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STATISTICAL ANALYSIS

Data were analyzed with descriptive statistics (mean ± SD), p<0.05 was considered statistically significant by using SAS. 2010. Version 9.1 ed.

RESULT AND DISCUSSION

Result

The group of relapsing-remitting multiple sclerosis (RR-MS) subjects in the relapsing phase (n=40) and an equal number of controls, the mean age of patients was 36.5±2.19 and in control was 36.5±2.19, and the mean EDSS patients was 2.66 ± 0.91. The results revealed that the mean value of serum Anti-MOG antibody concentration in patients was 554.85±39.73 and 315.20 pg/ml±28.49 in controls and the p-value was 0.0211. This means that there is a significant increase in serum Anti-MOG antibody concentration in patient compared to control (Table1 & Figure 1)

Also Anti-MOG has a negative correlation between age and concentration (r= -0.12) and significant positive correlation between expanded disability status scale (EDSS) and concentration (r= 0.28) (Table 2).

Table1: Serum levels of Anti-MOG antibody (pg/ml) in MS patients and controls

<table>
<thead>
<tr>
<th>Group(No.)</th>
<th>Mean ± SE Concentration (pg/ml)</th>
<th>Mean ± SE Age</th>
<th>Mean ± SE EDSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (40)</td>
<td>554.85 ± 39.73</td>
<td>36.5±2.19</td>
<td>2.66 ± 0.91</td>
</tr>
<tr>
<td>Control(40)</td>
<td>315.20 ± 28.49</td>
<td>36.5±2.19</td>
<td></td>
</tr>
<tr>
<td>T-test value</td>
<td>138.74 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-value</td>
<td>0.0211</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1: concentration of anti-MOG in serum of patients and controls

Table 2: Correlation coefficient (r) between anti-MOG concentration and both age and EDSS

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Variable</th>
<th>Correlation coefficient (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-MOG Ab</td>
<td>Age</td>
<td>-0.12 NS</td>
</tr>
<tr>
<td>Conc.pg/ml</td>
<td>EDSS</td>
<td>0.28 *</td>
</tr>
</tbody>
</table>

* (P<0.05), ** (P<0.01), NS: no significant.

EDSS = Expanded Disability Status Scale

Discussion

The results of this study which showed high serum levels of Anti-MOG antibody in MS patients compared to controls suggesting the role of anti-MOG Abs in demyelination and pathogenesis of MS disease.

Berger et al. showed that the occurrence of serum anti-MOG and to lesser extent antimyelin basic protein-specific IgM antibodies seemed to predict the incidence of new relapses in early MS patients [13]. Zhou et al. reported that the transduced cell lines were used to quantify antibody responses in serum. They found increased antibody reactivity to native MOG in MS patients compared with patients with other inflammatory disease of the CNS or healthy age matched control donors. According to the course of disease, all MS patient groups had higher antibody levels in serum compared with controls [14]. The number of patients with antibodies against native MOG was highest in primary progressive MS (PP-MS) patients [14].

Also antibodies against MOG cause demyelination in vitro [15] and in animal models of multiple sclerosis [16-17]. Mantegazza et al. examined serum and CSF reactivity of MOG by ELISA in a large cohort of MS patients with disease of varying severity and type. They found that mean serum levels of anti-MOG antibodies in MS and non-inflammatory -CNS patients were significantly higher than in healthy controls, while the frequency of positivity in patients with autoimmune peripheral nervous system (Aut PNS) disorders (Guillain-Barre syndrome) was similar to that of controls. These results indicate that anti-MOG antibodies are usually present only in patients with a CNS condition. Within the MS series, the secondary progressive subgroup had a significantly higher mean OD than the other MS subgroups [18]. Anti-MOG antibodies known to have demyelinating potential [15-16, 19] may be implicated in the degenerative changes (axonal and gial loss) characteristic of the secondary progressive form of the disease [20-22]. Mazzucco et al. found that MS patients and those with other neurological diseases, but not controls, had serum reactivity against a synthetic glycosylated MOG fragment but not against non glycosylated fragments [23].

Lassmann et al. found that the structure of the CNS lesions depended on the balance between encephalitogenic T cells and anti-MOG antibody. When EAE was induced with circulating anti-MOG antibody resulted in ubiquitous perivascular demyelination in the spinal cord and medulla oblongata [24].

Berger et al. showed that patients with a clinically isolated syndrome, the initial detection of serum antibodies against MOG and MBP predicts early conversion to clinically definite multiple sclerosis, whereas the absence of these antibodies suggests that the patient will remain disease-free for several years [13]. Patients who were seropositive for both anti-MOG and anti-MBP antibodies had clinically definite multiple sclerosis within a mean of 7.5 months. They found that the seropositivity for anti-MOG or both anti-MOG and anti-MBP antibodies, but not the number of lesions seen on MRI, was associated with an increased risk of relapse of multiple sclerosis. Thus, Berger et al. study demonstrates the importance of analysis of these antibodies, which is simple to perform and less expensive than MRI, can be used to predict the individual risk of the first relapse and therefore of clinically definite multiple sclerosis. So for patients who have seronegative antibody, may have a chance of remaining relapse-free for several years after the initial demyelinating event [25] and immunomodulatory therapy might be postponed until necessary [13].

The predictive value of this antimonyl antibodies may be important for counseling purposes or for early treatment to prevent the disease from getting worse [26, 25].

Mantegazza et al. also found a direct correlation between disability (EDSS score) and anti-MOG titer in progressive forms of the disease[18], which is in agreement with the present study which shows that there is a significant positive correlation between EDSS and anti-MOG concentration (r= 0.28). By contrast Karmi et al. found no correlations between serum anti-MOG antibody levels and clinical parameters (disease course, EDSS score, and disease duration) [22].

Matsiota et al also showed that MS patients have higher levels of natural autoantibodies in their CSF compared to both healthy controls and patients with other neurological diseases [27]. Zhou et al. also identified serum antibodies against a strictly conformational epitope of MOG. The occurrence of antibodies with demyelinating properties further supports the pathogenic role of the humoral immune system in MS and calls for the development of B cell [28, 29].

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In contrast Iglesias et al. shown that MOG-specific Abs and T cells are present in healthy controls as well as in MS patients [30].

Also previous studies supported the present study by showing that concentration of Anti-MOG Abs in MS patients is higher than healthy subjects [14, 18], and several papers have reported the presence of anti-MOG antibodies in the serum and cerebrospinal fluid (CSF) of MS patients [31-36]. This immune response, when compared to a healthy control, was significantly elevated, implying an active antigen driven process.

The results of the present study showed the critical role of anti-MOG Abs in MS disease and the demyelinating potential of this biomarker and give a hope for early treatment to prevent the disease from progressing. Further studies on more MS populations and also, in vitro assessment of Anti-MOG Abs interaction are recommended to confirm the present results.

CONCLUSIONS

In this study of relapsing remitting multiple sclerosis patients have been identified and documented. MS appears to be a heterogeneous disease, with the presence of Ab-superimposed pattern. Inflammatory demyelinating diseases of the CNS includes MS could be better defined in the future according to the humoral response. The finding of specific and sensitive diagnostic parameters that allow the early diagnosis of MS will help to better define the most suitable therapeutic option or to develop new therapeutic agents. The study of auto-Abs is certainly an important way to better understanding the pathogenesis and to specify the different subtypes of CNS demyelinating diseases and to find an early therapeutic solution for the patients.

ACKNOWLEDGEMENTS

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REFERENCES

10. Anne H., Cross and Waubant E. MS and the B cell controversy. BBA-Molecular Basis of Disease 2010;07:020.


