Memantine Treatment in Sickle Cell Disease: A One-Year Study of Its Effects on Cognitive Functions and Neural Processing

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Introduction

Sickle Cell Disease (SCD) is the most frequent cause of hereditary hemolytic anemia worldwide. There has been a growing interest in researching the cognitive and neural deficits characteristic of patients with this disease, which may have profound impacts on quality of life, daily living, academic achievements, employment, and social functioning. This study evaluates the neurocognitive and electrophysiological effects of one-year Memantine treatment — which acts as an antagonist for the N-methyl-D-aspartate (NMDA) receptor subtype of the glutamate receptor — in adolescents and young adults suffering from SCD, hypothesizing enhancements in cognitive functions and neural processing. Fourteen participants with SCD underwent cognitive and neural assessments using the Wechsler Intelligence Scale and a computerized task-switching paradigm with concurrent brain event-related potential (ERP) recordings. Assessments were conducted before (T1) and after (T2) one year of Memantine treatment, focusing on processing speed, working memory, attention, and executive function. ERP measurements targeted changes in brain response patterns during task switching.

Results

Memantine treatment, which was well tolerated, led to significant improvements in cognitive performance, particularly in processing speed, as demonstrated by the Digit–Symbol Coding and Symbol Search tests (Fig 1). These results suggest enhanced visuospatial and graphomotor speed, working memory, and attention. The task-switching test showed reduced error rates post-treatment (Fig 2), indicative of decreased cognitive load and improved executive control.

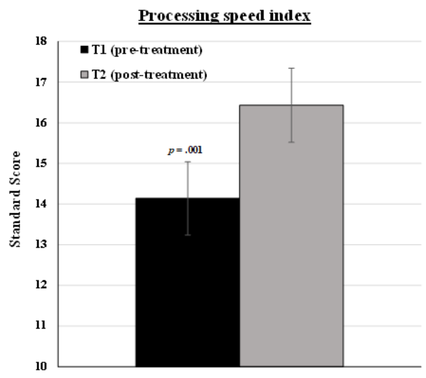

Fig 1. Mean Processing Speed Index (PSI) standard scores at T1 and T2.

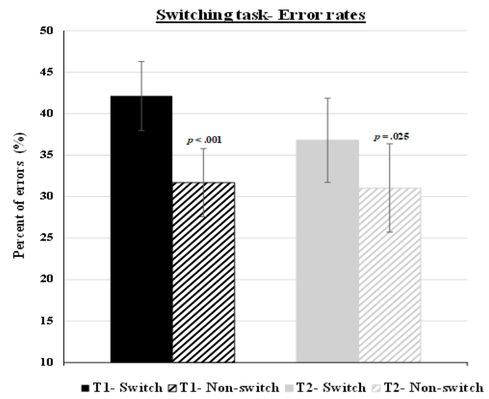

Fig 2. Mean error rates in the switching task at T1 and T2 in the Switch and Non-switch conditions.

Electrophysiologically, alterations in P1 and P3 amplitudes at frontal and parietal scalp locations post-treatment indicated more efficient perceptual and cognitive neural processing in tasks requiring cognitive flexibility (Fig 3+4+5+6).

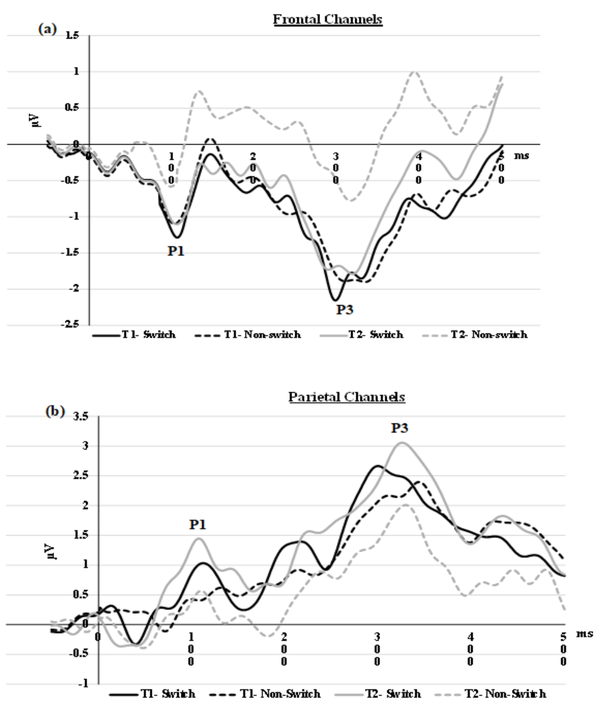

Fig 3. Grand averaged ERPs for Switch and Non-switch conditions at T1 and T2 at frontal (a) and parietal (b) channels.

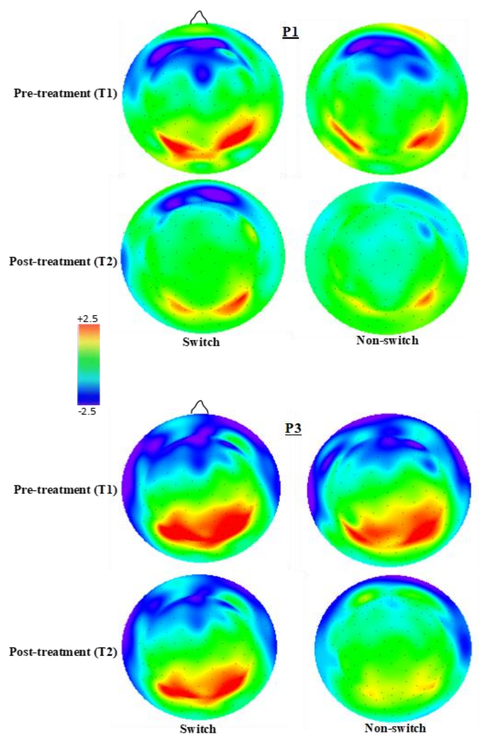

Fig 4. Voltage distribution of P1 and P3 under switch and non-switch task conditions, represented as scalp maps at T1 and T2.

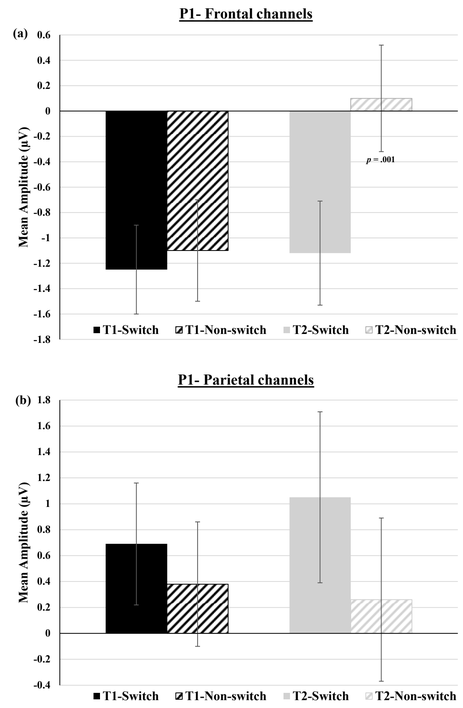

Fig 5. Mean amplitudes of P1 for T1 and T2 in Switch and Non-switch conditions at frontal (a) and parietal (b) channels.

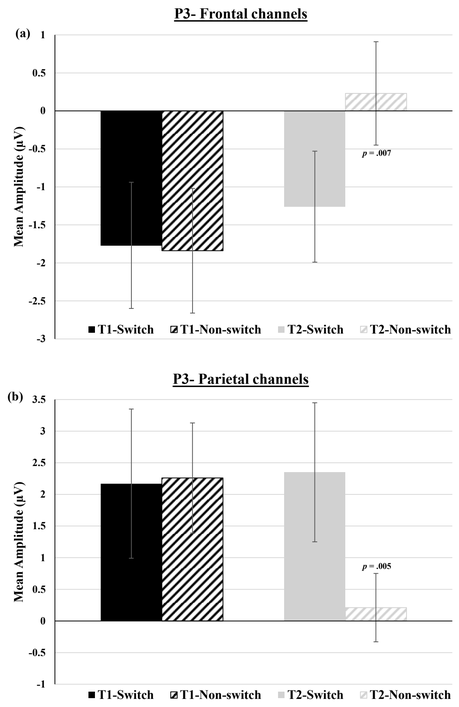

Fig 6. Mean amplitudes of P3 for T1 and T2 in Switch and Non-switch conditions at frontal (a) and parietal (b) channels.

Conclusions

This study demonstrates cognitive and neural improvements following one year of Memantine treatment, underscoring its potential role in managing the neurocognitive deficits associated with SCD.

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