ORIGINAL RESEARCH PAPER
Volume-8 | Issue-10 | October - 2019 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CROSS SECTIONAL STUDY ON QUANTITATIVE & QUALITATIVE ALTERATION IN PLATELETS IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE).

Pathology

Vijayta Singh Assistant Professor, Autonomous state medical college, Bahraich.

Mili jain* Assistant Professor, Department of Pathology, K. G. M .U, Lucknow. *Corresponding Author

ABSTRACT

Patient of SLE presents with wide range of clinical manifestations. Thrombocytopenia is a common hematological manifestations with prevalence ranging from 7to 30% in large series SLE patients and is an independent risk factor for increased mortality. Thrombosis with prevalence >10% is also a major complication in SLE patients. Aims of this study is to evaluate quantitative and qualitative alterations in platelet parameters in SLE patients.
Prospective enrollment of 40 newly diagnosed SLE patients was done. Clinical evaluation for thrombotic/ bleeding symptoms along with assessment of platelet count (PC), and aggregation(PA) was done. They were not under any medication that might interfere with platelet function.
Comparison between cases and controls shows significant alteration in PA in cases compare to age matched controls. PA was decreased in 11 out of 40 patients (p value 0.027) and thrombocytopenia was seen in 5 out of 40 patients (p value 0.544). Follow up SLE patients with thrombocytopenia had increased mortality 3 out of 5 in compare to 5out of 32 total follow up in SLE patients.
We concluded that PA and PC were altered in SLE patients but these alterations had no correlation with thrombotic and bleeding manifestation. However SLE patients with thrombocytopenia presented with worsening of symptoms during follow up.

KEYWORDS

Systematic Lupus Erythematosis (SLE), Platelet aggregation (PA), Platelet count, clinical manifestations.

INTRODUCTION-

Systemic lupus erythematosus (SLE) is an autoimmune disease in which organ and cells undergo damage initially mediated by tissue binding autoantibodies and immune complexes and leads to various clinical manifestations. Ninety percent of patients at the time of diagnosis are female of childbearing age. Prevalence of SLE in united state at the time of diagnosis is 10 to 400 per 1,00,000 depending upon race and gender[1].

Patient of SLE presents with wide range of clinical manifestations. The major hematologic manifestations of SLE are hemolytic anemia, leukopenia (<4000/µl) lymphopenia (<1500/µl), and thrombocytopenia (<100,000/µl) in absence of any offending drugs[1].

SLE is rare in India with prevalence is less than 3.2 per 1,00,000 and the median age of onset in Indian SLE patients is 24.5 years and the gender ratio of female to male is 11.1 [2].

Thrombocytopenia is common hematological manifestations in patients of SLE and the causes of thrombocytopenia is heterogeneous but the most common mechanism is auto antibodies against platelet surface receptors may leads to thrombocytopenia and defective platelet aggregation (PA).

Aims of this study are to evaluate quantitative and qualitative alterations in platelet parameters in SLE patients.

MATERIALS AND METHODS:

Prospective enrollment of 40 newly diagnosed SLE patients and 40 controls of same age and sex attending rheumatology OPD was done in department of pathology, KGMU Lucknow in year 2015-2016. Clinical evaluation for thrombotic / bleeding symptoms along with assessment of platelet count (PC), and PA was done.

Patients on anti platelet drugs including asprin were excluded from the study.

Platelet count**:** Manual as well as automated platelet counts was done.
Platelet aggregation studies- Further evaluation of a suspected defect of platelet function was performed through study of platelet aggregation in response to platelet-stimulating agent. These studies will be performed using platelet-rich plasma (PRP) harvested from blood collected using sodium citrate as the anticoagulant. In patients with abnormally high circulating platelet counts, patients PRP will be diluted with their own platelet-poor plasma to allow standardization of platelet aggregometry. Aggregation studies will be done within three hours of sample collection using 5 micro moles of ADP in AGGRO/LINK.

Data was entered into microsoft excel and analyzed using Epi Info 7.1.3.0 statistical software. Data was expressed in term of mean and SD. P value <0.05 was taken as significant.

RESULT:

Comparison between cases and controls shows alteration in PA and PC however this alteration was statistically significant in PA (Table 1). PA was decreased in 11 out of 40 patients (p value 0.027) and thrombocytopenia in 5 out of 40 patients (p value 0.544). Follow up during one year 3 out of 5 SLE patients with thrombocytopenia presented with worsening of symptom.

Table 1. Comparison of Various Investigations in Cases and Controls

Investigations	Cases			Controls			Statistical signi. (Student t test)	
	No.	Mean	SD	No.	Mean	SD	't'	'p'
Platelet count (lacs)	40	1.99	0.92	40	2.11	0.76	-0.609	0.544
Platelet Aggregation	35	53.40	22.44	40	67.00	12.45	-3.298	0.002
MPV	40	10.32	2.37	40	9.39	1.10	2.257	0.027

Above laboratory investigations were performed in the cases as well as in controls. Mean values of Platelet counts and Platelet aggregation of Cases were found to be lower than that of Controls while mean values of MPV of Controls was found to be lower than that of Cases. On applying student t-test difference in mean values of Platelet aggregation and MPV between Cases and Controls was found to be statistically significant.

Table 2. : Comparison of Laboratory Investigations of Cases

	Patients with thrombosis(A)			Patients without thrombosis(B)			Statistical Significance Betwee A And B	
	NO.	MEAN	SD	NO.	MEAN	SD	't'	'P'
Platelet Aggregation	6	52.50	15.59	29	53.59	23.84	0.106	0.916
Platelet count (lacs)	6	1.76	0.62	34	2.03	0.96	0.658	0.514
MPV	6	12.03	2.09	34	10.01	2.31	-1.996	0.053

We divide patients of SLE into two groups with thrombosis (A) and without thrombosis (B) (table.2). In this study 15% patient with SLE had thrombotic manifestation. However if we compare mean values of PC, PA and MPV there is no statistical significant correlation between cases with and without thrombotic manifestation .

DISCUSSION-

In the present study, thrombocytopenia was seen in 12.5% of patients with SLE as compared to control. The mean platelet count in SLE patients was 1.99±0.99 lac/cumm and 2.11±0.76lac/cumm in controls, however this correlation was not statistically significant (Table11).

M. Kuwana et al studied that thrombocytopenia was the major hematological complication in patients with systemic lupus erythematosus (SLE), the pathogenesis of thrombocytopenia in SLE patients was heterogeneous, but the most common mechanism was believed to be increased platelet clearance mediated by anti-platelet autoantibodies. Their finding demonstrate that two types of autoantibodies causing thrombocytopenia in patients with systemic lupus erythematosus were anti-GPIIb/IIIa and antiTPOR (thrombopoietin Recepters) antibodies. [3].

Cines DB demonstrated the other potential mechanisms causing thrombocytopenia include thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, haemophagocytic syndrome, antiphospholipid syndrome and impaired thrombopoiesis. Antiplatelet autoantibodies in ITP patients preferentially recognize platelet surface glycoproteins (GP), and the most common target was GPIIb/IIIa [4].
In present study, platelet aggregation with ADP was reduced in 70.6 % of SLE patients with mean value was (53.40±22.4) % and this correlation was statistically significant (p value 0.002). (Table1, 2).

However if we takes cut off for platelet aggregation is 50%, in our study 34.29 % patients had platelet aggregation below 50% which is considered as decreased platelet aggregation. It was also seen that only 2.86 % of patients had raised platelet aggregation.

Thrombosis is seen in only 15% SLE patients and is not related to abnormal platelet aggregation.

Hence it concludes that defective platelet aggregation is not a causative factor for thrombosis. In the study of Dorsch CA et al. it was suggested that defective platelet aggregation in SLE patients may be partially related to a storage pool deficiency state [5].

However study of Pereira et al [6] concluded that activated platelets are known to participate in thrombus formation. There was evidence that platelet activation occurs in SLE and was associated with thrombosis.
Escolar G et al. investigated the platelet-aggregating effect of plasma from systemic lupus erythematosus (SLE) patients (n = 19) under flow conditions. Aliquots of the SLE plasmas with (n = 10) or without (n = 9) anticardiolipin antibodies (ACAs) were added to anticoagulated blood. Plasma from normal donors was used as a control. Results of the present study indicate that the presence of APAs in SLE plasma promotes platelet aggregation under flow conditions. These observations may help to explain the pathophysiology of the thrombotic events occurring in patients with APAs [7].

CONCLUSION-

PA and PC were attributed to antiplatelet antibodies in SLE patients but these alterations had no correlation with thrombotic and bleeding manifestation. However SLE patients with thrombocytopenia presented with worsening of symptoms during follow up.

REFERENCES-

1. Bevra Hannahs Hahn, Longo DL, Fauci A, Kasper D, Hauser S, Jameson J et al. Systemic Lupus Erythematosus. In D.L.Longo. Harrison's principles of internal medicine, 18th edition. New York. The Mc Graw hill professionals. 2011; 391(2):2724-35.
2. Malviya AN, Singh YN, Singh RR, Kapoor SK et al. Prevalance of SLE in India. Lupus1993; 2: 115-8.
3. Kuwana M, Kaburaki J, Okazaki Y, Miyazaki H, Ikeda Y. Two types of autoantibody-mediated thrombocytopenia in patients with systemic lupus erythematosus. Rheumatology. 2006 july; 45 (7):851-54.
4. Cines DB, Blanchette VS. Immune thrombocytopenic purpura. N Engl J Med. 2002 Mar 28 ;346(13):995–08 .
5. Dorsch CA, Meyerhoff J. - Arthritis Rheum. Mechanisms of abnormal platelet aggregation in systemic lupus erythematosus. August 1, 1982; 25 (8); 966-73
6. Pereira J, Alfaro G, Goycoolea M, Quiroga T, Ocqueteau M, Massardo L, et al. Circulating platelet-derived microparticles in systemic lupus erythematosus. Association with increased thrombin generation and procoagulant state. Thromb Haemost. 2006 Jan; 95(1):94-9.
7. Escolar G, Font J, Reverter JC, López-Soto A, Garrido M, Cervera R et.al. Plasma from systemic lupus erythematosus patients with antiphospholipid antibodies promotes platelet aggregation. Arterioscler. Thromb. February 1, 1992; 12 (2):196-200.