INCREASE OF IgG-BEARING PERIPHERAL BLOOD LYMPHOCYTES AND HYPO IgG-GLOBULINAEMIA IN MINIMAL CHANGE GLOMERULONEPHRITIS AND FOCAL GLOMERULOSCLEROSIS

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Introduction

Minimal change glomerulonephritis (MCG) is a primary glomerulonephritis with nephrotic syndrome, of unknown aetiology and pathogenesis. However various lines of evidence suggest that immunological mechanisms are involved. One of the immunological abnormalities is a decrease in serum IgG concentration, while the IgM serum level is often elevated, particularly in children. IgG decrease may not only depend on the proteinuria, which is highly selective, Giangiacomo et al [1] proposed a defect in the T-cell function which mediates conversion of IgM to IgG.

In this study we investigated the peripheral blood lymphocyte (PBL) surface markers related to serum immunoglobulin levels in 10 cases of MCG and also in 10 cases of focal glomerulosclerosis (FGS), because the latter is considered by many authors [2] to be a complication of MCG.

Material and methods

The percentage of PBL E, EA, ZYC' rosette forming cells (RFC) and lymphocytes with surface membrane immunoglobulins (SmIg) was evaluated in 20 patients with primary glomerulonephritis and nephrotic syndrome: 10 MCG (5 adults and 5 children) and 10 FGS (all adults), in 5 cases with MCG in sustained remission for 1 year after steroid therapy, and in 30 normal controls, age and sex matched. The histological type was determined by kidney biopsy. The serum concentration of immunoglobulins (IgG, IgA, IgM) was determined according to Mancini et al [3]. Lymphocytes were obtained from heparinised peripheral blood according to Boyum [4], and E-RFC were performed according to Aiuti et al [5], EA-RFC according to Meroni et al [6], ZYC'-RFC according to Huber et al [7], SmIg according to Looor et al [8] with labelled goat antisera to total human Ig and anti u,o,y,o, heavy chains, and F(ab)2 sera anti IgG in some cases. We performed this test after lymphocyte incubation at 37°C in culture medium to eliminate extrinsic Ig from the cell surface.
Figure 1
FOCAL GLOMERULOSCLEROSIS

Figure 2
Results

Figures 1 and 2 show no important differences between patients and normal controls at the level of peripheral blood E-RFC, EA-RFC, ZYC'-RFC and SM Ig cells. It should be pointed out that cells with complement receptors were investigated by mouse ZYC'. In this condition $C_{3d}$ receptors are predominantly detected.

Utilising monospecific antisera Sm IgG cells were significantly ($p<0.001$) increased, as we found also using F(ab)$_2$ sera. The other cells with surface membrane Ig were normal.

At the same time these patients presented a significant ($p<0.001$) IgG serum decrease with normality of the other Ig (Figure 3). The Sm IgG rate in MCG cases with sustained remission for 1 year, after steroid therapy was withdrawn, was normal (Figure 4).

![SERUM Ig](image)

*Figure 3*

658
Conclusions

Our results show an increase of SM IgG lymphocytes with a simultaneous decrease of IgG in both types of glomerulonephritis. This could be related to their common pathogenesis.

In our cases the IgM to IgG switch seems to occur at cellular level, so that the hypothesis of a conversion defect in IgM-IgG synthesis is unlikely [1]. Moreover the IgM values were normal.

Instead, the data suggest a selective failure of SM IgG lymphocytes which are unable to transform into IgG-secreting plasma cells, as reported in other primary Ig deficiency [9]. This failure could depend on an imbalance in T-cell function, which disappears after a long period of sustained remission.

References