-3992.

14. Jónsdóttir T, Gunnarsson I, Risselada A, Henriksson EW, Klareskog L, van Vollenhoven RF. Treatment of refractory SLE with rituximab plus cyclophosphamide : clinical effects, serological changes, and predictors of response. Ann Rheum Dis. 2008; 67: 330-334.

15. Aguiar R, Araújo C, Martins-Coelho G, Isenberg D. Use of Rituximab in Systemic Lupus Erythematosus: A Single Center Experience Over 14 Years. Arthritis Care Res. 2017; 69: 257-262.

16. Ponticelli C, Moroni G. Hydroxychloroquine in systemic lupus erythematosus (SLE). Expert Opin Drug Saf. 2017; 16: 411-419.

17. Terrier B, Mouthon L. Lupus érythémateux systémique - traitements par anticorps monoclonaux et molécules recombinantes. Med Sci (Paris). 2013; 29: 65-73.

18. Lekpa FK, Marini A, Claudepierre P, Chevalier X. Intérêt du rituximab dans le traitement du lupus érythémateux systémique. Lettre Rhumatol. 2008; 340: 12-14.

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