IS THERE ANY RELATIONSHIP BETWEEN SEROLOGICAL RESPONSIVENESS TO THE EPSTEIN-BARR VIRUS ANTIGENS AND SERUM LEVELS OF MANNOSE BINDING LECTIN IN PATIENS WITH LUPUS NEPHRITIS AND PRIMARY GLOMERULONEPHRITIDES?

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INTRODUCTION

A link between the Epstein-Barr virus (EBV) infection and the development of systemic lupus erythematosus (SLE) and some forms of primary glomerulonephritides (PGN) has been suggested. On the other hand, it has been shown that insufficiency of mannose binding lectin (MBL) retards the EBV infection. MBL appears to have a dual mode of action. Increased MBL leads to enhanced complement activation and tissue damage, while its deficiency results in aggravation of autoimmunity and increased susceptibility to secondary infections.

AIM

The aim of our study was to compare the levels of serum MBL and antibodies to EBV antigens in patients with PGN, lupus nephritis (LN) and healthy controls (C).

MATERIAL AND METHODS

Patients with **lupus nephritis**	**32**
- Active lupus nephritis (aLN)	**23**
- Inactive lupus nephritis (inLN)	**9**
Patients with **primary glomerulonephritides**	**78**
- IgA-Mesangial proliferative GN (MesPGN)	26
- Non-IgA-MesPGN	26
- Membranoproliferative GN (MPGN)	2
- Idiopathic membranous GN (IMGN)	15
- Focal segmental glomerulosclerosis (FSGS)	6
Healthy controls (C)	**36**

a-LN means SLEDAI ≥10 including symptoms of renal involvement.

The serum levels of MBL and antibodies (Abs) to EBV early antigen (EA), EBV viral capsid antigen (VCA) and EBV nuclear antigen-1 (EBNA-1) were determined in immunoglobulin (Ig) G, A and M classes using the specified enzyme-linked immunosorbent assays.

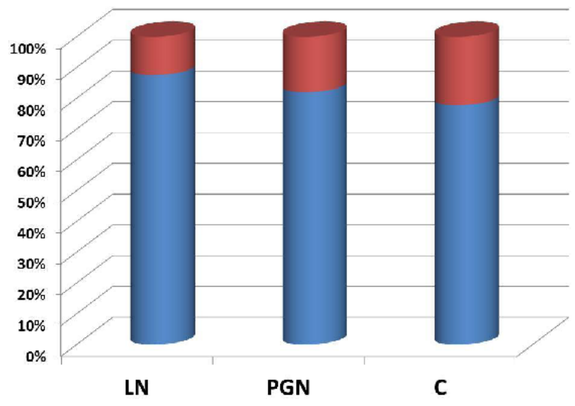

Fig. 1. Percentage of subjects positive for EBV EBNA1-IgG (in blue) in LN, PGN and C

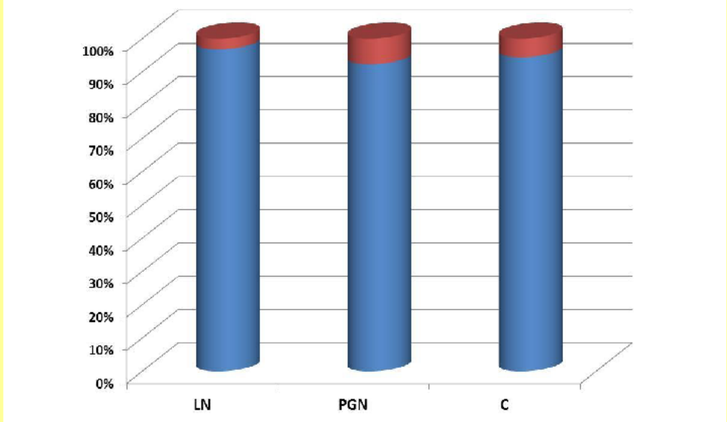

Fig. 2. Percentage of subjects positive for EBV VCA-IgG (in blue) in LN, PGN and C

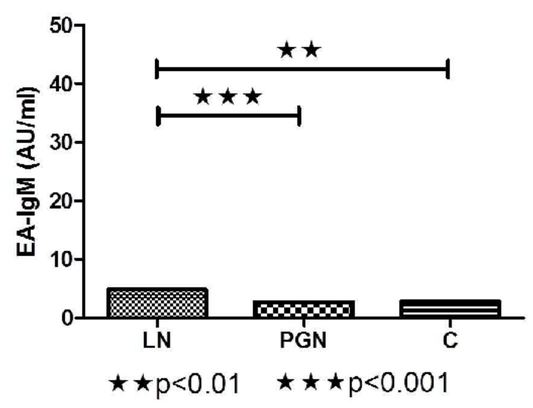

Fig. 3A. Median levels of EA-IgM in sera of patients with LN, PGN and C

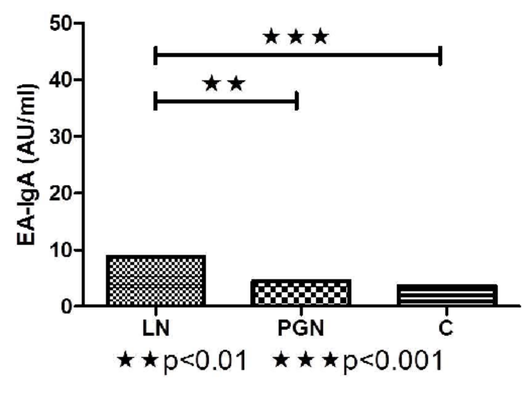

Fig. 3b. Median levels of EA-IgA in sera of patients with LN, PGN and C

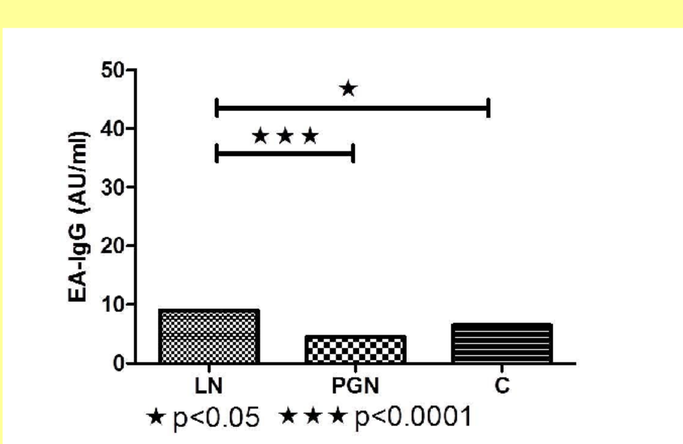

Fig. 3C. Median levels of EA-IgG in sera of patients with LN, PGN and C

RESULTS

The median levels of MBL were 1.11 (range 0.009 to 8.0) in PGN, 0.66 (range 0.015 to 4.61) in LN and 1.22 (range 0.01 to 6.64) μg/ml in C with no significant differences between these groups and also subgroups of PGN. The majority of LN (90.6%) and PGN (82%) patients, and also subjects from the C group (77.1%) were shown to be seropositive for EBNA-1 IgG revealing previous EBV infection **(Figure 1)**. This was also confirmed by seropositivity for VCA IgG in 96% of patients with PGN, 96 % of those with LN and 94% of C **(Figure 2)**. Interestingly, the median levels of anti-EA-IgM, anti-EA-IgA and anti-EA-IgG Abs in LN differed significantly compared to both PGN and C **(Figures 3A, B and C)**.

However, no significant correlation between the levels of these Abs and serum concentrations of MBL was noted in LN. In contrast, significant correlations between serum levels of MBL and anti-EBV-EA-IgA and anti-EBV-VCA-IgA Abs could be found in MesPGN ($r=0.23$; $p<0.05$ and $r=0.26$; $p<0.05$, respectively). This was particularly apparent in IgA-MesPGN, where the above correlations were significantly higher (**Figures 4A and B**).

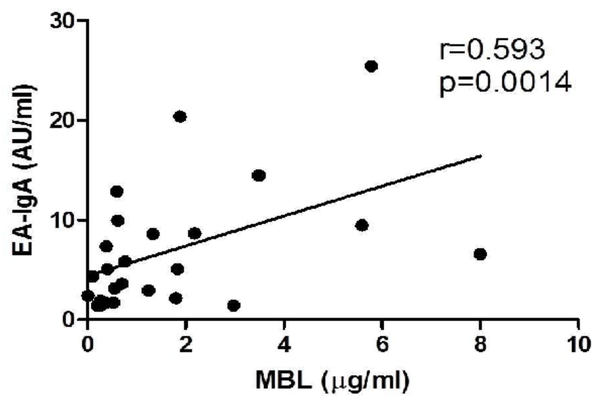

Fig. 4A. Correlation between serum levels of MBL and EA-IgA in patients with IgA-MesPGN

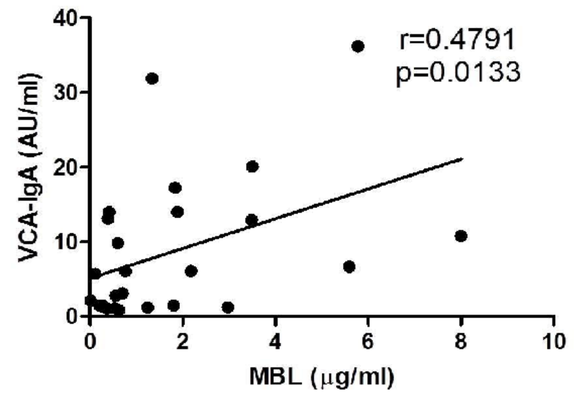

Fig. 4B. Correlation between serum levels of MBL and VCA-IgA in patients with IgA-MesPGN

CONCLUSIONS

MBL, being a component of the innate immunity system, seems to control the response to EBV in PGN (particularly in IgA-MesPGN), but this control is lost in SLE with renal involvement. Further studies are required to elucidate this issue.