he was 18 was admitted for the first time in our unit for alcoholic detoxification. He had been medicated with 45 mg od of methadone for his opiate dependence. At admission he was started on tramadol 300 mg od, diazepam 20 mg od and thiamine 200 mg od. Five days later he presented with ataxia, mental confusion, incoherent speech and asterixis. Lab investigations showed raised blood ammonia (182 mcg/dl, r.v – 27-102), liver enzymes (AST – 123 U/L, r.v – 10-44; ALT - 66 U/L, r.v – 10-34; GGT – 265 U/L, r.v – 11-50), total bilirubin (1,46 mg/dil, r.v<1,0) and unconjugated bilirubin (1,06 mg/dl, r.v – 0-0,02), normocytic normochromic anaemia, thrombocytopenia (45 x 103/uL, r.v – 150-400) and a reactive VDRL test [with positive titter of FTA Abs (2+) and negative T.P.H.A]. Blood manganese levels were normal. Cytochemical and microbiological cerebrospinal fluid study was normal. Brain MRI showed bilateral and symmetric pallidal T1-weighted signal hyperintensities. Lactulose 45 mL bid and 500 mL/day of a glucose parental solution was added to the above regimen. A gradual clinical improvement occurred along with ammonia, liver enzymes, bilirubin and platelets normalization. Two weeks after admission he was discharged home asymptomatic.

The onset of symptoms five days after the admission could not be explained by a methadone and/or alcohol withdrawal syndrome. Typically these syndromes develop within 72 hours after substance intake discontinuation.[1]

Continuous alcohol intake along with a chronic hepatic condition such as hepatitis C diminishes liver ability to detoxify noxious agents.[2] Moreover, the use of diazepam which is known to be hepatotoxic, the delayed prescription of lactulose and a protein rich diet could have contributed to the increase in blood ammonium and also explain the onset of symptoms delayed up to five days after the admission.

The presence of high levels of ammonium in an adequate clinical context suggests the diagnosis of hepatic encephalopathy.[3] This is true for grade III and IV encephalopathies. However, as ammonia levels do not correlate with the severity of hepatic encephalopathy, nor is there a specific change in hepatic tests for diagnosing it, imaging studies are of value in grade I and II encephalopathies. As in our patient, brain MRI can demonstrate a high resonant globus pallidus supporting the diagnosis.[3]

The type of symptoms, neuroimaging and laboratory findings are highly suggestive of basal ganglia manganese deposition.[3,4,5] Studies have shown that manganese deposition in basal ganglia can result from both portal-systemic shunting and liver dysfunction.[5] Blood manganese levels often yield conflicting results since they do not accurately reflect concentrations of the metal in the brain.[5]

Treatment with lactulose and glucose parental solutions is indicated in these cases.[3]

This case highlights the importance of maintaining a high index of clinical suspicion in alcoholic patients presenting with neuropsychiatric symptoms. These patients can be easily considered as only having a withdrawal syndrome and this can be both misleading and potentially dangerous.

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Use of Autism Diagnostic Interview[1] (ADI-R) in clinical practice

Dear Editor,

The ADI-R is the 'gold standard' research interview to get papers published in international peer reviewed journals. This is satisfactory if you want to define autism in terms of so called narrow autism – an out of date concept of autism – but a definite part of the autism spectrum, but only a part of the autism spectrum who meet ADI-R criteria and ADOS-G[2] (Autism Diagnostic Observation Schedule). For research purposes it is legitimate to define a narrow part of the autism spectrum. This tells us nothing about the prevalence of Autism Spectrum Disorders in the general population or in clinical practice. Indeed using these narrow criteria[3] gives you a prevalence of autism of 25 per 10,000. When you use the broader autism spectrum you get a true rate of autism of 116 per 10,000.[4] I see many parents who come to me in great distress, knowing that their child has autism but having being told that their child did not have autism based on ADI-R. This instrument is not appropriate to making a sole diagnosis of autism in clinical practice. It not uncommonly misses High Functioning Autism. In addition Ventola[5] has shown that the ADI-R was significantly "under diagnosing toddlers".

Even in the research context in many developing countries and indeed I have heard researchers from Australia making the same point the expense of getting trained in these instruments is prohibitive and is inhibiting autism research. It is time to move on from the ADI-R.

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