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Update on NMOSD
and MOGAD

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Clinical features of NMOSD and MOGAD: a single center study from South Brazil

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Introduction

Neuromyelitis Optica Spectrum Disorder (NMOSD) and Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease (MOGAD) are distinct autoimmune disorders affecting the central nervous system. Their epidemiological profiles, initial clinical presentations, and treatment approaches influence patient care.

Methods

This study retrospectively analyzed electronic medical records from 2003 to 2024 and compared the clinical characteristics of AQP4+, seronegative NMOSD and MOGAD from a tertiary hospital in the South of Brazil.

Results

We included 26 patients: 13 with NMOSD (12 AQP4+ and 1 seronegative) and 13 with MOGAD. NMOSD patients were predominantly white (76.9%), female (84.61%), and had a mean onset age of 41.84 years (range 20-65). Their most common initial presentation was transverse myelitis -TM (69.3%) and unilateral optic neuritis - ON (30.7%), with a median of 5.15 relapses and an annualized relapse rate (ARR) of 0.386. Recovery after the first attack was complete only in 7.7% at six months. In contrast, MOGAD had an onset age of 31.5 years (range 4-70) with a more balanced gender distribution (61.5% female). Bilateral ON was most common (46.15%), followed by unilateral ON (30.76%). Less frequent initial presentations included ADEM (15.38%), short TM (7.69%), and longitudinally extensive TM (7.69%). MOGAD had fewer relapses, averaging 1.53/patient, with an ARR of 0.252 and good recovery in 76.9%. Relapsing disease was present in 92.3% of NMOSD and 33.3% of MOGAD. Other autoimmune pathologies were observed in 25% of AQP4+ NMOSD, while absent in the other groups. The management of acute episodes was similar across all groups. Long-term therapy was started in 100% of patients with NMOSD and 69.23% of those with MOGAD. In both groups, azathioprine was the most commonly relapsing prevention therapy chosen as first line.

Conclusion

Our findings underscore clinically significant differences between NMOSD and MOGAD, requiring identification of clinical phenotypes and treatment responses for each group.

References

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