

Association of clinical presentation with anti-nuclear antibody specificities among patients with systemic lupus erythematosus

Sir,

Systemic lupus erythematosus (SLE) is characterized by the production of a wide range of autoantibodies directed

against a multiplicity of autoantigens. Anti-nuclear antibody (ANA) test by immune fluorescence assay (IFA) is the gold standard test while ANABLOT detects the majority of autoantibodies in SLE patients. The human epithelial carcinoma-2 cell line is used to detect ANAs, which gives different patterns according to the antigens against which autoantibodies are produced. These autoantibodies are detected by indirect IFA.^[1] Strong associations between ANA specificities and IFA patterns have been observed. Specificity for anti-dsDNA, histone, and DNA/histone gives homogeneous-ANA (H-ANA) pattern, whereas Sn-RNP, SmD1, U1RNA give speckled-ANA (S-ANA) pattern. Other patterns observed are nucleolar-ANA (N-ANA) and cytoplasmic-ANA (C-ANA) pattern.^[2,3]

This study included 100 (96 females) patients with SLE (mean age of onset 27 ± 9.5 years) where in the association between ANA specificities and clinical presentations were studied. The mean SLEDAI score at clinical evaluation was 6.0 ± 7.5 . These patients were further categorized as lupus nephritis (LN) ($n = 56$) and SLE without nephritis ($n = 44$) based on WHO criteria. ANA was tested by IFA using commercial kits from AESKUSLIDES, Germany and ANA specificities were detected by ANABLOT, AESKUBLOTS, Germany. The association between autoantibody specificities and clinical manifestations were calculated by Fisher's exact test. Frequency of ANA, anti-dsDNA autoantibodies were 100% and 84%, respectively. Out of 100 SLE patients studied, H-ANA pattern was seen in 75% patients, S-ANA in 19% N-ANA in 4%, and C-ANA pattern in 2% patients by IFA. H-ANA pattern and dsDNA autoantibody positivity was strongly associated with clinical manifestations such as renal manifestations (odds ratio [OR] = 10, $P = 0.0026$), malar rash (OR = 4.444, $P = 0.046$), with oral ulcers (OR = 25.6, $P = 0.0014$). Similarly, H-ANA pattern and anti-SmD1 antibody positivity was also associated with malar rash (OR = 9.318, $P = 0.003$), oral ulcer (OR = 7.625, $P = 0.0376$). Also H-ANA pattern and anti-Ro/SS-A antibodies were associated with photosensitivity (OR = 8.167, $P = 0.0113$) [Table 1].

There are ethnic differences in association of clinical manifestations and ANA specificities. In a study from Sweden, S-ANA pattern was reported to be less associated with anti-dsDNA antibodies as compared to organ damage and H-ANA pattern among LN patients. The present study showed an association of H-ANA pattern with malar rash, photosensitivity and oral ulcers with multiple ANA specificities. Till today, the entire geoepidemiological picture of SLE showing

Table 1: Association of ANA specificities (by ANA BLOT) with clinical manifestations in SLE patients (n=100) and with H-ANA pattern (n=75)

Autoantibody specificities	All SLE (n=100)						
	Arthritis (n=70)	Malar rash (n=56)	Photosensitivity (n=36)	Oral ulcers (n=10)	Renal involvement (n=56)	Neurological disorders (n=8)	Hematological disorders (n=16)
Anti-dsDNA (n=84)	18	14	6	6	52	5	1
Anti-Sm (n=40)	16	10	8	4	12	4	0
Anti-nRNP (n=22)	4	2	2	0	10	0	4
Anti-Ro/SS-A (n=16)	0	0	10	0	6	0	0
Anti-La/SS-B (n=8)	2	2	0	0	4	0	0
Autoantibody specificities	H-ANA pattern (n=75)						
	Arthritis (n=56)	Malar rash (n=32)	Photosensitivity (n=24)	Oral ulcers (n=6)	Renal involvement (n=44)	Neurological disorders (n=4)	Hematological disorders (n=6)
Anti-dsDNA (n=68)	16	8	6	4	42	2	0
Anti-Sm (n=38)	12	10	8	3	20	2	0
Anti-nRNP (n=16)	2	0	1	0	8	0	3
Anti-Ro/SS-A (n=10)	0	0	6	0	5	0	0
Anti-La/SS-B (n=4)	2	0	0	0	2	0	0

H-ANA: Homogenous pattern in ANA by IFA, ANA: Anti-nuclear antibody, SLE: Systemic lupus erythematosus, IFA: Immune fluorescence assay

variations in the clinical manifestations and associated autoantibodies is not clear in India. More reports from different regions of the country are needed to throw light on the association of clinical manifestations and ANA specificities.^[4,5]

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References

1. Rahman A, Isenberg DA. Systemic lupus erythematosus. *N Engl J Med* 2008;358:929-39.
2. Fritzler MJ. Clinical relevance of autoantibodies in systemic rheumatic diseases. *MolBiol Rep* 1996;23:133-45.
3. Janwityanuchit S, Veraseritniyom O, Vanichapuntu M, Vatanasuk M. Anti-Sm: Its predictive value in systemic lupus erythematosus. *ClinRheumatol* 1993;12:350-3.
4. Frodlund M, Dahlström O, Kastbom A, Skogh T, Sjöwall C. Associations between antinuclear antibody staining patterns and

clinical features of systemic lupus erythematosus: Analysis of a regional Swedish register. *BMJ Open* 2013;3:e003608.

5. Alba P, Bento L, Cuadrado MJ, Karim Y, Tungekar MF, Abbs I, *et al.* Anti-dsDNA, anti-Sm antibodies, and the lupus anticoagulant: Significant factors associated with lupus nephritis. *Ann Rheum Dis* 2003;62:556-60.

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