A man in his 40s with repeated seizures

A man in his forties had been repeatedly assessed over a period of 15 years at neurological departments and specialised epilepsy hospitals. The conclusion was always psychogenic, non-epileptic seizures (PNES), but there were no proposals for treatment. Prior to the current treatment he had also undergone several psychiatric assessments, which found the seizures to be dissociative and consistent with psychogenic, non-epileptic seizures. Psychotherapeutic and medical approaches had been ineffective.

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The patient's first seizure occurred in connection with a high fever during holiday travel. The seizure consisted of loss of consciousness and atypical convulsions. He was assessed in hospital, including by a neurologist, but no signs of organic brain pathology were found. Increased holiday alcohol consumption combined with high fever were postulated as possible triggering factors. During the next six years he had two similar episodes in connection with a respiratory infection and fever.

Alcohol is known to trigger seizures, exacerbate seizure control in epilepsy patients or induce epilepsy. In every third acute admission after an epileptic attack, more alcohol than normal has been consumed prior to the episode (1). Episodes triggered by fever are also common, but more frequent in children. As a rule these do not cause brain damage (2).

Eight years after the first seizure, he began suffering recurrent seizures which started with involuntary movements, mostly of the extremities, during full consciousness and accompanied by hyperventilation. These movements were followed by loss of consciousness without urinary or faecal incontinence and with amnesia concerning the seizure period. The convulsions could last from a few minutes to 40 minutes, and could also occur in series continuing for several hours. On some days he might suffer no seizures, on other days several. Overall, he averaged 6–30 seizures per month. The seizures were provoked by job-related stress, insomnia and situations with many people present and noise. After the episodes he was usually sleepy and fatigued, with back- and knee-pain that could last for several days.

After a further three years he experienced increasing difficulty in finding words for objects. He also found that on «bad days», prior to new attacks, both speech and movements were slower.

During these years he was admitted repeatedly to the Neurology Department for assessment, and underwent a lumbar puncture, EEG and MRI of his head. There were no findings that could explain his condition. The EEG was interpreted as mildly pathological with slightly high slow activity over the left front temporal region, but without definite epileptic activity. The conclusion was psychogenic, non-epileptic seizures. He received no medical treatment apart from escitalopram and diazepam, which had no observable effect.

Psychogenic, non-epileptic seizures can be defined as an observable, sudden seizure-like change in behaviour or consciousness which resembles an epileptic seizure but is not accompanied by the EEG changes that are characteristic of epileptic seizures or by other evidence of epilepsy or other somatic causes. However, many believe that cases of acute admissions with major, dramatic, GTC-like seizures where psychiatric examination does not reveal a mental disorder could be classified as conversion/dissociation. As a result these seizures are classified as dissociative/psychogenic non-epileptic seizures.

On three occasions he was assessed at specialised epilepsy centres. 24-hour, 26-channel long-term EEG monitoring was conducted during which the patient experienced three of his characteristic seizures. The monitoring revealed short bursts of rhythmic 5–6 Hz theta interictal activity, partly spiked temporally with a left-sided predominance, which created uncertainty as to whether it could be a matter of a pathological condition or a rare physiological variant. However, it was concluded that the seizures were psychogenic, and both diazepam and escitalopram were discontinued.

A further two neurological assessments later yielded the same result. On one occasion, 45 mg intramuscular diazepam was administered during a prolonged seizure, without effect.

The differential diagnostic procedure for distinguishing between psychogenic, non-epileptic seizures and epilepsy has improved over the past 30 years, particularly since the introduction of video EEG monitoring (3). The occurrence of psychogenic, non-epileptic seizures in the general population is estimated at about 1.5/100 000 (4, 5) and 25–30 % of all those referred to epilepsy centres because of treatment resistant epilepsy receive this diagnosis (6, 7). Inadequate diagnosis can prevent appropriate mapping, processing and treatment of the underlying psychosocial causes, resulting in prolongation of the disorder and associated stigma and of the patient's status as an invalid. In those cases where ineffective anticonvulsant medication is prescribed, stressful side effects may also occur.

The conclusion of a psychiatric assessment was that the patient had «dependency and conflict-avoidance tendencies», but no grounds were found for further assessment/treatment. He was also assessed and treated by a psychologist in private practice, but the traditional screening instruments used yielded no evidence of a mental disorder. However, the Minnesota Multiphasic Personality Inventory (MMPI) (8) revealed conflict-avoidance features, psychosomatic reaction tendencies, unconscious suppression, intensified control, aggression inhibition and limited self-insight. It was assumed that the patient had a relatively limited coping and reaction repertoire, with a tendency towards dissociation and a psychosomatic seizure reaction in stressful situations. Therapy focused primarily on insight into the personality background to the seizures, triggering factors, and how seizures could be prevented. The patient cooperated well, but the therapy resulted in no lasting improvement in the frequency or severity of the seizures.

During six weeks of observation and assessment in a psychiatric department, several incidents involving loss of consciousness and convulsions were observed weekly. The patient was discharged with the diagnosis dissociative convulsions (F 44.5). He tried pregabalin for a few weeks, but without effect.

The problems in question resulted in increasingly frequent sick leave and finally in disability pensioning, 14 years after the first seizure.

According to ICD -10, dissociative convulsions may «mimic epileptic seizures very closely in terms of movements, but tongue-biting, bruising due to falling, and urinary incontinence are rare, and consciousness is maintained or replaced by a state of stupor or trance.» (9).

After the patient had reported over time, apparently for no reason, a growing tendency to depressivity and irritability, especially prior to seizures, his primary doctor referred him again to a regional psychiatric centre with a request for «psychological assistance in tackling situations when his seizures occur». The patient described himself to the psychiatrist as a very energetic and emotionally unstable person who responded with irritability and excessive emotional reactions to small incidents. He mentioned problems in keeping calm, particularly when he felt engaged, and that in periods before he was put on disability pension he was able to work more than 12–14 hours a day, with little need for sleep (3–4 hours per day) and little sense of tiredness. Evidence of bipolar disorder type II was found, and the patient agreed to lamotrigin treatment. The drug was titrated up in the normal way to a daily dose of 200 mg.

A number of studies indicate that bipolar disorders are often underdiagnosed in primary care, partly due to an absence of typical manic symptoms (10, 11). Symptoms such as slightly euphoric mood, unusual irritability and decreased need for sleep over a period of at least four days may be enough to cause suspicion of bipolar disorder (10, 11). Lamotrigin is used both as a mood stabiliser for bipolar disorders and as an anticonvulsant for epilepsy. Existing evidence indicates that bipolar disorders and epilepsy may have some biological mechanisms in common (12, 13).

Some weeks after the start of treatment, a considerable, stable improvement took place in the patient's state of health and quality of life. His seizures grew fewer and shorter, the patient felt his mood was more stable, he had better control of his feelings and he stopped feeling unsteady. A few months later his daily dose was increased to 300 mg. Some weeks after that, he averaged one fifteen minute seizure every second week. He was still less irritable and his mood was more stable. During one period he experienced a number of conflict-filled situations and on some days he had a number of seizures, but he still remained generally more stable and calm. He described his quality of life as substantially improved. After a consultation with the neurologist, the dose was gradually increased to 600 mg daily, and serum lamotrigin was measured as 29 mmol/ml (10–60 mmol/l). After this the seizures occurred an average of 1–2 times per month and lasted for 15–20 minutes. About 2.5 years after starting on lamotrigin, the patient has seizures at intervals of 3–4 weeks. He is satisfied with his treatment, feels stable and reports a good quality of life.

BOX 1

Bodde et al's five-level model of psychological factors that may be involved in psychogenic, non-epileptic seizures (14).

a) **Psychological factors** that may cause the condition, such as sexual abuse and personality disorders.

b) **Vulnerability factors,** which may cause a predisposition for psychosomatic reactions, such as a tendency to dissociate and a limited ability to regulate stress or resolve conflicts.

c) **Shaping factors,** such as close relatives with similar symptoms.

d) **Triggering factors,** such as stress and inter-personal conflict.

e) **Prolongation factors,** such as gains in the form of attention and care, plus avoidance of coping and situational challenges.

Discussion

Our patient had repeated neurological assessments over a period of 15 years at both neurological departments and specialist epilepsy centres. The consensual conclusion was psychogenic, non-epileptic seizures, but there were no treatment proposals. Prior to the present assessment and treatment he had also undergone several psychiatric/psychological assessments, which found the seiz-

ures to be psychogenic (dissociative) and consistent with psychogenic, non-epileptic seizures. Psychotherapeutic and medicinal approaches had not been effective.

Bodde et al. (14) outline a five-level model of psychological factors that may be involved in psychogenic, non-epileptic seizures (Box 1). This model resembles other models used to explain somatoform disorders. When applied to our patient, he had a) emotional instability, personality-related defensive tendencies and a bipolar disorder b) problems in regulating everyday stress, c) clear triggering factors in the form of overwork, insomnia and noise and d) the «gain» that the seizures had the effect of diverting the emotions and feelings that arose in the situations in which the seizures occurred (amnesia). The patient's psychosocial history is thus quite consistent with psychogenic, non-epileptic seizures.

The earlier psychological assessment of our patient was thorough, but did not lead to effective treatment. When the neurological assessment focuses only on excluding epilepsy, psychogenic, non-epileptic seizures becomes an exclusion diagnosis. A positive diagnosis is necessary in order also to acknowledge and treat the underlying mental disorder. It can be particularly challenging when the responsibility for different aspects of a composite problem fall to different disciplines – in this case, neurology and psychiatry.

Psychogenic, non-epileptic seizures are normally regarded as a condition that should be treated in the field of psychiatry (15). However, our example shows that these patients may fall between the two stools of neurology and psychiatry, instead of these specialities cooperating on assessment and treatment.

The neurological assessments also yielded non-specific EEG findings that were not consistent with «pure» psychogenic, non-epileptic seizures. For example: could the first seizures, which were accompanied by fever, have caused brain damage that the EEG did not reveal?

The stress-vulnerability model, in which an x-axis represents a continuous vulnerability or organicity dimension and a y-axis represents a continuous stress dimension, allows all possible combinations of organicity and stress (16). In this case history, it appears that it may have been difficult to discuss the possible implications of several possible pathological EEG findings while maintaining the diagnosis.

However, existing research has shown that psychogenic, non-epileptic seizures by no means exclude the possibility of comorbid epilepsy. Some studies have shown that up to 50 % of patients with the condition also have comorbid epilepsy, or have had it in the past, and that the epileptic component is severely underdiagnosed (17–19).

The comparison between psychogenic, non-epileptic seizures and epilepsy is logical in view of the fact that the most evident clinical manifestation is seizures. In an attempt to understand the pathogenesis in a broader biopsychosocial perspective, a broader differential diagnostic approach should be used.

The treatment with lamotrigin was started after a new psychiatric assessment concluded that the patient's mental problems were consistent with bipolar disorder type II. At that time the patient had been an invalid for several years because of his seizures.

The relationship between epilepsy and bipolar disorders has been the subject of extensive discussions, particularly in neurological publications. Several studies have found that epilepsy and affective disorders have many common pathophysiological features (20–24). Extensive comorbidity of affective disorders, particularly depressions (25), bipolarity (26, 27) and epilepsy, the discovery of an increasing number of common pathogenetic features and the benefit derived from anticonvulsants and antidepressants by patients in both diagnostic groups (28) indicate that these two conditions are more closely related than previously believed.

Our case history, with the effectiveness of lamotrigin, may indicate that psychogenic, non-epileptic seizures should be considered in connection with both fundamental organic brain pathology, which cannot always be detected with existing examination methods, and with bipolar disorders. Our definite recommendation is that the diagnosis and treatment of patients with this condition require close cooperation between neurologists and psychiatrists.

The patient has consented to the publication of the article.

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