



Clinical Continuum of Systemic Lupus in Saudi Arabia

Abdul Sattar Khan*, Naushad Abid and Rabel Khawaja

Family & Community Medicine Department, College of Medicine, King Faisal University, Saudi Arabia

*Corresponding author: Abdul Sattar Khan, Family & Community Medicine Department, College of Medicine, King Faisal University, Saudi Arabia, E-mail: drsattarkhan@gmail.com

Received date: March 17, 2016; Accepted date: April 05, 2016; Published date: April 22, 2016

Copyright: © 2016 Khan AS, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Systemic Lupus in Saudi Arabia

Systemic lupus erythematosus (SLE) is a clinically heterogeneous disease, which is autoimmune in origin, and characterized by the presence of autoantibodies directed against nuclear antigens. It is, by definition, a multi-system disease, and patients can present in different ways. The disease occurs in all populations, but the prevalence, spectrum and severity of the disease vary worldwide [1].

Saudi Arabia is a country in which people of different ethnic origin as well as expatriates live together. There are a few reports describing the clinical and laboratory features of SLE from different regions of the Kingdom [2] including our retrospective study from Eastern Saudi Arabia [2]. The purpose of this short review is to compare the clinical spectrum of adult systemic lupus in different regions of Saudi Arabia.

Looking at the prevalence of SLE in Arab countries studies suggest that the disease is prevalent in Arab world [3]. Al Attia in 2006 conducted a retrospective study in Abu Dhabi and found a total of 71 patients setting over a 12-year period. Of those 71, fifty-six patients were reported with SLE and 15 were considered to have borderline SLE. However, age and female sex distribution were not different in the two sub groups [4].

The etiology of SLE is unknown, however; genetic component significantly contributes as one of the major factors responsible especially in countries like Saudi Arabia, where the rate of consanguineous marriages is high. The study done by Qari et al. [5] identified the familial component of SLE. They reported five SLE positive families out of the seven, who were consanguineous and the mode of inheritance was autosomal recessive [5]. The human leukocyte antigen (HLA) has extensively been associated with the susceptibility to SLE. Motwee et al. [6] suggested the association between MHC (full form) class I and class II (HLA-A*29, HLA-B*51, HLA-DRB1*15, and HLA-DQB1*06) and susceptibility to SLE in the Saudi population. HLA-DRB1*15-DQB1*06 haplotype showed the highest risk factor for the disease in Saudi population, that is similar to the African American patients [6].

Studies done in eastern and central regions of the kingdom have shown that patients with SLE are found to be between the age group 20-30 [2,6-8]. However, there is change in the age group when compared to the data from the Riyadh region, which shows the low number for this age group 9 out of 46 patients in eastern region study were females [2] as compared to the population from western [7] and central Saudi Arabia [8], where female to male ratios were 5.5:1 and 9:1, respectively. The difference in age and sex distribution in study from Eastern region was attributed to relatively lower number of patients (only 46 patients), short duration of study as well as the inclusion of Saudi population only [2].

Lupus nephritis was seen in all regions of Saudi Arabia. Type 1V lupus nephritis was seen in 58.7% of the patient from eastern region and the results are comparable to other regions a [2,7-9]. The outcome of lupus nephritis varied among different regions. Our study from eastern region suggested that one patient progressed to chronic kidney disease and rest of them showed improvement [2]. However, the study from the Riyadh region on a larger sample showed a higher number of patients progressing towards chronic kidney disease, which was attributed to the older age of onset, presence of hypertension, elevated serum creatinine and proliferative lupus nephritis [9]. Improved survival in Eastern and Western regions was attributed to the younger age of onset, normal initial serum creatinine and early response to treatment with immunosuppressive drugs [2,7].

Non-specific constitutional symptoms like fatigue and malaise was seen in all 46 patients from Eastern region study and this was attributed to F to anemia but the possibility of other contributing factors like depression and sleep deprivation were not excluded, as identification of these factors needs thorough neuropsychiatric assessment [2]. Lack of patients' awareness about this symptom was considered as a contributor for low prevalence of fatigue in other region [7].

Fever was seen in all 46 patients from Eastern Saudi Arabia [2] and this was similar to the results of a study from the central region [10]. But was reported at high rate in western region as compared to eastern and central regions of Saudi Arabia [7]. Fever was related to multiple factors such as increased disease activity [2] or underlying infections [10]. Neuropsychiatric manifestations were similar in almost all the regions [2,7,8]. However headache was also seen in about 28.3% of patients from eastern Saudi Arabia, which is higher as compared to the other regions [2,5,6].

Musculoskeletal manifestations seen in the cases from Eastern region were mostly arthralgia and myalgia [2] and this was different from other studies in which arthritis was found to be more prevalent [7,8,10]. Cardiovascular and pulmonary manifestation seen in different ranged from serositis to deep venous thrombosis were almost similar in all the regions [2,5,10]. Hematologic abnormalities seen in most patients were anemia and thrombocytopenia but the study from eastern region [2] suggested more patients with thrombocytopenia than in Western Saudi [7]. Anemia in all the studies was multi factorial in origin and varied from anemia of chronic disease to hemolytic [2,5,7,8,10]. Serologic test results from almost all the studies were found to be similar with high titers ANA and AntiDsDNA. However, relatively lower complement levels were observed from Eastern region [2] and this was attributed to increased disease activity.

The common causes for hospital admission in most studies were infections and active lupus [2,10,11]. Systemic infections were related to multiple factors, including high disease activity reflected by

hypocomplementemia, renal disease and the use of immunosuppressive drugs, including steroids [10,11]. Infections and active SLE were the common etiologies reported [11]. In a study by Alzeer [12] studying the outcome of Saudi Systemic lupus patients in ICU, it was found that the SLE patients admitted to the ICU had a lower mortality rate than some of the previous reports. Patients with SLE with high APACHE score, $>$ or $=20$, poor health status, thrombocytopenia and multi organ dysfunction syndrome had poor prognosis in the ICU [2].

In conclusion systemic lupus in Saudi Arabia is seen more in young females of child-bearing age. Clinical features are variable; however renal disease and nonspecific constitutional features were seen similar in almost all the studies. Prognosis is also variable and infections and active disease were the main determinant of mortality. Prospective studies are needed with collaboration among different regions to elaborate further the characteristics of the disease.

References

1. Tikly, Mohammed, Navarra SV (2008) Lupus in the developing world—is it any different? *Best Practice & Research Clinical Rheumatology* 22: 643-655.
2. Abid N, Khan AS, Al Otaibi FH (2013) Systemic lupus in Eastern Saudi Arabia. A comparative study. *Lupus* 22:1529-1533.
3. Locus, Gene Map “Systemic Lupus Erythematosus.” Centre for Arab Genomic studies 2015.
4. Al Attia, Haider M (2006) Borderline systemic lupus erythematosus (SLE): a separate entity or a forerunner to SLE? *International journal of dermatology* 45: 366-369.
5. Qari A, Al-Mayouf S, Al-Owain M (2009) Mode of inheritance in systemic lupus erythematosus in Saudi multiplex families. *Genet Couns* 20: 215-223.
6. Al-Motwee S, Jawdat D, Jehani GS, Anazi H, Shubaili A, et al. (2013) Association of HLA-DRB1*15 and HLA-DQB1*06 with SLE in Saudis. *Ann Saudi Med* 33: 229-234.
7. Al Arfaj AS, Khalil N (2009) Clinical and immunological manifestations in 624 SLE patients in Saudi Arabia. *Lupus* 18: 465-473.
8. Qari FA (2002) Clinical pattern of systemic lupus erythematosus in western Saudi Arabia. *Saudi Med J* 23: 1247-1250.
9. Al Arfa AS, Khalil N, Al Saleh S (2009) Lupus nephritis among 624 cases of systemic lupus erythematosus in Riyadh, Saudi Arabia. *Rheumatol Int* 29: 1057-1067.
10. Al-Rayes H, Al-Swailem R, Arfin M, Sobki S, Rizvi S, et al. (2007) Systemic lupus erythematosus and infections: A retrospective study in Saudis. *Lupus* 16: 755-763.
11. Heller T, Ahmed M, Siddiqi A, Wallrauch C, Bahlas S (2007) Systemic lupus erythematosus in Saudi Arabia: Morbidity and mortality in a multiethnic population. *Lupus* 16: 908-914.
12. Alzeer AH, Al-Arfaj A, Basha SJ, Alballa S, Al-Wakeel J, et al. (2004) Outcome of patients with systemic lupus erythematosus in intensive care unit. *Lupus* 13: 537-542.