SHORT REPORT

Acute oropharyngeal palsy is associated with antibodies to GQ1b and GT1a gangliosides

C P O’Leary, J Veitch, W F Durward, A M Thomas, J H Rees, H J Willison

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assays were performed with a panel of gangliosides comprising GM1, GM2, GM3, GD1a, GD1b, GD3, GT1a, GT1b, and GQ1b in a standard enzyme linked immunosorbent assay (ELISA), and a thin layer chromatography (TLC) overlay technique as previously described. Titres were calculated by end point dilution analysis. Our normal ranges for antiganglioside IgG antibodies are set at <1/500 (2 SD above the mean of a panel of normal and disease control serum samples). IgG subclass analysis was performed as previously described.

For statistical analysis the antibody titres were log transformed. Unpaired Student's t test was then used to assess the differences of the titres of anti-GQ1b and anti-GT1a antibodies between the group of three oropharyngeal patients and the 10 patients with Miller Fisher syndrome. The ratios (GQ1b:GT1a and GT1a:GQ1b) of the log transformed titres were estimated for each individual patient and the Mann-Whitney U test was used to assess the differences in the ratios between the two groups.

### Results

IgG antibodies to GQ1b and GT1a gangliosides were found in high titer in all three patients (table 2). Reactivities found by ELISA were confirmed using TLC overlay (not shown). Raised antibodies to GD3 (two patients) or GD1b (one patient) were also present but remained within the normal range. No patients had detectable antibodies to GT1b, GM1, or to the other gangliosides tested. IgG subclass analysis showed that both the anti-GQ1b and anti-GT1a IgG antibody reactivities resided in the IgG3 subclass in patients 1 and 2, and in the IgG1 subclass in patient 3 (data not shown).

All 10 patients with Miller Fisher syndrome had raised antibody titres (table 2) to GQ1b and nine had raised GT1a antibodies. The patient with Miller Fisher syndrome with a low GT1a titre (1/220) had clearly raised titres to GQ1b (1/2600) and to GT1b (1/3200). Many of the patients with Miller Fisher syndrome and oropharyngeal patients also had detectable antibody titres to structurally related gangliosides such as GD1b, GT1b, and GD3 (table 2). Although these titres were below 1/500 in many instances and would not be considered significant in isolation, the fact that they occurred in the presence of high titres to GT1a and GQ1b does suggest that they are a significant element of this acute immune response.

In the oropharyngeal patients the mean antibody titres to GT1a (1/6500) were higher than those to GQ1b (1/2250); by contrast with the patients with Miller Fisher syndrome the mean GT1a titre (1/5000) was lower than the mean GQ1b titre (1/8000). However, statistical analysis of the titres to either antigen showed no significant differences between the patients with oropharyngeal palsy and patients with Miller Fisher syndrome (P = 0.19 for GQ1b; P = 0.78 for GT1a). Similarly analysis of the ratios of the paired values between both groups just failed to achieve significance at the 5% level (0.05 < P < 0.10).

### Discussion

The oropharyngeal variant of Guillain-Barré syndrome may occur in isolation but usually occurs as a transitional syndrome with other features of either Guillain-Barré syndrome or Miller Fisher syndrome. Although these three patients may fit into a transitional category as
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with their very close structural similarity. The absence of reactivity with GT1b indicates that a terminal disialylated galactose (common to both GQ1b and GT1a but absent on GT1b) is an essential feature of the reactive epitope. The relative reactivity with GQ1b and GT1a does not statistically correlate with the clinical pattern of cranioropharyngeal involvement in the Miller Fisher syndrome or oropharyngeal patient groups. However, the data tend towards the GT1a/GQ1b titre ratio being higher in the oropharyngeal group than in the Miller Fisher syndrome group, suggesting that anti-GQ1b and anti-GT1a antibodies may be markers for oropharyngeal and pharyngeal palsy respectively. In addition, the presence of anti-GD1b or anti-GD3 antibodies seems to be associated with more prominent sensory involvement, as previously suggested. The few patients studied preclude detailed statistical analysis but these concepts are compatible with previously published data. Although the relative reactivity of GQ1b in human ocular nerves compared with peripheral nerve has been analysed and found to be high, its quantitative relation with GT1a and other disialylated gangliosides throughout the cranio-ocular region is unknown.

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