

Obsessionality, obsessive–compulsive disorder, and temporal lobe epilepsy

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Abstract

We evaluated the prevalence of obsessive–compulsive disorder (OCD) in patients with temporal lobe epilepsy (TLE) and we investigated the hypothesis that obsessionality may represent a trait in TLE. Eighty-two consecutive patients with epilepsy, 62 with TLE and 20 with idiopathic generalized epilepsy (IGE), and 82 matched healthy controls were evaluated using the SCID-IP, Y-BOCS, MMPI-2 (specifically the Psychasthenia and Obsessiveness scales), BDI, and STAI Y1 and Y2. Nine of the TLE patients, none of the IGE patients, and one of the controls had a diagnosis of OCD. Psychasthenia and Obsessiveness scores were significantly higher in the TLE than in the IGE and control groups. Patients with TLE and OCD differed significantly with respect to history of depression when compared with patients with TLE without OCD, whereas there were no differences in age at onset and duration of epilepsy, seizure pattern and frequency, MRI features, laterality of the EEG focus, antiepileptic drug therapy and combinations, and BDI scores.

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1. Introduction

Behavioral changes in patients with epilepsy have been observed for many years. Early studies suggested a relationship between epilepsy and the development of specific personality characteristics, including obsessional traits, thus speculating about the existence of a behavioral syndrome, particularly in patients with temporal lobe epilepsy (TLE) [1–3].

Obsessive–compulsive symptoms (OCS) can present within a discrete disorder (obsessive–compulsive disorder; OCD) or as a part of some other psychiatric syndromes. Moreover, the etiology of OCS remains unclear, and there seems to be evidence of pathological

dysfunction in a variety of areas, for example, limbic system, basal ganglia, and orbitofrontal regions [4].

In the literature, despite single cases [5–8], a specific link between epilepsy and OCD has not been reported, although a recent study indicated a 22% prevalence of OCS in a group of 30 patients with drug-resistant epilepsy [9].

Whether behavioral abnormalities in epilepsy represent a trait (a distinguishing feature of the subject's nature) or a state (dependent on the role of the disease in the patient's life) and whether these abnormalities are specific to different epilepsy syndromes remain open questions. By definition, a *trait* is not pathological but is simply a distinguishing feature of a subject's personal nature and is present in each individual to a greater or lesser degree. This approach is well known and compatible with a quantitative psychometric approach

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to personality but has not been systematically applied to psychiatric symptoms in neurological disorders, to our knowledge.

The main problems in previous reports are the lack of systematic data, a selection bias derived by enrolling patients with drug-resistant epilepsy (the usual population in tertiary referral centers), the overuse of self-rating scales without a formal clinical evaluation, and the relative underutilization of widely accepted and standardized psychometric instruments.

The present study sought to evaluate the association between obsessionality and TLE from two different points of view: (1) the prevalence of OCD in TLE patients and the variables that may be associated; (2) the presence of obsessionality as a trait in patients with TLE when compared with healthy subjects and patients with other epilepsy syndromes such as idiopathic generalized epilepsy (IGE).

2. Patients and methods

2.1. Patients

All adult consecutive patients with a diagnosis of TLE or IGE (epilepsy with generalized tonic–clonic seizures of awakening, juvenile myoclonic epilepsy, and juvenile absence epilepsy) admitted to the epilepsy clinic (a second-level center) of the Department of Neurology, Amedeo Avogadro University in Novara, Italy, during 2003, were asked to enroll in the present study. Epilepsy was diagnosed according to the ILAE criteria [10] (clinical features, EEG or video/EEG, and neuroimaging) by at least two different neurologists not involved in the present study. We also enrolled a control group of healthy subjects, randomly selected from the general population, matched for age, gender, and education level.

We excluded patients younger than 18 years of age, patients with an uncertain diagnosis of TLE or IGE, patients with a reading level less than 6th grade, patients with learning disabilities or mental retardation, and, in general, patients with a Mini Mental State Examination (MMSE) <24.

After explanation of the procedures all subjects were asked to give informed consent.

2.2. Measures and procedures

All subjects were evaluated by a fully trained clinical psychologist (E.M.) under the supervision of a neuropsychiatrist (M.M.).

First, all subjects were assessed with the Structured Clinical Interview for DSM-IV Patient Version (SCID-IP) for OCD diagnosis and the Yale–Brown Obsessive Compulsive Scale (Y-BOCS). Second, we evaluated the presence of obsessionality as a trait, assessing all sub-

jects with the Minnesota Multiphasic Personality Inventory 2 (MMPI-2 adult version), looking specifically at the clinical scale Pt (Psychasthenia) and the content scale OBS (Obsessiveness). The Pt scale was originally developed to measure the general symptomatic pattern labeled by Janet as *psychasthenia* [11], not commonly used today, which is characterized by excessive doubts, compulsions, obsessions, and a rigid and perfectionist personality with unreasonable fear. Psychasthenia can be considered very close to modern OCD. The OBS scale was developed to identify patients with obsessive–compulsive behavior, maladaptative ruminations, or obsessive thoughts in general [12]. Previous studies had confirmed that the MMPI-2 can be used in patients with epilepsy, and only the Hs and Sc scales may need a modest correction in selected cases [13,14].

The MMPI-2, along with other scales (Beck Depression Inventory and State–Trait Anxiety Inventory Y1 and Y2), was administered in a standardized manner and in the same sequence to all subjects. MMPI-2 was scored by a computer using Psy-System II (version for Windows, O.S. Florence, Italy). Raw scores on each scale were converted to uniform *T* scores which have the same percentile and corrected for the *K* scale. MMPI-2 profiles were considered valid if fewer than 15 items were omitted and if in accord with validity scales. Data included in the analysis were *K*-corrected scale *T* scores. Scores >65 were considered pathological.

We excluded subjects who did not complete all the procedures. Initially, we compared patients with TLE and IGE and controls with respect to Pt and OBS *T* scores. Subsequently, we compared different TLE groups classified for seizure frequency for both scales. Finally, patients with TLE with OCD (TLE + OCD), patients with TLE without OCD (TLE – OCD), patients with IGE, and controls were compared with respect to Pt and OBS scores. The TLE + OCD and TLE – OCD groups were also compared on the following items: age; gender; personal history of psychiatric disorder (classified as mood disorder, psychotic disorder, personality disorder, behavioral abnormalities, or other); age at onset of epilepsy; duration of epilepsy; seizure frequency (classified as free, 1–10, 11–20 or >20 seizures/month); presence of psychic auras (especially fear auras); MRI features; laterality and localization of EEG epileptic abnormalities; anticonvulsant drug regimen and combinations; and BDI and STAI Y1 and Y2 scores.

χ^2 analysis or Fisher's exact test was used for categorical data, while ordinal and linear data were assessed by nonparametric tests and one-way analysis of variance. Given the number of analyses, a Bonferroni correction was applied, and differences and correlations were considered significant at $P < 0.003$. Statistical analysis was performed using the Statistical Package for Social Sciences (Version 12 for Windows, SPSS, Inc., Chicago, IL, USA).

3. Results

We enrolled 164 subjects: 82 patients with epilepsy (38 males), mean ± SD age (range) 37.4 ± 15.0 (18–75) and 82 matched controls (38 males), mean ± SD age (range) 37.8 ± 16.9 (18–76). In the group of patients with epilepsy, 62 (25 males) were diagnosed with TLE and 20 (13 males) with IGE.

Seizure frequency and antiepileptic drug (AED) regimen in the TLE group are detailed in Table 1. The most frequently prescribed drug was carbamazepine (CBZ) (61.3%). All IGE patients were seizure free for at least 1 year and on monotherapy: 18 (90%) were taking valproate (VPA), and only 2 (20%), lamotrigine (LTG).

According to the SCID-IP, 9 patients (14.5%) in the TLE group, none in the IGE group, and only one (1.2%) in the control group had a diagnosis of OCD ($P = 0.002$). The severity of OCD was Y-BOCS = 12.7 ± 4.6 in the TLE group and Y-BOCS = 10 in the control group.

Pt and OBS scores were significantly higher in the TLE group (Pt = 56.4 ± 12.3, OBS = 57.0 ± 11.0) than in the IGE (Pt = 46.6 ± 7.4, OBS = 47.2 ± 8.0) and control (Pt = 48.4 ± 9.4, OBS = 49.1 ± 9.9) groups (Pt $P < 0.001$, OBS $P < 0.001$) (Fig. 1). Pt and OBS scores were significantly different in TLE + OCD (Pt = 67.4 ± 10.8, OBS = 65.8 ± 12.5), TLE – OCD (Pt = 54.3 ± 11.5, OBS = 55.8 ± 10.4), IGE (Pt = 46.6 ± 7.4, OBS = 47.2 ± 8.0), and control groups (Pt = 48.4 ± 9.4, OBS = 49.1 ± 9.9) (Pt $P = 0.003$, OBS $P = 0.004$) (Fig. 2).

There were no differences in Pt and OBS scores of TLE patients who were seizure-free (Pt = 56.2 ± 11.8, OBS = 57.2 ± 11.0), who had 1–10 seizures/month (Pt = 57.0 ± 14.6, OBS = 57.9 ± 11.1), who had 11–20 seizures/month (Pt = 53.0 ± 14.1, OBS = 55.0 ± 8.4), and who had >20 seizures/month (Pt = 59.5 ± 16.2, OBS = 57.5 ± 11.9) (Fig. 3).

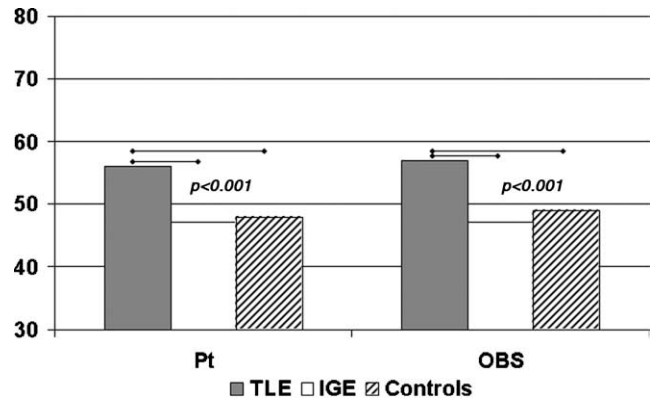


Fig. 1. Psychasthenia (Pt) and Obsessiveness (OBS) scores in patients with TLE, and IGE and controls.

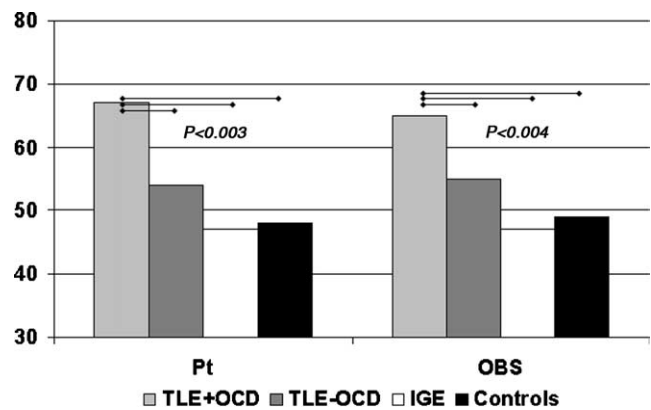


Fig. 2. Psychasthenia (Pt) and Obsessiveness (OBS) scores in patients with TLE and OCD (TLE + OCD), TLE without OCD (TLE – OCD), IGE and controls.

Similarities of and differences between the TLE + OCD and TLE – OCD groups are summarized in Table 1. There was a statistically significant difference in previous psychiatric history, mainly depression, in

Table 1
Demographic data and variables of patients with TLE with and without OCD

Variables	TLE with OCD (9)	TLE without OCD (53)	P value
Gender, male/female	4/5	21/32	>0.05
Age, years ± SD	31.8 ± 6.6	42.4 ± 15.5	>0.05
Age at onset, years ± SD	16.5 ± 11.9	27.8 ± 18.1	>0.05
Duration of disease, years ± SD	15.4 ± 13.2	14.6 ± 13.3	>0.05
Previous psychiatric history, no/depression/psychosis/OCD/other	2/4/2/1/0	44/8/0/0/1	<0.001
MRI features, normal/left/right/bilateral damage	9/0/0/0	42/1/7/3	>0.05
Presence of hippocampal sclerosis	0	5	>0.05
EEG epileptic abnormalities, normal/left/right/bilateral	0/5/2/2	2/21/12/18	>0.05
EEG epileptic abnormalities, normal/anterior/posterior	0/7/2	2/26/25	>0.05
AED regimen, mono/dual/polytherapy	5/4/0	41/9/3	>0.05
Secondary generalized tonic-clonic seizures	4	31	>0.05
Simple partial seizures	3	13	>0.05
Complex partial seizures	6	32	>0.05
Seizure frequency, free/1–10/11–20/>20 per month	5/3/0/1	41/9/2/1	>0.05
BDI, median (range)	8 (3–34)	5 (0–48)	>0.05
STAI Y1, mean ± SD	48.3 ± 11.7	40.0 ± 10.6	=0.04
STAI Y2, mean ± SD	55.6 ± 10.2	44.0 ± 12.5	=0.01

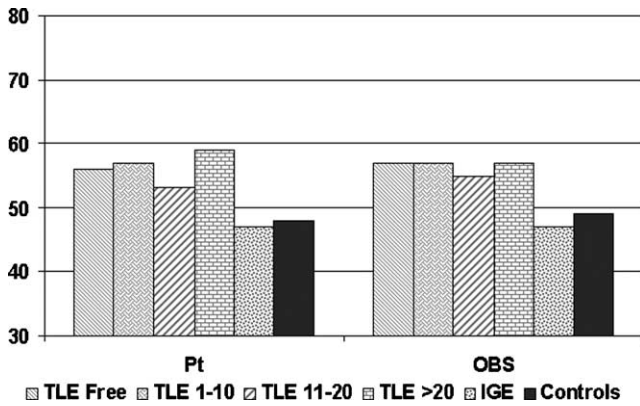


Fig. 3. Psychasthenia (Pt) and Obsessiveness (OBS) scores in patients with TLE according to seizure frequency, patients with IGE, and controls.

patients with TLE and OCD (77.8%) when compared with patients with TLE without OCD (4.3%) ($P < 0.001$). There were no differences in age at onset of epilepsy, duration of epilepsy, seizure pattern and frequency, MRI features, laterality of EEG focus, AED therapy and combinations, and BDI scores. Differences in STAI Y1 and Y2 were not considered statistically significant because of the Bonferroni correction.

4. Discussion

To our knowledge, this is the first study addressing, in a representative sample of patients with epilepsy, two relevant problems in the literature, namely, the prevalence of OCD in TLE and whether obsessiveness represents a state or a trait. By definition, a trait is not pathological but simply represents a distinguishing feature of a subject's personal nature and is present in each individual to a greater or lesser degree. This approach is highly compatible with the quantitative psychometric approach to personality testing of MMPI-2. In the past, there have been many criticisms of MMPI studies in epilepsy [3], and it was in part to overcome such difficulties that Bear and Fedio developed their rating scale (Bear and Fedio Inventory) [2], but, compared with the previous version, the MMPI-2 represents a completely revised and renormed psychometric test. It represents a well-known, widely reproduced and standardized test for adult psychopathology. In our study we investigated only Pt and OBS scales so we were justified in comparing healthy controls and patients with IGE and TLE without correction for seizure content, and we decided to recruit, as a control group, healthy subjects and not subjects with a chronic medical condition or a neurological disorder because psychological factors seem to be less relevant than biological factors in OCD genesis [15]. Although it has been shown that Pt scores can be

elevated in patients with other anxiety disorders (such as panic attack disorder, generalized anxiety disorders, and phobic disorders), the association between Pt and OBS scales makes the specificity for obsessiveness spectrum symptoms definitely higher. A potential confounding variable may be the presence of specific manifestations, such as fear auras in the TLE population, which could falsely elevate Pt, OBS, and STAI scores, but in our sample only three patients (4.8% of the TLE group) had auras consisting of fear.

Looking at the MMPI-2 Pt and OBS scales, patients with TLE had higher T scores than IGE patients and controls (Fig. 1), and T scores of patients with TLE without OCD, though in the normal range, are not similar to scores of controls or patients with IGE but are between the scores of these subjects and those of patients with TLE and OCD (Fig. 2). Comparing patients with TLE and different seizure frequencies (especially seizure-free patients) with IGE and control subjects (Fig. 3), we observed again that TLE patients score higher, though in the normal range, than IGE patients and controls on specific obsessiveness scales with no relationship to severity of the epilepsy, etiology, or medication type or combinations. All these pieces of evidence, taken together, suggest that obsessiveness is a trait in TLE patients and only those with a biological vulnerability (indicated by the previous psychiatric history) develop OCD. Therefore, we might speculate that there is a link between some personality traits and the involvement of mesolimbic structures, reopening the question of whether a specific limbic system behavioral syndrome exists in patients with TLE.

Some authors have suggested that psychopathology is generally associated with epilepsy, and differences may be inferred only with respect to disease severity and psychosocial reasons, with no relationship to a generalized or localized syndrome [16]. Our study provides more evidence that the involvement of different brain structures, in different epileptic syndromes, plays a role in the susceptibility to develop specific psychopathological syndromes. The fact that our IGE patients were similar to normal controls is in line with the literature reporting a range of personality features in IGE different from those investigated in our study [17].

Another interesting point to take into account is the role of different AEDs in the susceptibility to develop psychopathology in epilepsy. Although TLE and IGE patients were more likely to be on CBZ and VPA therapy, respectively, there is no evidence that these two AEDs have different activities in anxiety disorders, especially OCD. Therefore, we do not think that this variable may be of value in explaining our results.

In our study, we found that 14.5% of patients with TLE have a diagnosis of OCD. A previous study [9], using the Obsessive Compulsive Inventory, reported a

22% prevalence of OCS but in patients with drug-resistant TLE, and the authors did not formally diagnose OCD. By contrast, we used the SCID-IP and Y-BOCS to evaluate OCD prevalence in a group of patients admitted to a second-level epilepsy clinic, excluding a possible bias due to selection of patients with drug-refractory seizures as in tertiary referral centers or in pre-surgical audits. It is important to emphasize that only one of nine patients in our OCD group had a previous psychiatric diagnosis of OCD, suggesting that OCD is underdiagnosed in patients with epilepsy, probably because it is difficult for epileptologists not trained in psychiatry to make such a diagnosis, and underlining the role of neuropsychiatrists in epilepsy clinics and centers.

In comparing patients with TLE and OCD with patients with TLE without OCD, we did not observe any difference in age at onset, duration of disease, seizure frequency and pattern, AED therapy, and EEG laterality, the latter also in accord with other studies [9,18]. This observation is rather intriguing because it may suggest that variables other than the role of disease (probably anatomical ones) are implicated. This study provides more evidence of the role of the limbic system in obsessiveness in general and OCD in particular. Another study suggested that OCD in patients with neurological disorders may be due to structural damage to specific frontal–limbic–subcortical circuits [19], and a key role of the limbic system has been shown in terms of paroxysmal activity by different authors [20,21]. In fact, complete remission of OCD after epilepsy surgery has been reported [22].

The amygdala seems to be crucial to affective and motivational elements of OCD [23]. The amygdala has dense connections with the striatum that are presumed to support an efficient system for driving automated behavior in response to danger, while reciprocal connections with the extended amygdala, including the ventral striatum and the bed nucleus of the stria terminalis, may help to mediate the anxiolytic by-products of repetitive behaviors [24]. In our study we failed to show peculiar MRI features in patients with TLE and OCD, but it is clear that future investigations of the amygdala are needed to understand the biological basis of obsessionality in epilepsy.

5. Conclusions

This study demonstrates that OCD has a higher prevalence in TLE than in the general population and is often underdiagnosed. Further, our results suggest that obsessiveness is associated with TLE per se, suggesting a link with limbic structures and supporting the hypothesis that behavioral manifestations in epilepsy are specific to epilepsy type.

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