Measles Virus Genome in Patients with Lupus nephritis and Glomerulonephritis

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Dear Sir,

A significant increase of mean antibody titers to measles virus has been found in systemic lupus erythematosus (SLE) [1-8] and in glomerulonephritis (GN) [7-9]; the mean antibody titers to measles virus in GN patients were similar to those in SLE patients: 5.9 and 6.1, respectively, versus 4.5 in controls [8]. The results of these investigations have suggested virus persistence in these diseases with its possible role in etiology and pathogenesis.

Measles virus persistence might be confirmed by measles genome investigation. Measles virus genome was found in lymphocytes of patients with measles [10] and in patients with subacute sclerosing panencephalitis [2, 3, 10]. The disease, apparently closely connected with persistent measles infection was very rarely detected in the lymphocytes of a healthy population. As far as we know, there is only the report by Robertson et al. [11] concerning measles virus genome in SLE. These authors found it in 1 of 3 of their patients. We did not come across such investigations in GN.

Our present work was aimed at the determination of measles virus genome in the peripheral blood lymphocytes together with antibodies to measles virus in blood serum of lupus nephritis (LN) and GN patients. Blood samples collected from 36 LN patients (2 males and 34 females, age range 15-60 years) and from 87 GN patients (12 acute and 75 chronic; 49 males and 38
females, age range 16-59 years studies. Percutaneous renal biopsy was performed in 53 of 87 GN patients and in 22 of 36 LN patients. The control group consisted of 13 patients with bone fractures and 10 pregnant women with no renal disease. Measles virus genome was investigated by dot hybridization of RNA extracted from peripheral blood lymphocytes by ultracentrifugation of guanidinium thiocyanate.

**GN**

**LN**

**Controls**

The results of measles genome investigation did not depend upon sex and age, disease duration, clinical presentation, and morphological form. The measles genome was found more often in active disease (59.3 vs. 25% in patients with chronic GN in remission).

There was no close correlation between genome detection and antibody titer. The viral genome was found in all patients patients with LN and in 63.6% of the patients with GN with a high antibody titer and in 55.0 and 51.3%, respectively, of those with a low titer (fig. 1). In none of the controls was a virus genome detected.

Our results support an association of measles virus with LN and GN. They indicate a possible pathogenetic role of virus persistence during relapse of GN.

**References**


