MEMBRANOUS LUPUS NEPHRITIS: IMMUNOGLOBULIN DEPOSITS AND CLINICAL CORRELATIONS

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INTRODUCTION AND AIMS

Systemic lupus erythematosus (SLE) is an autoimmune disease with a nonspecific activation of autoreactive B cells and polyclonal autoantibody production with tissue deposition of immune complexes. Lupus nephritis histological hallmark, mostly in proliferative classes, is a "full-house" pattern of immunoglobulin deposition (IgG, IgA, IgM) and complement. However, Haas [1] showed that, unlike proliferative classes, membranous lupus nephritis (MLN) depicted a "full-house" pattern only in 65% of patients. In addition, Jennette *et al* [2] described even lower rates of "full-house" immunoglobulin pattern deposition, around 29%, in MLN. The importance of "full-house" pattern deposition in MLN disease expression is still a matter of debate. It remains to be determined any eventual association of "full-house" deposition with clinical disease aggressiveness or even to transformation into proliferative forms.

METHODS

- ✓ All MLN patients submitted to kidney biopsies from July 1999 to August 2007 were included
- ✓ Tissue biopsy was studied by conventional methods: light microscopy and immunofluorescence
- ✓ Patients were classified according to immunoglobulin glomerular cappilary wall deposition in "rich form" (rIF), with two or more deposited immunoglobulins, and "poor form" (pIF) with a single and exclusive IgG deposition
- ✓ Clinical and laboratorial data were collected at baseline, after one year and at the end of follow-up. Treatment was decided by the clinical staff based on literature protocols.

RESULTS

We included 15 patients in pIF group (25%) and 46 in rIF (75%). At baseline, pIF and rIF groups were similar regarding age, complement level, ANA, anti-DNA antibody, proteinuria (rIF 4.6g ± 3.6 x pIF 4.4g ± 5.7 g/day) – Table 1.

Interestingly, pIF was significantly associated with a lower eGFR (pIF 78.6 ± 40 vs rIF 96.3 ± 34 ml/min/1.73m2, p=0.04). After one year of follow-up, the rIF group showed a higher eGFR (rIF 103 ± 32 vs pIF 76 ± 36 ml/min/1.73m2, p=0.01) – Table 1.

At the end of follow up, the rIF group showed a tendency to have a higher eGFR (rIF 80 ± 39 vs pIF 63 ± 33 ml/min/1.73m2, p=0.1) - Table 1.

DISCUSSION

LMN is known as an immune complex deposit disease with all classes of immunoglobulin deposition in most patients. However some authors showed a non "full-house" pattern of deposition in 35% up to 70%. Immunoglobulin molecular – IgG, IgA and IgM – differ in their physicochemical and immunological effector mechanisms such as complement binding and inflammation profile, therefore influencing the type of injury that results after immune complex deposition. It is known that immune complexes formed by IgG induce classical complement activation with generation of membrane attack complex while IgA immune complex, as in IgA nephropathy, shows a weak complement activation, probably via the lecitin pathway. There are no literature information on clinical aggressiveness of IgG or IgA immune complexes in MLN.

	rIF	pIF	p
N	46	15	xx
Age (years)	33 ± 10	34 ± 12	ns
MDRD Baseline (ml/min/1.73m²)	96 ± 34	78 ± 40	0.04
MDRD after 1 year (ml/min/1.73m²)	103 ± 32	76 ± 36	0.01
MDRD Final (ml/min/1.73m²)	80 ± 39	63 ± 33	ns
PTN Baseline (g/day)	4.6 ± 3.6	4.4 ± 5.7	ns
PTN Final (g/day)	1.2 ± 1.8	2.1 ± 3.8	ns
C3 (mg/dL)	85 ± 32	96 ± 52	ns
Follow-up (years)	8.6	8.1	xx

Table 1. Baseline and follow-up features

SUMMARY

We found 25% of MLN patients with only one deposited immunoglobulin ("poor IF").

"Poor IF" group compared to "Rich IF" group showed:

- Lower baseline MDRD (78 ± 40 vs 96 ± 34, p 0.04)
- Lower MDRD after one year (76 ± 36 vs 103 ± 32, p 0.01)

Studies are needed to elucidate the role of different patterns of deposits in the pathogenesis or progression of MLN.

REFERENCES

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