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Introduction: Negative symptoms, a core feature of chronic schizophrenia, are notoriously resistant to treatment. New therapeutic strategies are proposed based on the hypothesis of a relation between glutamatergic neurotransmission and schizophrenia.

Method: Case report.

Case presentation: Male patient, 51 years old, suffering from schizophrenia since the age of 22. He was stabilized on risperidone long-acting injection -37.5mg/2 weeks- and biperiden 4mg/day (due to persistent extrapyramidal tremor). His prominent symptoms were the negative ones: Affective flattening, avolition, asociality and poverty of speech. Memantine 10mg/day was added. One and a half month later the patient spontaneously admitted to spend more time with his relatives enjoying their company more than in the past. He was assessed with the Scale for the Assessment of Negative Symptoms (=96), the Scale for the Assessment of Positive Symptoms (=3), the Mini Mental State Examination (=26) and the Calgary Depression for Schizophrenia Scale (=2). Memantine was increased to 20mg/day. Two months later, affective flattening and asociality continued to improve; avolition and poverty of speech remained generally unchanged (SANS: 76, SAPS: 1, MMSE: 26, CDSS: 1). No additional side-effects were noted.

Conclusion: Memantine blocks the excessive influx of calcium through the activated N-methyl-D-Aspartate (NMDA) receptors which are part of the glutamatergic system. Neuroprotection of the glutamate neurons could hypothetically ameliorate several symptoms of schizophrenia that are thought to be mediated by them. In the literature the findings regarding its addition to atypical antipsychotics are conflicting, however it is proposed as potentially helpful for patients with severe residual psychopathology.