HAEMATOLOGICAL PATTERN IN SAUDI NATIONALS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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SUMMARY: A case-control study was conducted on a group of Saudi patients suffering from systemic lupus erythematosus (SLE), in an attempt to compare the values of haematological parameters in SLE patients with those in normal age and sex matched controls. Females were mainly less than 40 years old and males were in the 35-72 years age group. The results of haematological parameters showed mild to severe anaemia in 68.75 % of the patients, leukocytosis in 6.25 % and leukopenia in 15.6 % of the patients. Majority of the SLE patients had normocytic - normochromic anaemia and all patients had elevated erythrocyte sedimentation rate.

Key Words: Systemic Lupus Erythematosus, Haematological Pattern, Saudi Arabia.

INTRODUCTION

Systemic Lupus Erythematosus (SLE), a multisystem disorder, is largely a disease of unknown aetiology, though both genetic and environmental factors seem to play an important aetiological role in susceptible individuals (25). It is a chronic and inflammatory disease that may affect the skin, joints, kidney, nervous system, various membranes or other organs of the body, producing various abnormalities that often differ from one individual to another (21). There is sufficient evidence to suggest that the tissue injury associated with SLE is a result of abnormal immune mechanisms in which there is some disturbed tolerance or failure of self - recognition (19, 20). Antibodies produced against many cellular components and native DNA and antigenantibody complex are demonstrated in the blood of patients suffering from SLE and hence it is classified as an immune-complex disease (20).

Clinically, wide variations are reported in the manifestations of SLE (2, 6, 9, 23, 24). In addition haematological, biochemical and immunological abnormalities are encountered at a variable prevalence in SLE patients (3, 4, 9, 17).

We conducted studies on a group of Saudi SLE patients in an attempt to determine the haematological, biochemical and immunological abnormalities in these patients. This paper presents the abnormalities of the common haematological parameters in Saudi SLE patients and compares the results with those in normal age and sex matched controls.

MATERIALS AND METHODS

Thirty-two Saudi SLE patients attending the Rheumatology Clinics at the Riyadh, Al-Kharj Hospital (RKH), and diagnosed applying the criteria of the American Rheumatism Association (7) for SLE diagnosis were investigated. This study also included 32 age and sex matched normal controls

who had volunteered to be included in the study. The SLE patients were 23 females and 9 males with ages ranging between 13-65 years in females and 35-72 years in the males.

Ten milliliters of blood was collected from each patient prior to receiving any treatment and from the members of the control group by venipuncture in EDTA tubes. Haematological parameters and red cell indices i.e. mean cell volume (MCV), mean cell haemoglobin (MCH) and mean cell haemoglobin concentration (MCHC), were estimated in fresh blood using Coulter Counter Model - S - Plus with a haemoglobinometer attachment (Coulter Electronic Limited, Cold Harbour Lane, Harpenden, Hartfordshire, England).

Ten milliliters of blood was collected in tubes containing trisodium citrate solution (3.1 %) for the estimation of erythrocyte sedimentation rate (ESR) using the Westergren Method (8).

RESULTS

The results of total haemoglobin (Hb), red blood cell count (RBC), white blood cell count (WBC), packed cell volume (PCV), the red cell indices, and ESR were fed on the computers at the Computer Centre, King Saud University, Riyadh. Using the Statistical Analysis System (SAS), the mean and standard deviation were obtained separately for the patients and controls. The significance of the difference in the mean of any parameters in the patients and control group was determined by applying the Students 't' test. P values less than 0.05 were considered as statistically significant.

The age distribution in the SLE patients (male and female separately) is presented in Figure 1. In the female group SLE was more common in the younger age group compared to the males.

The distribution of the haematological parameters and the red cell indices in the SLE patients and the control group are presented in Figures 2-4. The mean, standard deviation and the P values are presented in Table 1, and the prevalence of abnormality in the haematological parameters in SLE patients in presented in Table 2.

DISCUSSION

Diversity of the clinical presentation, tissue involvement, and prognosis in SLE is the cause of it being regarded more as a syndrome rather than a disease. It is suggested that the clinical heterogeneity

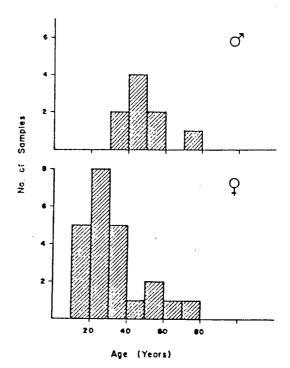


Fig - 1: Frequency distribution histogram showing the age distribution in male and female SLE patients.

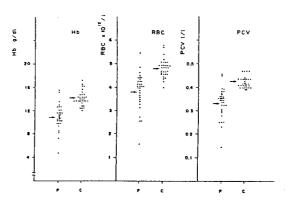
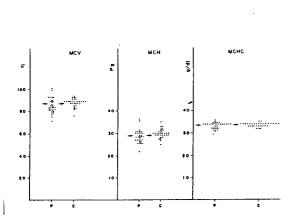


Fig - 2: Distribution of Hb, RBC, and PCV in the SLE patients and controls. The arrow points to the mean value.

P = SLE Patients; C = Control

of SLE reflects host variables and different aetiological stimuli (6). Hence, each SLE patient may apprear different from another in several aspects.

The investigation of haematological values in Saudi SLE patients and comparison of the results with those of age and sex matched controls revealed that the prevalence of anaemia (i.e. Hb level of 12.0 g/dl or lower) was high and 68.75 % (i.e. 22/32) of the SLE patients were anaemic where as 50 % patients had Hb levels of 11.0 g/dl or lower. In most of the anaemic patients the haemoglobin level was above 10 g/dl, though 8 patients had Hb levels less than 10 g/dl. In all patients except two the anaemia



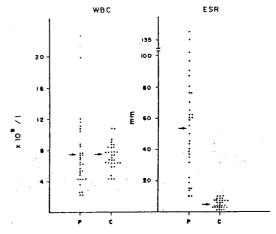


Fig - 3 : Distribution of MCV, MCH and MCHC in the SLE patients and controls. The arrow points to the mean value. $P = SLE\ Patients; \qquad C = Control$

Fig - 4: Distribution of WBC and ESR in the SLE patients and controls. The arrow points to the mean value.

P = SLE Patients; C = Control

	Mean ± SD		
Parameters	SLE Patients	Control	P*
Total Hb (g / dl)	10.90 ± 2.11	14.13 ± 1.2	0.0001
RBC x 10 ¹² /l	3.79 ± 0.72	4.76 ± 0.47	0.0001
WBC x 10 ⁹ /l	7.65 ± 5.22	7.31 ± 1.8	0.727
PCV (1/1)	0.328 ± 0.061	0.425 ± 0.029	0.0001
MVC (fl)	85.65 ± 6.93	87.45 ± 3.75	0.219
MCH (pg)	28.73 ± 2.95	28.92 ± 2.07	0.176
MCHC (g/dl)	33.27 ± 1.17	33.69 ± 0.81	0.114
ESR (mm)	53.67 ± 34.85	5.03 ± 2.75	0.0001

 \ast P values less than 0.05 were considered statistically significant. Table 1 : The value of haematological parameters in SLE patients and age and sex matched controls.

Parameters	Abnormality	No. of SLE Patients	Prevalence (%)
Total Hb	Hb ≤ 12 g / dl	22	68.75
	Hb≤11 g/dl	16	50.0
RBC	< 0.38 x 10 ¹² /l	10	31. 25
PCV	≤ 0.37 1/1	24	75.0
WBC	> 12 x 10 ⁹ /l	2	6. 25
	$\leq 3.8 \times 10^9 / 1$	5	15. 63
ESR	≥ 20 mm	32	100
MCV	< 75 fl	2	6. 25
МСН	< 25 fl	2	6. 25
MCHC	< 30 g/dl	0	0

Table 2 : Prevalence of abnormalities of haematological parameters in SLE patients.

was normo chromic-normocytic. Two of the male patients were severely anaemic with Hb levels of 4.7 and 7.2 g/dl. In addition, two of the patients (both females) had hypochromic - microcytic anaemia, indicative of either iron deficiency or associated thalassaemia, a genetic disorder occurring at a high prevalence in different regions of Saudi Arabia (11, 12). Low haematocrit (<0.37 1/1) was encountered in 75 % (24/32) of the patients. Other studies have also reported a high prevalence of anaemia in SLE patients ranging from 38 % in some reports (14) to 78 % in others (16). In most reports the anaemia encountered was normochromic-normocytic (4, 13, 22), however, in a few studies majority of the SLE patients had microcytic-hypochromic anaemia and in these patients defective intramedullary iron utilization was suggested (5). Several other causes of anaemia in SLE patients (4), both immune and nonimmune, which are known to accompany several inflammatory process, are also suggested in SLE patients (9).

Leukopenia was first noticed in SLE patients in 1923 (15). Later, reports showed variable prevalence ranging from 35 % (14) to 80 %(16). In the present study 15.6 % of the Saudi SLE patients had leukopenia which is the lowest prevalence so far reported in all studies. Leukopenia in SLE patients is indicative of active disease and is believed to be caused by the production of antibodies against the leukocytes or in some cases due to decreased marrow production or marrow inhibition (10, 18, 26). Both granulocytopenia and lymphopenia have been reported in SLE patients and a number of immunologic and non-immunologic causes including central or peripheral destruction of the granulocytes and lymphocytes have also been suggested (4).

In the present study, leukocytosis was encountered in 6.25 % of the patients. Leucocytosis is rare and occurs in SLE patients with associated active infections. However, several SLE patients may have intercurrent infections without any rise in the white cell count (9).

The most common finding in the Saudi SLE patients was an elevation of the ESR. In all other studies elevation of ESR has commonly been reported in upto 94 % of the SLE patients (1). However, normal rates may occur with active disease. This increase in sedimentation rate is partially attributed to the red cells clumping for rouleaux formation to occur, often because of the associated abnormal antibodies in SLE (9).

In summary, this study has revealed several haematological abnormalities in Saudi SLE patients. It has also shown that though majority of the patients may have one or more haematological abnormalities, there are some patients who appear haematologically normal and do not present any signs of anaemia or leukopenia except for an elevated ESR.

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REFERENCES

- Armas Cruz R, Harnecker J, Ducach G, Jalil J, Gonzalez F: Clinical diagnosis of systemic lupus erythematosus. Amer J Med 1958; 25: 409.
- Ballou SP, Khan MA, Kushner I: Clinical features of systemic lupus erythematosus. Arthritis Rheum 1982; 25 (1): 55-60.
- 3. Bonafede RP, Staden MV, Klemp P: Hepatitis B Virus infection and liver function in patients with systemic lupus erythematosus. J Rheumatol 1986; 13 (6): 1050-1952.
- Budman DR, Steinberg AD: Hematologic aspects of systemic lupus erythematosus-Current concepts. Ann Intern Med 1977; 86: 220-229.
- 5. Cartwright GE, Lee GB: The anaemia of chronic disorders: Br J Haemat 1971; 21: 147-152.

- Christian CL: Systemic lupus erythematosus Clinical manifestations and prognosis. Arthritis Rheum 1982; 25 (7): 887-888.
- Cohen AS, Reynolds WE, Franklin EC, Kulka JP, Ropes MW, Shulman LE, Wallace SL: Preliminary criteria for the classification of systemic lupus erythematosus. Bull Rheum Dis 1971; 21: 643-648.
- Dacie JV, Lewis SM: Practical Haematology. Churchill Livingstone, Edinburg 1975; 580-582.
- Dubois EL, Wallace DJ: Clinical and laboratory manifestations of systemic lupus erythematosus. In: Wallace DJ, Dubois EL (eds). Lupus Erythematosus, Lea and Febiger, Philadelphia 1987; 317-449.
- Duckhman DJ, Rhyne RL Jr, Smith FE: Retardation of colony growth of invitro bone marrow culture using sera from patients with Felty Syndrome, disseminated lupus erythematosus (SLE), rheumatoid arthritis and other disease states. Arthritis Rheum 1975; 18: 323-333.
- El-Hazmi MAF: Haemoglobin disorders: A pattern for thalassaemia and haemoglobinopathies in Saudi Arabia. Acta Haemat 1982: 68: 43-51.
- El-Hazmi MAF: Abnormal haemoglobins and allied disorders in the middle east Saudi Arabia. In: Bowman JE (ed).
 Distribution and Evolution of Haemoglobin and Globin Loci 1983; 239-249.
- Estes D, Christian CL: The natural history of systemic lupus erythematosus by prospective analysis. Medicine (Baltimore) 1971; 50: 85-95.
- Fries JF, Holman HH: Systemic lupus erythematosus, A clinical analysis. In: Major Problems in Internal Medicine IV. Philadelphia. WB Saunders Co 1975; 79.
- Goecherman WH: Lupus erythematosus as a systemic disease, JAMA 1923; 80: 542-547.
- Harvey AM, Shulman LE, Tumulty PA, Conley CL, Schoenrich EH: Systemic lupus erythematosus: Review of literature and clinical analysis of 138 cases. Medicine (Baltimore) 1954; 33: 291-437.
- Isenberg DA, Patterson KG, Todd Pokropek A, Snaith ML, Goldstone AH: Haematologic aspects of systemic lupus erythematosus. A reappraisal using automated methods. Acta Haemat 1982; 67: 242-248.
- Kimbal MR, Wolf SM, Talal N: Marrow granulocyte reserves in the rheumatic diseases. Arthritis Rheum 1972; 16: 345-352.
- Mackey I: Autoimmunity in relation to lupus erythematosus. In: Wallace DJ, Dubois EL (eds). Lupus Erythematosus. Lea and Febiger, Philadelphia 1987; 44-52.
- Nakamura RM, Tucker ES: Immune complex diseases. In: Ritzmann SE, Daniels TL (eds). Serum Protein abnormalities - Diagnostic and clinical aspects. Little, Brown and Company, Boston 1975; 295-330.

- Schur PH: Systemic lupus erythematosus. In: Wyngaarden JB, Smith LH (eds). Cecil Textbook of Medicine, WB. Saunders Company, Philadelphia 1985; 1852-1857.
- Shearn MA, Pirofsky B: Disseminated lupus erythematosus: Analysis 34 cases. Archs Intern Med 1952; 90: 790-807.
- Studenski S, Allen NB, Caldwell DS, Rice JR, Polisson RP: Survival in systemic lupus erythematosus - A multi - variate analysis of demographic factors. Arthritis Rheum 1983; 30 (12): 1326-1332.
- 24. Syrop CH, Varner MW: Systemic lupus erythematosus. Clin Obst Gynecol 1983; 26 (3): 547-556.
- Talal N: The etiology of systemic lupus erythematosus. In: Wallace DJ, Dubois EL (eds). Lupus Erythematosus. Lea and Febiğer, Philadelphia 1987; 39-43.
- Wolf SM, Kimbal HR, Talal N: Abnormal granulocyte response to etiocholanolone in rheumatic diseases (abstract). Clin Res 1969: 16: 326.