risk phenotype for MOGAD, although one was diagnosed with a likely parainfectious myelitis and another as transverse myelitis associated with Sjogren's syndrome.

Conclusion A CSF-restricted MOG-IgG profile is rare, with over half of these patients having an alternate non-MOGAD diagnosis confirmed. We strongly recommend the use of serum as the biospecimen of preference in testing for MOG IgG. We additionally urge caution in the interpretation of CSF-restricted MOG-IgG in unselected patients who do not have clinical, radiological or ancillary investigations consistent with MOGAD.

3 MANAGING DISEASE ACTIVITY DURING TREATMENT WITH NATALIZUMAB IN RELAPSING-REMITTING MULTIPLE SCLEROSIS

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10.1136/bmjno-2024-ANZAN.3

Background/Objectives Failure of natalizumab to control disease activity in multiple sclerosis (MS) is an uncommon event. It is currently unknown whether switching to an alternative high-efficacy disease-modifying therapy offers any benefits over persevering with natalizumab.

Methods Patients from the MSBase cohort suffering failure of natalizumab therapy, defined as breakthrough relapses or $\geq$3 new or gadolinium-enhancing lesions on MRI, were identified. Multivariable Andersen-Gill cumulative hazard models were used to analyse the associations of several variables with the risk of further relapses following natalizumab failure. Subsequent treatment decision was included as a time-dependent exposure. Secondary analyses evaluated the effect of treatment decisions on the risk of subsequent new MRI activity, expanded disability status scale (EDSS) worsening, and disease-activity free survival.

Results 1,131 natalizumab-treated patients fulfilled the inclusion criteria. Of these, 85 de-escalated treatment, 39 switched to anti-CD20 therapy, and 28 switched to alemtuzumab. Following natalizumab failure, switch to an anti-CD20 therapy was associated with a significantly lower risk of relapse (HR=0.51, 95%CI=0.29–0.88) compared to continuing natalizumab. Treatment de-escalation, or cessation, were associated with an increased risk of subsequent relapses (HR=1.40, 95% CI=1.10–1.79; HR=1.89, 95%CI=1.09–3.28, respectively). We did not find any evidence of a difference for switching to alemtuzumab, or outcomes studies in the secondary analyses.

Conclusion We have shown that following natalizumab failure, switching to B-cell depleting agents (ocrelizumab and rituximab), but not alemtuzumab, is associated with a clinically meaningful reduction in the risk of relapses in comparison to continuing natalizumab therapy. Clinicians should consider B-cell depletion following natalizumab failure where appropriate.

4 HYPOGAMMAGLOBULINEMIA AND INFECTION RISK IN MYOTONIC DYSTROPHY TYPE 1

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10.1136/bmjno-2024-ANZAN.4

Background/Objectives Hypogammaglobulinemia is a common yet under-recognised feature of myotonic dystrophy type 1 (DM1). The aims of our study were to assess whether low immunoglobulin G (IgG) levels are associated with an increased risk of infection in DM1 patients and to ascertain the association between immunoglobulin levels and cytosine-thymine-guanine (CTG) repeat length in the *DMPK* gene.

Methods We conducted a single-centre, retrospective cross-sectional study of 65 adult patients with DM1 to determine the frequency of IgG deficiency, the association with CTG repeat expansion size, and the infection risk profile.

Results Forty one percent (41%) of DM1 patients had IgG deficiency despite normal lymphocyte, IgA, IgM, and albumin levels. There was an association between CTG repeat expansion size and the degree of IgG deficiency (F = 6.3, p-value 0.020). There was no association between IgG deficiency and frequency of infection in this group (p-value 0.428). Most infections were aspiration pneumonia with evidence of oropharyngeal dysphagia on swallow assessment.

Discussion IgG deficiency is a frequent occurrence in DM1 patients and is associated with increasing CTG repeat expansion size. Reduced IgG levels were not associated with increased infection frequency. Routine monitoring of IgG levels and immunoglobulin supplementation in DM1 patients may not be necessary unless there is an unexplained history of recurrent infections.

5 NEUROPROTECTIVE EFFECTS OF METFORMIN IN DIABETIC PERIPHERAL NEUROPATHY

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10.1136/bmjno-2024-ANZAN.5

Background Diabetic peripheral neuropathy (DPN) affects around 50% of the 500 million persons with type 2 diabetes worldwide and is considered disabling and irreversible. Currently no treatment for DPN is available and previous studies have failed to demonstrate benefits for strict glycaemic control. Metformin is an oral biguanide known to have several metabolic and cellular actions. The present study was undertaken to assess the effect of metformin on peripheral neuropathy outcomes in type 2 diabetes.

Methods 69 type 2 diabetes participants receiving metformin were recruited and had clinical assessment, peripheral nerve ultrasound, nerve conduction studies and nerve excitability studies performed. 318 participants who were not on metformin were also concurrently recruited and assessed, and 69 were matched to the metformin participants for age, sex,