Epilepsy & Behavior Case Reports 10 (2018) 106–110

Contents lists available at ScienceDirect

Epilepsy & Behavior Case Reports

journal homepage: www.elsevier.com/locate/ebcr

Case Report

Epilepsy and concomitant obsessive–compulsive disorder

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ARTICLE INFO

Article history:
Received 5 February 2018
Received in revised form 12 June 2018
Accepted 6 July 2018
Available online 20 July 2018

Keywords:
Epilepsy
Obsessive–compulsive disorder
Cognitive Behavioural Therapy
SSRI
Quality of life

ABSTRACT

People with epilepsy (PWE) often suffer psychiatric symptoms which can impact them more than seizures. Affective and psychotic disorders are well recognized as occurring more frequently in PWE than the general population. Less is known about obsessive–compulsive disorder (OCD) in PWE, despite it being as disabling and distressing. We sought to explore the association between epilepsy and OCD with casereports by identifying ten PWE and concomitant OCD. Demographics, seizure classification, neurological, surgical, psychiatric and psychological treatment as well as quality of life were examined. A detailed analysis was performed for three of them, to explore the lived-experience of patients with the two conditions. This is followed by a discussion of how treatment for co-morbid epilepsy and OCD can be appropriately tailored to be patient specific and provide the greatest potential for improvement.

1. Introduction

In 1890, Culerre first proposed an association between 'onomatomania' and epilepsy [1]. Later studies in the 1970s hinted at the emergence of obsessional traits as part of a specific behavioural syndrome in PWE [2]. In the general population, the prevalence of OCD is estimated to be around 2.3% [3]. This prevalence is considerably raised in general epilepsy clinics. Hamed et al. [4] reported that 39.7% of their patients had some obsessive-compulsive symptoms and 11.2% of people met DSM-IV criteria for OCD.

Temporal lobe epilepsy (TLE) is associated with a higher prevalence of OCD than other forms of epilepsy. Ertekin et al. [5] administered the Yale-Brown Obsessive Compulsive Scale [6] to groups of patients with generalized epilepsy and temporal lobe epilepsy. Obsessive–compulsive symptoms were significantly more disruptive for patients with TLE than other forms of epilepsy. This association stands to reason, given that our current understanding of the pathophysiology of OCD relies heavily upon the limbic system [7].

When a sample of TLE patients from a tertiary epilepsy clinic completed the Obsessive–Compulsive Inventory, 22% of those surveyed scored within the clinical range [8]. It is likely that this is an overestimation due to the selection bias inherent in recruiting people with drug-resistant epilepsy. However, in a secondary care clinic, Monaco et al. [9] found that 14.5% of TLE patients had OCD. People with TLE also displayed more sub-clinical obsessive personality traits than patients with generalised seizures, supporting the role of the limbic system in both general obsessive tendencies and OCD. Of note, only one of these people had a previous diagnosis of OCD, suggesting it seriously under-recognised in epilepsy clinics.

Psychiatric comorbidity has been shown to have a greater impact on the quality of life than seizure-related factors [10], with some studies finding that the greatest contribution is made by anxiety levels [11]. Obsessive–compulsive symptoms have been shown to directly affect the quality of life in people with epilepsy, as well as exerting an indirect influence through seizure-control and depressive symptoms [12]. Certainly OCD is a debilitating disorder; Bobes et al. [13] found that these patients reported worse social, emotional and mental health functioning than those with depression, heroin dependency, haemodialysis, or kidney transplants.

Here, we report a case series of people living with both disorders in order to obtain a greater understanding of the relationship between the two.

2. Materials and methods

Prior to commencing, approval was gained from the Department of Clinical Effectiveness at King's College Hospital, London (KCH) and the Clinical Audit Lead for Neurosciences at KCH. A non-consecutive, retrospective chart review was conducted for patients attending five epilepsy

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https://doi.org/10.1016/j.ebcr.2018.07.001

Table 1
Clinical characteristics of patients with obsessive–compulsive disorder and epilepsy. *CBZ = carbamazepine, SV = sodium valproate, LEV = levetiracetam, TPM = topiramate, LTG = lamotrigine, PGB = pregabalin, GBP = gabapentin, PHT = phenytoin, CLB = clobazam, PHB = phenobarbital, LCS = lacosamide, CLO = clonazepam, ZON = zonisamide, LOR = lorazepam, PMD = primidone, CBT = Cognitive Behavioural Therapy, SSRI = Selective Serotonin Re-uptake Inhibitor.*

Patient Number	1	2	3	4	5	6	7	8	9	10
Age (years)	37	40	28	55	45	46	29	49	49	42
Handedness	Left	–	Right	–	Right	Left	Right	Right	Right	Right
Epilepsy Onset Age (years)	22	6	–	16	5	18	4	13	20	9
Seizure Type	Generalised tonic–clonic & Myoclonic	Focal impaired awareness & Focal to bilateral tonic–clonic	Focal aware, sensory	Focal impaired awareness	Focal to bilateral tonic–clonic	Focal impaired awareness	Focal impaired awareness	Focal impaired awareness & Generalised tonic–clonic	Generalised Tonic–clonic	Focal impaired awareness & Focal to bilateral tonic–clonic
Current Medication	CBZ	CBZ, TPM, LTG,PGB	CBZ, LEV	GBP	SV, CBZ, LCS	CBZ, LCS, CLO	CBZ, LEV	ZON, LTG, CBZ	CBZ, LEV, GBP	ZNS, CBZ, LOR, PMD
Previous Medication	SV, LEV	LEV, GBP, PHT, CLB	GBP	CBZ, SV, PHT, PHB	LEV, PGB	LTG, PGB	CLB, SV, LTG, PGB, TPM	LEV, LCS	LTG, PHT	PGB
Resective Surgery	No	No	Left temporal lobectomy	No	Left temporal lobectomy	Right selective amygdalohippocampectomy with second further extension	No	No	No	No
Vagal Nerve Stimulator	No	Yes, ineffective	No	No	No	Yes, ineffective	No	No	No	No
Age of OCD Onset	–	–	–	–	–	26	18	20	26	28
Impact on ADL	Impaired relationships. Difficulty leaving home	Difficulty sleeping	Difficulty working	Unable to leave house to come to hospital	Unable to work; was homeless for 2–3 years	Impaired relationship, avoids parents	Difficulty getting a job and going to college	Difficulty leaving house, serious hoarding, suicidal	Hospitalisation, unable to work	Hospitalisation, suicidal ideation
Psychiatry Review	No	No	Yes	Yes	Yes	Yes	Yes	Yes	–	–
CBT	No	No	Yes	Yes	–	Yes	Yes	No	Yes	Yes
SSRI (mg)	No	No	Fluoxetine (40)	Escitalopram	Escitalopram (10)	Citalopram (20) Sertraline (25)	No	Paroxetine (30), Fluoxetine (20)	Paroxetine (20)	Citalopram (30), Fluoxetine (20), Sertraline (50)
Other seizure types and Disorders (including focal emotional aware)	No	Post-ictal psychosis, without focal emotional aware seizures	Depression	No	No	No	Depression	Depression	Post-ictal Psychosis, without focal emotional aware seizures	Post-ictal psychosis, without focal emotional aware seizures

clinics at KCH between 2012 and 2013. Of these, ten patients with complex or drug-resistant epilepsy were identified from the cohort who had also received a formal diagnosis of concomitant OCD. Clinical information was gathered about these individuals and the key characteristics of their disorders and management. Medical, psychiatric and surgical treatment was noted. A deeper case review was then performed for the three people with the most extensive clinical documentation in order to broaden our understanding.

3. Results

A summary of the clinical characteristics of the cohort can be found in Table 1.

3.1. Patient 6

Patient six is a 46 year-old, left-handed man with no family history of epilepsy or psychiatric disorder. At age 18, he was diagnosed with focal impaired awareness seizures and epilepsy. At age 26 he was diagnosed with OCD, although he could identify patterns of obsessional thinking preceding his epilepsy diagnosis by four years. His OCD is predominantly formed of obsessions. The focus of his ruminations centres on a fear of harming others. To combat the associated anxiety he checks through his memories to see if he has committed a crime. These thoughts are extremely distressing for him and have disrupted his personal relationships.

Histopathology from a right amygdalohippocampectomy at age 35 confirmed the presence of hippocampal sclerosis. Initially the surgery significantly reduced seizures, but over a course of years their frequency eventually returned to pre-operative levels. His OCD was treated with citalopram and specialist cognitive behavioral therapy, although he struggled to engage with this.

Cognitive Behavioural Therapy (CBT) is a psychotherapeutic intervention which enables people to develop tailored coping techniques focussed on challenging negative cognitions, behaviors and emotions. It is most commonly delivered one to one between a qualified therapist and the patient at regular intervals for between 6 and 12 sessions over 2 or 3 months.

At 39 years old he had a second surgical procedure to extend his previous resection. This resulted in some improvement in both his seizures and obsessive thoughts. One year subsequent to surgery, both seizures and OCD worsened. He underwent an intensive four-day course of exposure and response prevention which reduced his ruminations by a third.

More recently he was implanted with a vagus nerve stimulator, which had limited success. There were attempts to increase his citalopram dose, however, he suffered with lethargy and it was reduced to his standard dose. He has had a third course of CBT but he struggled to implement these techniques without direct guidance.

Of note is the chronological relationship between his epilepsy and OCD. A synchronous improvement and decline in both conditions following his second surgical procedure was evident. It remains the case, however, that he had little success with repeated treatment for either his epilepsy or OCD.

3.2. Patient 9

Patient nine is a 49 year-old right handed male with no family history of epilepsy or psychiatric disorder. Aged six months he suffered a traumatic brain injury and was delayed in reaching his developmental milestones. At 11 years old he suffered a haemorrhage from a left choroidal artery arteriovenous malformation and required neurosurgery.

Initially he suffered focal seizures which were managed with carbamazepine and phenytoin. He began experiencing obsessive–compulsive symptoms at 18 years old, which became increasingly severe. He suffered intrusive thoughts that he would harm others and was fearful of sharp objects and contamination. He attempted to alleviate the distress from these thoughts with ritualistic checking. These safety behaviours were maintained, and his anxiety reinforced, through his avoidance of fearful stimuli. He suffered his first generalised tonic–clonic seizure at 20, following withdrawal of carbamazepine.

Over the next ten years, he had three psychiatric admissions for treatment of his OCD. During his first admission, he underwent a program of graded exposure and response prevention therapy and reported 90% reduction in his symptoms. Adequate seizure control remained a challenge for him. He had several episodes of post-ictal psychosis when his obsessive–compulsive symptoms also worsened. The second admission had to be curtailed due to a worsening of his seizures. He was readmitted once his seizures were better controlled, and he reported a 70–80% improvement in his symptoms of OCD.

His OCD has remained fairly stable since, apart from a period where his ritualistic behaviors returned following an increase in his dose of levetiracetam. He no longer experiences post-ictal psychosis and has had several extended periods of seizure freedom. For this patient, periods of good seizure control proved vital in enabling him to overcome his OCD through psychological intervention.

3.3. Patient 10

Patient ten is a 42-year-old right-handed male with focal impaired awareness seizures and focal to tonic–clonic seizures, since the age of nine. Radiological investigation demonstrated right hippocampal sclerosis.

When he was in his mid-twenties, a family member died by suicide. He developed OCD aged 28. He experiences repeated intrusive and distressing thoughts that he will also die by suicide and has obsessional thoughts around certain colours and numbers. To reduce his anxiety, he engages in ritualised hand-washing behavior and avoids certain colored objects.

He was referred to Neuropsyhiatry at the age of 33 when experiencing several psychotic episodes during periods of seizure freedom. His seizure frequency varied in the medium-term and his OCD symptoms worsened during periods of good seizure control. While his seizures were fairly stable he began CBT for OCD. There were some improvements but these lacked consistency as his ability to resist engaging in safety behaviours was impaired after a seizure.

At age 35, he went through another period of improved seizure control with an associated deterioration of his mental state. This leads to an inpatient admission for treatment of psychosis. Soon after this admission he suffered a subarachnoid hemorrhage. The next year he was admitted for CBT treatment of his OCD which was having significant impacts on his life. His seizurecontrol worsened at this point. At 39 years old, he had an eight-week period of hypomania following a severe generalized tonic–clonic seizure.

His seizures remain poorly controlled. Despite this, he is clear that it is the OCD that is most problematic for him. He has struggled to consistently use techniques learnt during CBT and manages his anxiety with lorazepam. He finds his OCD highly distressing and feels that it is a greater burden on his quality of life than his epilepsy.

4. Discussion

The majority of patients in our case series suffered with TLE. This is likely due to the greater prevalence of OCD reported in this group. It may also be reflective of the fact that people with TLE are over-represented in tertiary epilepsy care.

In each of the expanded case reports, both the patient and treating doctor noted a relationship between severity of seizures and OCD symptoms.

There has been a drive to establish the neurobiological underpinnings of OCD [14, 15]. Some investigators suggest that there could be a shared mechanism with some forms of epilepsy, explaining the

increased coexistence of the two conditions. Kaplan [16] has previously reviewed the biological association. The temporary respite from OCD in one of our cases reflects surgical case reports demonstrating resolution of OCD following temporal lobectomy [17], and also its *de novo* occurrence [18].

As yet, there is no tailored intervention for OCD in people with epilepsy, despite the fact that epilepsy may have a bearing on the extent to which an individual is able to engage with the treatment. Success in CBT is dependent upon utilising strategies in-between sessions [19]. For those with on-going seizures, this would be greatly impeded. It may also affect their ability to attend regular weekly appointments. Many people with epilepsy face cognitive impairments from seizures, medications or surgery, which needs to be considered when working in the cognitive domain. This may also reduce the individual's ability to use strategies in the long-term. It is advisable for the therapist carrying out the intervention to have an understanding of the different ways in which an individual's epilepsy could affect therapy. Previous studies have found CBT designed for people with epilepsy experiencing anxiety or depression to be effective when delivered at both a group [20] or an individual level [21–23]. All of the three patients detailed above required repeated courses of psychological intervention.

For more severe and enduring forms of OCD, or if psychological intervention alone has not been effective, additional treatment with selective serotonin inhibitors (SSRIs) is recommended. The majority of patients in our series, seven out of ten, had tried at least one SSRI.

Pittenger et al. [24, 25] outline convergent strands of evidence from genetics, animal models, neuroimaging and neurochemical investigation, which implicate glutamate dysregulation in the cortical–striatal–thalamic circuits as underlying the pathophysiology of OCD. Of particular relevance for people with concomitant Epilepsy and OCD is research using the glutamate-modulating drugs topiramate [26–29] and lamotrigine [30–32], in addition to usual SSRI, for the treatment of refractory OCD in people without epilepsy. The effectiveness of these medications for those with both epilepsy and OCD may represent a promising avenue for further investigation.

One other psychopharmacological point of note would be the role of anti-seizure medications in association with the emergence or exacerbation of OCD in those susceptible, where it was previously at a subclinical level. Much has been written on levetiracetam in this regard [33–35]. Suspecting pre-existing obsessive–compulsive symptoms in someone who was being considered for this medication, might therefore encourage a clinician to advise an alternative.

Conclusion

Our case series has hopefully outlined the clinical characteristics of people with epilepsy and OCD. As the relationship between these two conditions is yet to be fully elucidated, and given the impact on people's quality of life, there is clearly a need for future research. To this end, prospective studies examining the relationship between epilepsy type, seizure frequency, chronicity and OCD symptomatology are required.

Declarations of interest

None.

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