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Stammering and bipolar affective disorder

Dear Editor,

Stammering is a common disorder affecting 1% of the population.[1] It is characterised by frequent repetition or prolongation of sounds or syllables or words or by frequent hesitations or pauses that disrupt the rhythmic flow of speech. Stammering usually begins in childhood between the ages of three and five, and while as many as one in 20 children stammer at some point, approximately two out of three recover spontaneously.[2] There is 74% overall recovery rate and those who recover usually do so by their early teens. Stammering is more common in men and occurs independent of race, culture, education or socio-economic status.

The cause of stammering is unknown however a positive family history is found in 65% of cases.[3] In monozygotic twins, concordance rates for stammering ranges from 75% to 89%.[4] Some studies have reported structural brain differences in children who stammer,[5] with the cerebellum and basal ganglia being implicated.[6-8] However, studies do not support a physiological cause of stammering and the more common view is that conditions such as shyness and social anxiety result from stammering.[10] One repeated finding is that when people sing they do not stammer which suggests that stammering may occur more when there is ambiguity in what to say and when to say it. During singing there is no such ambiguity as the words are learnt and rehearsed.

Case report

A 30 year old mother of two presented to the Accident & Emergency department 10 days post partum. She described a four day history of elated mood, sleep disturbance, racing thoughts and over activity. She had auditory hallucinations of her dead mother's voice and believed that someone was putting thoughts into her mind. She denied anxious or avoidant personality traits and her general physical health was normal. Routine investigations revealed no biological or haematological abnormalities.

She had developed a persistent developmental stammer in childhood and had no past history of mania. However, her treating psychiatrist noted that she had pressured speech on admission and that she was no longer stammering. She was commenced on antipsychotic medication and her manic symptoms resolved during a 10 day admission. At follow-up in the outpatient department, it was noted that her mood was euthymic and that she did not show evidence of stammer in her speech. She was seen at out-patient appointments over the following two years and her stammer had not returned at any point.

Discussion

Even though most of the evidence suggests that social anxiety is secondary to stammering, this case highlights the importance of mental state among people who stammer or have any form of speech difficulties. At the time of presentation DO'R was grandiose and disinhibited, with increased self-esteem and self-confidence. She was also over talkative with flight of ideas and pressure of speech and was less anxious and reticent in conversation. As stammering is associated with a reduction in self-confidence and self-esteem and assertiveness training forms part of the treatment, it seems plausible that situations in which self-confidence is increased could lead to a reduction in stammering. One such situation occurs during manic episodes. In this case DO'R's stammer did not return at two-year follow up and we can only hypothesise that her manic episode resulted in the resolution of her stammer.

One aspect of note was the complete resolution of this lady's stammer even after two years follow-up. Her return to speech fluency was not due to antipsychotic medication as her stammer had resolved prior to the commencement of treatment. Further, most studies suggest that antipsychotic medication can induce stammering rather than treat it and although some studies have reported a limited benefit this is usually short lived and not long lasting. In the absence of a clear biological cure to her stammering it is more likely that psychological factors have resulted in sustained improvement. The resultant improvement in her self-esteem and confidence after the birth of her baby could have reduced the chances of her stammer returning. The relationship between self-esteem, mood state and stammering will be better established over time. At the time of follow-up DO'R was euthymic and was off treatment. If she later develops a depressive episode and her stammer returns then this would point more conclusively to a relationship between her mental state and stammering.

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Basal ganglia MRI T_1-weighted signal hyperintensities in an alcoholic patient

Dear Editor,

Substance abuse disorders encompass a broad range of clinically heterogeneous and interchangeable syndromes which could confound diagnosis.

A 35-year-old-male patient with a history of chronic hepatitis C since 2002 and opiate and alcohol dependence since

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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/dl, 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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