We read with interest the excellent article by Rascol and colleagues on the use of amantadine in Parkinson's disease and other movement disorders.[1] One movement disorder where amantadine has shown some utility that the authors did not mention is catatonia. Catatonia is a neuropsychiatric syndrome that occurs in association with psychiatric disorders such as schizophrenia and bipolar disorder but also various neurological disorders, including autoimmune encephalitis. Characteristic clinical signs include stupor, mutism, posturing, catalepsy, echophenomena, mannerisms, stereotypies and negativism. Catatonia has been associated with significant morbidity with complications including pneumonia, acute kidney injury, pulmonary embolus.

Standard treatment for catatonia consists of benzodiazepines and electroconvulsive therapy (ECT). Given that amantadine is a treatment for various antipsychotic-induced movement disorders, it is perhaps unsurprising that the first study of amantadine for catatonia showed benefit in the context of antipsychotic-induced catatonia. [2]

Since this time, amantadine has also demonstrated effectiveness in catatonia of various other aetiologies, sometimes leading to resolution of motor signs within hours. [3] It is particularly useful where benzodiazepines have been unsuccessful and is now considered a second-line therapy where electroconvulsive therapy is not available. [4]

The use of amantadine in catatonia highlights its similarities with Parkinson's disease. Both disorders feature hypokinesia and rigidity, which may reflect the involvement of similar basal ganglia circuity, despite very different aetiological mechanisms. [5] Likewise, Rascol and colleagues highlight the use of amantadine for minimally conscious states, which may closely resemble catatonia phenomenologically but have a very different pathophysiology.

We declare no competing interests.

**References:**


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