Case Report

Obsessive Compulsive Disorder Presenting as Neurological Emergency

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ABSTRACT

Chronic epilepsy is leading to behavioral changes including obsessive-compulsive symptoms has been well-studied and shown to be about 22%, but the converse has not been reported. Here, we present a case discussion of a 45-year-old female, who presented with recurrent seizures with hyponatremia, which latter was ascribed to her undiagnosed obsessive compulsive disorder (OCD). This patient later did well on anti-obsessional treatment without any antiepileptic. This embarks the need for detailed psychiatric evaluation for patients in emergency care settings and gives a rare presentation of OCD.

Key words: *Hyponatremia, obsessive compulsive disorder, seizure*

INTRODUCTION

Obsessive compulsive disorder (OCD) can manifest with a wide range of clinical pictures.[1] On the other hand, there has been a long-standing observation that patients with various types of epilepsy/seizures have a higher incidence of many psychiatric disorders including OCD.[2-7] Though the equation of seizures leading to OCD is a known phenomenon, but to our best knowledge, we could not find literature showing OCD to cause seizures directly or indirectly. Here we present a case of undetected OCD presenting as repeated seizures, which is an indirect relationship might be first of its kind in literature.

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CASE REPORT

A 45-year-old female from rural Kashmir (India), married with four children, with previous three to four admissions for generalized tonic-clonic seizures from last 3 years was presently again admitted with two episodes of generalized tonic clonic seizures. As in previous instances she again had a low serum sodium level of 115 mEq/L. Rest of the investigations viz., hemogram, liver function tests, kidney function tests, blood sugar, urine examination, calcium, potassium, chloride, protein levels and lipid levels were in normal range. Her magnetic resonance imaging scan of brain and ultrasonography abdomen was normal. Every time her seizure was ascribed to the only abnormal finding of hyponatremia of 110 mEq/L, 114 mEq/L, 121 mEq/L, in previous admissions and the 115 mEq/L at the present. There was no apparent cause for this low sodium except for high intake of thiazides at the first admission. In spite of changing thiazides, she again had seizures with hyponatremia every time. She was evaluated for other possible causes of seizures and hyponatremia, but no concrete cause was found. Subsequent cerebrospinal fluid examination was normal. One out of three electroencephalograms

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showed nonspecific epileptic discharges. Considering her repetitive enquiring behavior and restlessness a psychiatric evaluation was sought.

On detailed psychiatric evaluation she verbalized pathological doubts, excessive cleanliness, excessive worries, repetition of acts. Her husband corroborated the same and reported her excessive intake of water. She had typical obsessions and compulsions. Upon further interview, she said that whenever she took water she felt as if she did not take and did not get satisfied, and she took more and more water for the same. Many a times she knew that she has taken a lot of water, but she felt compelled to take more. She further said that her idea of repeatedly drinking water was useless, but she could not resist it and had taken about 6-8 L of water on the day of seizure. Same had happened in the past seizures. She also described the similar repeated intake of antihypertensive tablets (thiazides) prior to her first seizure around 3 years back. Her husband further described her habits of taking medications over the counter, from her local health workers, changing and ill formed pain and ache complaints and corroborated her behaviors of intrusiveness, excessive washing, cleaning, checking and perfectionism. She described the intrusiveness of these thoughts and the disturbance in other psychosocial spheres for more than 15 years as was also reported by the family members. She had been put on tablet phenytoin sodium 300 mg daily since her first seizure, without any significant benefit.

On psychiatric evaluation she was diagnosed as OCD (International Classification of Diseases-10 Criteria) and put on fluvoxamine and cognitive behavioral therapy (CBT), however other causes of hyponatremia and polydipsia were still being ruled out. Tablet fluvoxamine was slowly titrated from 50 to 200 mg/day over 5 weeks, and CBT sessions were also given twice weekly. The patient showed a response to therapy. In the meantime, no apparent cause for his low serum sodium/seizure/excessive water intake could be found.

After a consensus with the neurologist, phenytoin sodium was slowly tapered from 6th month of the start of flouvoxamine, repeated electroencephalograms and serum sodium levels were followed and she was off phenytoin in 9th month. At present, after 28 months of follow-up she is significantly improved in OCD features and had no further seizures since then. Her Yale-Brown Obsessive Compulsive Scale score at start and at 28 months is 29 and 10 respectively. She scored 5 (markedly ill) for clinical global impressions severity (CGI-S) at start and at 28 months she scores 1 (very much improved) for CGI improvement and CGI-S also scores 1 (normal-not at all ill, symptoms of disorder not present past 7 days). Presently, she is only on fluvoxamine 200 mg/day, without any antiepileptic.

DISCUSSION

As our findings and investigations, we excluded our differential diagnosis for seizures such as central nervous system infections, space occupying lesions, drug intake, metabolic causes, and other possible causes for seizures. We could only find hyponatremia as the most plausible factor. As has been reported, seizures could be the only obvious neurologic manifestation of more moderate levels of hyponatremia.[8,9]

Hyponatremia seems to be most possible cause in this case, as a correction, and future avoidance of hyponatremia prevented further seizures. In light of normal renal functions and normal metabolic profile; a subtle, undiagnosed renal/metabolic defect can explain the development of hyponatremia in an otherwise healthy female, although development of hyponatremia with excessive water intake is known.[10,11] In this case, hypnonatremia seemed to be the sole cause of seizures.

Our hypothesis of OCD leading to excessive water intake and subsequent development of hyponatremia and seizure is more tangible because treatment of OCD led to the overall improvement. More importantly our patient is off antiepileptics, has improved oral contraceptive features and had an overall improvement and functionality.

CONCLUSION

Since epilepsy affects more than 50 million people worldwide, 80% of them live in developing world and a substantial number has no attributable cause, our finding is important in finding an indirect etiology (OCD) of seizures.[12] It also points to the unawareness and the load of the psychiatric presentations in the society. We suggest more involvement of psychiatrists in emergency units, and team approach with other specialties. It will help in avoiding unnecessary continuous antiepileptic and more importantly it shows a different presentation of OCD (close to psychogenic polydipsia) for which both emergency residents and the psychiatrists should scratch into.

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