REVIEW ARTICLE

TITLE
Neuropsychological outcomes in paediatric MOGAD: clinical practice and future research.

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Abstract

MOG-ab associated disorders (MOGAD) are a recently characterised demyelinating condition that affects children and adults. Due to its recent discovery, the effect that MOGAD has on children's cognitive abilities is only just being understood. Some children with MOGAD, particularly those with Acute Disseminated Encephalomyelitis and Neuromyelitis Optica Spectrum Disorder phenotypes, experience impairment on tests of intellect, long-term memory, working memory and processing speed. Other young people with MOGAD have normal range cognitive performance. Some studies suggest cognitive difficulties only emerge over time and are linked to relapses. Brain lesions do not have a simple relationship to cognitive change and are found in those with and without cognitive impairment. All studies reviewed are limited by small sample size but suggest that children with MOGAD are at risk of cognitive impairment. Future research is required with sufficient power to determine the cognitive profile of a large sample of children with MOGAD. The role of disease variables on cognitive outcome should also be investigated. Paediatric patients with MOGAD require regular neuropsychological monitoring using screening and comprehensive neuropsychological assessment. This will allow the correct supports and neurorehabilitation strategies to be offered to these young people and guard against academic and occupational underachievement.

Key words: MOGAD, paediatric, cognition, neuroimaging, neuropsychology

Myelin oligodendrocyte glycoprotein (MOG) is found on the myelin sheath of the central nervous system (Bernard et al., 1997). Its exact function is unknown, but it is thought to be important in the myelination process (Johns & Bernard, 1999; Reindl & Waters, 2019). Recent technical advancements in cell-based assays revealed that antibodies against MOG (MOG-abs) are found in a group of patients with a specific clinical phenotype, separate from other demyelinating conditions(Tanaka & Tanaka, 2014; Waters et al., 2015). MOG-abs associated disorders (MOGAD) are now seen as a disease entity, requiring specific medical management and treatment (Jarius et al., 2018, for review see Wynford-Thomas et al., 2019).

Background

Incidence MOGAD in children is rare and seen in 0.31 per 100.000 children (de Mol et al., 2020). The clinical phenotype in MOGAD is wide and affects both adult and paediatric patients (Banwell et al., 2023). In children MOGAD presents as a demyelinating syndrome which can be monophasic or relapsing (de Mol et al., 2020). The E.U. paediatric MOG consortium consensus(Bruijstens, Lechner, et al., 2020), suggested four typical MOGAD clinical phenotypes in the paediatric population: (1) under 10 year olds with acute disseminated encephalomyelitis (ADEM), characterised by neurological deficits, encephalitis and brain MRI abnormalities in the acute phase (3 month post onset) (Krupp et al., 2013), (2) adolescents with Optic Neuritis (ON) with inflammation of the optic nerve(s) leading to unilateral or bilateral visual problems, typically including visual loss, reduced colour vision and reduced visual fields (Baumann et al., 2018; Hennes, 2017), (3) adolescents with transverse myelitis (TM), resulting in motor, sensory and bladder and bowel dysfunction (Hintzen et al., 2016), (4) neuromyelitis optica spectrum disorder (NMOSD) like phenotype characterized by recurrent ON or TM (Lechner et al., 2016). These four typical phenotypes of MOGAD have generally good outcomes with visual and motor disability improving, although not always back to baseline, and bladder and bowel symptoms often persisting (Bruijstens, Breu, et al., 2020). Around 30-50% of MOGAD paediatric patients have a relapsing disease course with all clinical phenotypes at risk of relapse (Bruijstens, Lechner, et al., 2020;

Hennes, 2017)**.** Research is mixed, but those who experience relapse tend to have poorer motor, visual and cognitive outcomes, with the burden of disability increasing with the number of relapses (Ramanathan et al., 2018). Treatment in the acute phase is high dose corticosteroid therapy, IVIG and plasma exchange, with long term immunotherapy only initiated if there is a second MOGAD attack (Bruijstens, Wendel, et al., 2020).

Brain Lesions in paediatric MOGAD

As the clinical presentation is heterogenous in MOGAD, the location and extent of brain lesions, or grey and white matter changes vary across each phenotype(Baumann et al., 2020). Those with ON and TM phenotypes can have little evidence of any brain lesions in tautological MRI scans, whilst children with ADEM MOGAD phenotype can have large and multifocal lesions affecting the white and grey matter in cortical, subcortical and the brainstem (Baumann et al., 2018; Hennes, 2017).

Whilst, there can be little evidence of brain changes in some individuals with MOGAD, two recent studies showed that brain growth is disrupted in children with MOGAD across phenotypes and even in those with a monophasic disease course (Bartels et al., 2023; Fadda et al., 2023). Fadda and colleagues (2023) showed reduction in grey matter growth in the thalamus, caudate and globus pallidus volumes of children with MOGAD, particularly in the first year post onset and continuing up to 16 years later (Fadda et al., 2023). They interpreted this to show that the first attack has a larger impact on brain development than later relapses. Bartels and colleagues (Bartels et al., 2023) also reveal that children with the ADEM phenotype of MOGAD had reduced brain growth over time. This aligns with adult research that suggests that those with MOGAD experience attack-mediated brain insults and that neurological deficits, including cognitive, are highly linked to attacks or relapses (Messina et al., 2022).

School performance in paediatric MOGAD

In this relatively newly discovered condition, initial outcome research relied upon the subjective report of school performance as an indirect measure of cognitive impairment. A large cohort study in France found that 26% needed school support performance (i.e., grades repeated, left school early, attended specialist schools) at the latest follow up (Deiva et al., 2020). Using a univariate analysis, they showed that ADEM phenotype, younger age of onset and deep grey matter lesions were associated with increased school support needed (Deiva et al., 2020). Hacohen and colleagues (2018), found that those with multiphasic disseminated encephalomyelitis or ADEM and then relapsing ON presentation, experienced greater need for specialist school placement or increased support within a mainstream setting(Hacohen et al., 2018).

More recently, Eyre and colleagues (under review) moved away from subjective report and were able to attain nationally standardised assessments from the Department for Education of young people with MOGAD and controls. Those with MOGAD had worse academic performance and increased school absence after clinical onset. They suggested that that school absence was not the only driver of poor academic performance, as this normalised within two years, and cognitive deficits likely contributed (Eyre et al., 2024). Overall, these studies that used school performance as an indirect measure of cognition, suggest that factors associated with cognitive impairment include younger age at onset, deep brain lesions to the thalamus or putamen and clinical phenotypes of ADEM, and relapsing MDEM and ADEM-ON.

Neuropsychological outcomes in Paediatric MOGAD

While school performance is likely to be highly correlated with cognitive difficulties, other factors apart from cognitive impairment can drive school-based difficulties (i.e., mental health issues, physical disability, fatigue). In the last four years studies that chart the neuropsychological function of children with MOGAD have emerged. By far the largest, Fabri and colleagues (2022) assessed 12 children with MOGAD as part of a prospective study in children with demyelinating conditions. Using a computerised standardized battery, the Penn Computerized Neurocognitive Battery, they assessed executive

function, episodic memory, complex cognition (language/non-verbal/spatial reasoning) and social cognition in those with MOGAD, paediatric onset multiple sclerosis (POMS) and controls (Fabri et al., 2022). The phenotype of the MOGAD within their group of 12 was highly variable (ADEM=1, ADEM-On=1, ADEM-TM=1, monofocal TM=3, monofocal ON=3, polyfocal TM=1, polyfocal TM and ON=1 and other=1) but all children had a relapsing course. Results showed that those with relapsing MOGAD had relatively mild cognitive deficits in comparison to controls. They had significantly poorer performance on the verbal reasoning subtest and their response times were slower than controls. This was contrasted with the POMS group who had significant impairment across multiple cognitive domains in comparison to controls and those with MOGAD (Fabri et al., 2022). They concluded that MOGAD is associated with less severe cognitive dysfunction than POMS, even in relapsing disease courses (Fabri et al., 2022).

This study may lack sensitivity due to it utilising an automated cognitive battery that is not equivalent to neuropsychological tests used in the clinic. The largest study published so far using standard neuropsychological tests is a case series of seven MOGAD patients aged 3-15 years (Tan et al., 2021). Again, the clinical phenotype was heterogeneous (ADEM, NMOSD, ON and MS) and four were relapsing and three monophasic. Results showed age-appropriate neuropsychological performance with 5/7 (71%) with normal range intellect, processing speed, working memory, episodic memory and attention. Interestingly, of these children with normal cognitive functioning all had brain lesions on MRI scan and some of these worsened over subsequent scans (Tan et al., 2021). Two patients with MS and NMSOD phenotypes respectively, showed neuropsychological impairment (scores below 80) in attention, executive functioning, processing speed and fine-motor speed. The authors attributed this to early age of onset and long disease course in one patient, and history of dyslexia in another. They went on to conclude in line with Fabri and colleagues (2022), that children with MOGAD experience a less severe clinical course, in comparison to POMS, including relatively intact cognitive functioning (Tan et al., 2021).

This idea that MOGAD has a relatively mild impact on cognitive functioning has been questioned by several single case studies (Kornbluh et al., 2020; Pandit et al., 2017; Tan et al., 2021). Pandit and colleagues (2017) were the first to describe a 17-year-old MOGAD patient who experienced a relapsing NMOSD. He had an aggressive disease course and experienced two attacks, presenting with lethargy and headaches. Brain MRI and neurocognitive tests 3 months after the second attack suggested ongoing cognitive impairment and brain lesions to the bilateral temporal and frontal regions with overall volume loss (Pandit et al., 2017).

Very few studies have looked at cognitive development in children with MOGAD longitudinally. One case study conducted serial neuropsychology assessments to determine cognitive trajectory of an individual with relapsing MOGAD with an ADEM phenotype (Kornbluh et al., 2020). The patient was assessed after the first episode and then a year later during neurorehabilitation after the second episode. Interestingly, in comparison to the findings so far, much more significant cognitive deficits were seen in this patient with below the normal range (<80) performance on verbal IQ, non-verbal IQ, memory, word reading and fine motor speed at both assessments. Most significant impairment was seen in fine motor abilities and word reading, which was in the very low (<70) range at both assessments. Using reliable change index calculations, they showed from assessment 1 to 2, there was a significant improvement in nonverbal reasoning, potentially due to practice effects, and significant decline in right hand fine motor speed (Kornbluh et al., 2020) . The cognitive impairment in this patient were reportedly linked to significant brain changes in the left and right frontal lobes after their first episode and by the second assessment widespread lesions across the cortex including frontal lobe, thalamus and putamen (Kornbluh et al., 2020).

Although not the focus of their investigation, the Tan and colleagues (2020) study included serial assessments of one MOGAD patient (patient 7) two years apart who had a NMOSD phenotype. They did not calculate reliable change scores, but performance on the verbal episodic memory and visual motor tasks moved from the average range at first assessment to the very low range at the

second assessment. This patient had lesions to the brainstem, left hypothalamus, and pons, with worsening lesions to the brainstem at the second assessment (Tan et al., 2021).

Limitations

While these initial studies suggest that some children with MOGAD are at risk of cognitive impairment, the results of these studies could have been biased by several factors. Most crucially the small sample size of all these studies means that individual participant attributes may be biasing the conclusions made and may not reflect disease factors. It is also possible in the case studies that there is sampling bias as children who are particularly unwell with MOGAD have been written up but not those who have a more benign disease course. Due to the small sample size, the role of MOGAD phenotype, relapses, brain lesions, age of onset and medication has not been possible to explore or control. Only two studies attempted to look at cognitive trajectory over time. In children it is crucial to look at the cognitive development and the impact of CNS condition as the brain develops and grows.

Is there a relationship between cognition and brain integrity?

The relationship between number of brain lesions and neurocognitive functioning is clearly complex and further research is needed as discussed below. No paediatric study to date has used neuroimaging techniques beyond radiological report to explore if brain integrity and cognitive outcome might be linked. One study of adults with MOGAD used structural MRI, DTI and resting state-fMRI scans, and found that grey matter volume of the right temporal gyrus and insular correlated with a task of processing speed (the Symbol Digit Modality Test) and a screening assessment of cognition (Montreal Cognitive Assessment Test (Zhuo et al., 2021). Interestingly, the grey matter volume of the left hippocampus and parahippocampal gyrus positively correlated with the California Verbal Learning Test, a test of verbal episodic memory. This study also revealed widespread grey matter reduction in frontal, temporal, insula, thalamus and hippocampus (Zhuo et al., 2021). This study therefore shows that adults with MOGAD grey matter changes are associated with corresponding neuropsychological

test performance and would suggest that brain changes may drive cognitive impairment in adults with MOGAD.

Clinical Implications

As we have seen, children diagnosed with MOGAD are at risk of cognitive impairment, alongside physical and sensory disability. Their cognitive outcomes vary and some children with MOGAD experience significant impairment in intellect, episodic memory, working memory and processing speed that worsens over time (Kornbluh et al., 2020); while others experience more subtle or no cognitive concerns (Fabri et al., 2022). In this relatively newly discovered CNS condition, paediatric neuropsychological services will need to have the resources to offer children with MOGAD neuropsychological assessment, support and intervention. Due to the wide range of cognitive abilities seen, a gold standard service for paediatric MOGAD could follow a preventative model of neuropsychological assessment for children at risk of cognitive impairment due to medical illness (Hardy et al., 2017). This would include: (1) universal monitoring or screening of all children with MOGAD diagnosis using caregiver questionnaire, clinical interview and brief computerized screening assessment, (2) targeted assessment using neuropsychological assessment of cognitive areas of need, (3) comprehensive neuropsychological assessment of all cognitive domains (Hardy et al., 2017). This model is responsive and only those who have evidence of deficits or reported difficulties at the universal monitoring phase would progress to targeted assessment (Hardy et al, 2017). Crucially it would enable all children with MOGAD to be screened and cognitive difficulties to be proactively and quickly identified in the clinic. Computerised screening batteries are often not utilised in clinical settings, but they have less issues with test/retest validity and do not need to be conducted by a qualified Neuropsychologist. Repeat monitoring would also enable the individual's cognitive trajectory to be looked at over time so that changes in the individual's abilities can be monitored. This would provide richer information for treating medical professionals to track impact of relapses and disease course. Change in cognitive trajectory would also initiate hypothesis driven and comprehensive

neuropsychological assessment to allow for deeper understanding of cognitive deficits. This would enable the appropriate support at home and school to be implemented, alongside cognitive neurorehabilitation and psychological therapy.

Future Research

Research into the neuropsychological outcomes in children with MOGAD is in its infancy and there are several avenues of research that will help clinicians understand how to best treat these individuals.

1. **Neuropsychological outcomes and the relationship medical variables** – the research reviewed showed widely varying cognitive outcomes in children with MOGAD, with some phenotypes such as ADEM and NMOSD related to impaired cognition, whilst ON and TM were associated with no or only mild cognitive weaknesses (Fabri et al., 2022; Kornbluh et al., 2020; Tan et al., 2021). Future research should aim to look at a large sample of children and young people with MOGAD that has sufficient power. Comprehensive neuropsychological assessment of these children with tests of intellect, long-term memory, working memory, processing speed, attention and executive function would enable a detailed understanding of this cognitive profile of children with MOGAD. Crucially a large sample size is needed so that impact of different medical variables can be understood potentially using regression analyses. Firstly, the role of the MOGAD phenotype on cognitive outcomes should be explored as literature reviewed suggest that some phenotypes are at much greater risk of cognitive impairment. It will also be important to explore if attacks/relapses are associated with worsening cognitive outcomes in children with MOGAD. The role of the age of onset has not been looked at yet in children with MOGAD, although there is a hint that earlier age of onset results in more need for support in school (Deiva et al., 2020). Finally, the possible impact of acute and repeated use of corticosteroids should be explored as they can have modest negative effects on working memory, long-term memory and executive functions for acute and long-term users (for review see Prado & Crowe, 2019).

2. **Longitudinal neuropsychological trajectories** – in children and young people it is important to look at cognitive trajectories over time to understand how CNS demyelinating condition might be impacting their development. In the research reviewed there is suggestion that even those with no current cognitive impairment and monophasic disease course have changes to brain growth over time (Fadda et al., 2023). It will therefore be important to look at long-term cognitive trajectories of children with MOGAD over the developmental period to see if there is evidence of change in an individual's cognitive trajectory. This could be through universal screening suggested above, or more tailored neuropsychological assessment at a few crucial time points in development (i.e., 8 years and 13 years). The impact of medical variables on cognitive trajectory should be investigated, for example, relapse rate may be a crucial factor to explore.
3. **Neuroimaging** –In adults with MOGAD there is a relationship between grey matter loss and cognitive performance (Zhou et al., 2021), whereas in children there is mixed evidence whether brain lesions and cognitive abilities are linked. Neuroimaging studies in paediatric MOGAD are needed that implement structural and DTI to give rise to quantitative measures of grey and white matter in children with MOGAD. This would allow the relationship between grey and white matter brain integrity and cognitive performance in children with MOGAD to be explored and better understood.

Conclusion

MOGAD is a relatively newly characterised demyelinating condition that affects children. The neuropsychological profile of children with MOGAD is only just being understood. Overall, the studies reviewed suggest a varied cognitive outcome, with some MOGAD children experiencing no cognitive deficits or mild ones. However, there are individuals with much more severe disease courses, often associated with NMOSD and ADEM phenotypes that have significant cognitive impairment that can progress over time. These individuals were seen to have significant white and grey matter brain lesions

on MRI scan. Although conversely there is also evidence of individuals with brain lesions and no cognitive impairment. All studies reviewed were limited by small sample size, but suggest that children with MOGAD are at risk of cognitive impairment and require ongoing neuropsychological assessment, monitoring and treatment. Future research is needed to fully understand the cognitive profile of children with MOGAD and explore how disease factors influence cognitive development over time.

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