IgG immunoadsorption in experimental allergic neuritis: effect on antibody levels and clinical course

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SUMMARY The effect of IgG immunoadsorption upon the course of chronic experimental allergic neuritis (EAN) is described. Miniature membrane plasma separators coupled with a Protein A (PA)-Sepharose immunoadsorbent column were used to perform upon conscious rabbits 5 IgG immunoadsorption treatments over 6 days. Quantitation of anti-myelin IgG and IgM by ELISA revealed that 55–65% of plasma IgG was removed per treatment. Rapid post-treatment antibody rebound was observed for anti-myelin IgG although no antibody overshoot above control levels could be observed. Anti-myelin IgM levels remained relatively unaffected by PA immunoadsorption. Comparisons of clinical scores between control and treatment animals showed that IgG immunoadsorption was significantly beneficial (day 1 post-treatment p < 0.001; day 2 post-treatment p < 0.05). However, rapid relapse was observed in all treatment animals such that by day 3 post-treatment no significant clinical difference between control and treatment groups could be observed. IgG immunoadsorption suppresses the clinical progression of chronic EAN in a manner similar to that seen with plasma exchange. This finding suggests that antibody modulates early disease pathogenesis.

The results from large controlled clinical trials have established that plasma exchange (PE) is an effective therapy for certain cases of Guillain-Barré syndrome (GBS). However, such trials of PE do not elucidate the mechanisms of disease nor the exact means by which benefit is derived. They do, however, suggest that humoral factors play a role in at least the early disease phase of GBS and leave open the possibility of specific targetting of suspected pathogenic factors such as antibodies.

Protein A immunoadsorption is attracting increasing scrutiny as a technique applicable in a therapeutic setting in humans. The main advantage of IgG immunoadsorption using protein A compared with PE is that it is a closed loop system which removes a suspected pathogenic factor without the necessity of wasteful discard and replacement of a large proportion of a patient’s plasma protein, the majority of which may be normal and fully functional.

In both acute experimental allergic neuritis (EAN) and chronic EAN PE has been shown to be of clinical benefit and this further confirms the usefulness of EAN as an experimental model of human peripheral demyelinating neuropathies such as GBS and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). Because of the potential use for protein A immunoadsorption in a variety of neuromuscular disorders including GBS and CIDP involving suspected pathogenic immunoglobulins, the effects of IgG removal using protein A immunoadsorption were examined in animals with chronic EAN.

Materials and methods

Induction of EAN Female outbred NZW rabbits (mean body weight 2.8 ± 0.2 kg) were housed in individual cages and given food and water ad libitum. To produce EAN the animals were inoculated with peripheral nervous system myelin prepared from fresh bovine cauda equina using a discontinuous sucrose density gradient centrifugation method similar to that of Norton and Poduslo. The inoculum consisted of bovine myelin, sterile saline and Freund’s complete adjuvant containing 0.5 mg/ml Mycobacterium tuberculosis (Commonwealth Serum Laboratories, Melbourne, Australia) mixed in the ratio of 1 g:1 ml:2 ml respectively and homogenised (Polytron, Kinematica). Under halothane/oxygen anaesthesia each animal received a single subcutaneous multiportal inoculation consisting of 0.25 ml to each paw and 0.25 ml to two separate sites on the back.

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Clinical assessment and blood sampling
Animals were periodically weighed and examined for signs of EAN and a clinical disability score assigned according to the classifications in table 1. Animals were scored independently by two observers during each clinical examination.

Two ml blood samples were taken by venepuncture from peripheral ear veins into sequestrene tubes and centrifuged at 500 g for 10 minutes. Plasma samples were stored at −70°C.

Blood access
Blood access was obtained via an externalised carotid-jugular arteriovenous shunt inserted under halothane/oxygen anaesthesia as described previously.4 Control animals underwent a sham operation under halothane/oxygen anaesthesia in which the right carotid artery and jugular vein were isolated and ligated.

Extracorporeal circuit
The extracorporeal circuit is depicted in fig 1. The membrane plasma separators were of the hollow fibre type made of polypropylene (Enka AG, Wuppertal, FRG). The filters had an effective surface area of 200 cm² with a maximum pore size of 0.5 μm. The blood side filter priming volume was approximately 2 ml. The immunoadsorption column was constructed using PA-Sepharose CL-4B (Pharmacia, Uppsala, Sweden) swollen and packed between 30 μm polyethylene supports in a 25 ml glass column with a length/diameter ratio of 6:1. Based upon the known IgG binding capacity sufficient PA-Sepharose was packed to enable a total binding capacity of approximately 500 mg representing at least 50% of the circulating plasma IgG in the rabbit. This column capacity for IgG has been confirmed previously.1

The column void volume was approximately 15 ml. Blood and plasma flow rates were regulated by a multi-channel peristaltic pump (Gilson Medical, Paris, France). Blood flow through the filter was maintained at 12 ml/min while plasma flow through the column was maintained at 2.5 ml/min enabling a plasma residence time within the gel of approximately 6 minutes. Total extracorporeal blood circuit volume was approximately 15 ml.

Immunoadsorption procedures
As animals reached a clinical grade of 2–3 they were paired and randomly allocated into control and treatment groups. Immediately after allocation the treatment animals received an externalised arteriovenous shunt and control animals underwent a sham operation.

Before each immunoadsorption procedure the extracorporeal circuit was primed with 0.15 M sterile saline. Animals were lightly restrained by wrapping in a surgical drape and each remained conscious and calm throughout the procedure. Just prior to connection to the circuit, rabbits received 1500 U heparin intravenously and this was allowed to distribute within the intravascular space. A pre-treatment blood sample was taken and the arterial extracorporeal line leading to the filter was then connected to the shunt and the blood circuit primed under arterial pressure to expel residual saline. When the blood circuit was complete the venous extracorporeal line was connected to the shunt and the pump started. The first 20 ml of saline effluent from the column were discarded after which time the post-column plasma line was reconnected to allow treated plasma to be returned to the animal. Column flow rate was measured prior to reconnection and the time necessary to process 1 plasma volume (30 ml/kg) calculated. After processing 1 plasma volume the peristaltic pump was stopped and the arterial line disconnected from the shunt. The extracorporeal circuit was then flushed with sterile saline to return the contents to the animal. Similarly, the column was flushed with 25 ml of sterile saline returning unbound column contents to the animal. The venous line was then disconnected from the animal and arterial and venous shunt cannulae were reconnected behind the ear. After a brief period (approximately 5 minutes) to allow vascular mixing, a post-treatment blood sample was taken.

The PA-Sepharose column was regenerated between immunoadsorption treatments by desorption with at least 3 column volumes of 0.1 M glycine-HCl (pH 2.8) and then equilibrated with sterile saline.

Treatment animals underwent immunoadsorption procedures five times within 6 days. Each was treated daily for the first 3 days followed by a day with no treatment and then the final two treatments were conducted on the 5th and 6th days.

Measurement of anti-myelin IgG and IgM
Anti-myelin IgG and IgM were quantitated using an ELISA assay which has been previously described in detail.8

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Table 1: Clinical assessment of EAN

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Slight postural abnormality, less lively</td>
</tr>
<tr>
<td>2</td>
<td>Mild but definite paraparesis</td>
</tr>
<tr>
<td>3</td>
<td>Moderate paraparesis, possible front leg weakness</td>
</tr>
<tr>
<td>4</td>
<td>Tetraparesis</td>
</tr>
<tr>
<td>5</td>
<td>Severe tetraparesis, still ambulatory</td>
</tr>
<tr>
<td>6</td>
<td>Severe tetraparesis, non-ambulatory, respiratory difficulty, possible incontinence</td>
</tr>
<tr>
<td>7</td>
<td>Moribund or dead</td>
</tr>
</tbody>
</table>
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**Measurement of biochemical and haematological parameters**

Biochemical analyses were conducted on an American Monitor Parallel autoanalyzer and haematological analyses were conducted using a Technicon H1 blood analyser.

**Results**

**Clinical features**

Out of a total of 20 animals inoculated with myelin, 17 animals were allocated into either control or treatment groups. In these animals onset of neurological signs of EAN occurred between days 12 and 16 post-inoculation. Three animals showed an EAN disease of late onset, one on day 23 and two on day 40 post-inoculation and were not included in the study. After pairing and random allocation, eight animals were assigned to the treatment group and nine to the control group. Mean clinical score at allocation for the control group was 2.56, SD 0.53 and for the treatment group 2.57, SD 0.53. One treatment animal died between the first and second immunoadsorption procedures from catastrophic shunt uncoupling and was not included in the final study.

Clinical data are depicted in fig 2. Data are displayed as arithmetic changes in clinical scores from the scores at allocation. Control animals showed a severe progressive neuropathy with one animal becoming moribund on day 44 post inoculation and two showing severe tetraparesis with respiratory difficulty on days 47 and 55 post-inoculation. Animals becoming moribund were immediately killed. Animals attaining a score of 6, that is, severe quadripareisis and respiratory difficulty (with possible incontinence) that did not show improvement after 2 days were killed and included in the data as attaining a score of 7.

In contrast to the progressive disease seen in the control group, treatment animals showed significant clinical remission as a result of IgG immunoadsorption. Statistical analysis of data taken directly after the course of treatments (days 7 and 8 post-allocation) using 2-tailed Mann-Whitney U tests showed significant differences between the two groups (p < 0.001; p < 0.05 respectively). Remission was transient and most animals showed a relapse by days 9 to 11 post-allocation.

**Blood biochemistry and haematology**

Changes in blood composition from a single immunoadsorption are shown in table 2. Percentage changes in haematological parameters were calculated using blood samples taken from five animals before and directly after the first immunoadsorption procedure. There was no change in plasma solutes, haematocrits or erythrocyte counts. The lack of effect upon haematocrits reflected minimal alterations in plasma volume. However, immunoadsorption did result in significant decreases in albumin and total protein concentrations and white cell counts. Changes in white cell counts have been previously reported in other forms of extracorporeal therapy such as haemodialysis.10 Total protein concentration was reduced by approximately 20% due mainly to IgG removal. The slight but significant decrease in albumin concentration associated with the extracorporeal procedures was due to haemodilution resulting from the return of blood in the venous line with saline and to a lesser extent nonspecific protein binding within the gel matrix.

**Plasma anti-myelin IgG and IgM**

Anti-myelin IgG and IgM levels were assessed in both groups and are depicted in figure 3. Antibody levels are displayed normalised to a pre-treatment level of 100%. Anti-myelin IgG levels rose steadily in the control group throughout the period of observation. In the treatment group, each IgG immunoadsorption decreased circulating anti-myelin IgG concentrations by 55–65%. Rapid post-treatment antibody rebound was observed after each procedure so that levels were re-established within 24 hours. However, the course of immunoadsorption treatments held anti-myelin IgG concentrations at pre-treatment levels in the treatment group so that at day seven post-allocation there were

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**Table 2 Changes in blood components after a single immunoadsorption**

<table>
<thead>
<tr>
<th>Component</th>
<th>% Change (Mean SD, n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na, K, Cl, Ca, PO₄</td>
<td>no change</td>
</tr>
<tr>
<td>Albumin</td>
<td>-13.79, 6.42 p &lt; 0.05</td>
</tr>
<tr>
<td>Total protein</td>
<td>-19.08, 7.45 p &lt; 0.01</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>no change</td>
</tr>
<tr>
<td>Red cell count</td>
<td>no change</td>
</tr>
<tr>
<td>White cell count</td>
<td>-47.58, 11.11 p &lt; 0.01</td>
</tr>
</tbody>
</table>

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![Fig 2 Mean changes in clinical score after allocation for the control group (open circles, n = 9) and treatment group (closed circles, n = 7). Bar indicates period of treatment. Significant differences were determined by two-sided Mann-Whitney U-tests; p < 0.001***; p < 0.05*.](image-url)
significant differences in anti-myelin IgG concentrations between control and treatment groups (2-tailed Student’s t test, p < 0.001). Anti-myelin IgG levels quickly increased after cessation of treatment so that two days later, that is, day nine post-allocation there was no significant difference in levels between groups.

Although rapid antibody rebound was observed, there was no evidence of antibody overshoot above control levels as a result of transient removal. The level in the treatment group was still identical to the control level when last measured at day 20 post-allocation.

Anti-myelin IgM levels are also shown in figure 3 normalised to a pre-treatment level of 100%. In the control group, levels were constant throughout the period of observation. In the treatment group, anti-myelin IgM levels appeared to decrease slowly during treatment although comparisons of pre- and post-immunoadsorption concentrations showed no differences and suggest no significant removal via specific binding to protein A. The slight decreases in anti-myelin IgM concentrations could be due to some entrainment in the PA-Sepharose column. Although after the course of immunoadsorption procedures anti-myelin IgM levels appeared to increase above control levels, statistical analysis using 2-tailed Student’s t test showed no significant differences.

Discussion

The results of this study demonstrate that a short intensive course of IgG immunoadsorption removing 55–65% of circulating IgG is sufficient to induce clinical remission during the early disease phase of EAN. The observed benefit in this study complements that previously seen in EAN with the use of membrane PE. The significant but temporary nature of remission is undoubtedly due to the severe chronic progressive type of disease of which the clinical, electrophysiological and histological features have been well documented. In both therapeutic studies assessment of clinical disability directly after the course of treatments showed significant clinical benefit. This was followed by rapid relapse within one week so that animals receiving a single course of treatment were in the long term clinically indistinguishable from controls. The situation is therefore similar to that in CIDP in man where the need for repeated exchanges to maintain benefit is well recognised, although in most cases the period between exchanges is somewhat longer.

The early disease phase in EAN in these animals is characterised by weight loss and mild to moderate paraparesis. At this stage detectable levels of anti-myelin IgG are present and show gradual increases during disease progression. High levels of anti-myelin IgM can also be detected in some but not all animals. The concomitant increases in clinical disability with increases in anti-myelin IgG combined with the ability of IgG removal alone to mimic the clinical remission observed in EAN with membrane PE and centrifugation PE suggest a pathogenic role for IgG antibodies. Intraneural transfer experiments involving whole EAN serum have demonstrated humoral factors which cause demyelination and conduction failure but although these experiments may detect the presence of pathogenic humoral factors, they fail to determine their nature. Anti-myelin activity of whole rabbit EAN serum has been demonstrated in organotypic PNS cultures to be complement dependent suggesting antibody involvement. Rat intraneural injection studies have clearly shown advanced myelin and Schwann cell damage with rabbit EAN serum well before a macrophage presence.

Intraneural injection of GBS serum into rat sciatic nerves also induces demyelination although the severity of effect is often less than with EAN serum. Recent detailed sequential morphometric studies confirm the ability of GBS serum to demyelinate peri-
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