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A case of dementia in a middle aged man following three decades of amphetamine dependence

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A 48 year old man presented to the outpatient clinic reporting memory decline interfering with his functioning. He would lose his place during tasks; forget appointments, and the names and faces of new and old acquaintances. Symptom commencement was unclear, but had been present for at least two years, first noticed when he ceased methamphetamine use.

The patient began smoking amphetamine from age 16. He soon moved to injecting amphetamine and later methamphetamine on a daily basis up to 3 or 4 times a day, for 30 years before ceasing due to incarceration in prison for drug related offences. He had not used psychostimulants for over 2 years. He was also a regular smoker of cannabis. The patient had a history of head injuries from motor vehicle accidents, fights and sporting injuries.

He suffered from hemochromatosis, which was well managed by regular venepuncture.

On examination, the patient was alert and orientated, and was broadly able to give a coherent history, but lost his place during long anecdotes. On frontal lobe testing he performed poorly on alternating hand sequences, the 'no-go' test and proverb interpretation. There were no frontal release signs. His neurological examination was unremarkable. On further cognitive testing, he scored 75/100 on the Addenbrooke's Cognitive Examination (ACE-R), with significant impairments in memory, verbal fluency and minor impairment in attention and orientation. Other domains were unremarkable. Blood tests including iron studies, metabolic parameters, syphilis serology, HIV and hepatitis screens were negative. CT and MRI of the brain were unremarkable.

Methamphetamine (MA) has been shown to be neurotoxic to both dopaminergic and serotonergic neurons in both human, but in particular animal studies. Studies have shown long term neuronal damage in abstinent MA users with significant reductions in basal ganglia and frontal white matter. [1,2] Until recently, the prevailing view was that methamphetamine use, even when the user achieved abstinence, caused a broad range of cognitive deficits, evidenced by deficits on cognitive testing and observed on neuroimaging studies.[3] However a critical review by Hart et al [4] attempted to dispel this view by examining the significance of cognitive deficits and neuroimagining changes in users, suggesting the clinical significance of these findings may be limited because the cognitive functioning had overwhelmingly fallen within the normal range when compared with normative data.

Despite neuroanatomical research highlighting selective damage to the medial temporal lobe including cingulate-limbic cortex, the functional cognitive deficits experienced by abstinent chronic MA users has not been well described [3,4] suggest that although cognitive impairment in users may be statistically significant, its clinical relevance, or every day import, is rarely specified. In the case of our patient, his cognitive impairment was clinically significant, functionally impairing and of sufficient degree to merit a diagnosis of dementia in view of the persistence of cognitive symptoms despite abstinence. It must be noted that ICD 10 diagnostic criteria do not adequately account for substance related dementia, but rather an amnesic syndrome whilst the DSM 5 provides for a Substance/Medication-induced Major or Mild Neurocognitive Disorder but provides specifiers for alcohol, sedatives and inhalants only, leaving psychostimulants being coded as "other". Differential diagnoses include other neurocognitive disorders such as Traumatic Brain Injury, Alzheimer's type or Frontotemporal Dementia, however these disorders neither account for the temporal nature of the cognitive decline, whilst the deficits on neurocognitive testing appear to be better accounted for by an MA induced process.

With worrying trends in the use of MA in Australia, notably an increase in the frequency of use in those who have used recently, and the increasing age of regular users, the longer term cognitive and functional impact on users, even in abstinence, may precipitate an increasing burden on already stretched health resources. The implications are serious, for users, their families, the health system, and for clinical research. Further research and clinical discussion is required to determine if we are about to experience a wave of MA induced neurocognitive disorders as the impact and burden of MA use broadens.

References

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