E-Da Medical Journal 1 (2014) 18-23

**Case Report**

# Simple Partial Epilepsy in a Psychiatric Patient Presenting with Uncontrollable Crying: A Case Report

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A young university student presented in his first psychiatric visit with sudden-onset episodic uncontrollable crying without feeling sad, episodic gooseflesh at anxiety, impulsive anger, cognitive difficulty and short-term depressive mood. Since adolescence, he had suffered from frequent episodic sensory and autonomic symptoms suggestive of possible partial epilepsy. The unusual episodic crying, atypical mood changes, and a past history of head injuries with hypoxia aroused the suspicion of posttraumatic epilepsy, though routine scalp EEG and standard MRI study showed no strong supports. Under the suspicion of partial epilepsy, he was treated with clonazepam and lamotrigine that kept him symptom-free with acceptable social performance. It is widely accepted that distinction among primary psychiatric disorders, partial epilepsy, and nonepileptic seizures is a challenge for physicians. This case report demonstrated the identification of unusually presented partial epilepsy through detailed chronological history-taking including symptoms and head injuries despite the lack of significant EEG and MRI findings. Clinically, the chance of underdiagnosis for partial epilepsy could be reduced with sufficient attention being paid to the core features of epileptic disorders and symptom spectrum of partial epilepsy.

**Key words:** uncontrollable crying, head injury, simple partial epilepsy, lamotrigine

Though it is widely accepted that differentiation between complex partial seizure disorder and psychiatric illness is a formidable challenge in psychiatric practice,[1-3] the recognition of simple partial seizure in psychiatric patients could be difficult and generally overlooked. While seizure disorders occur in 0.5-2% of the general population, the incidence is doubled to an estimated 1.0-4.0% among psychiatric inpatients across all diagnoses.[1] On the other hand, the occurrence in psychiatric outpatients is unknown and has not been well studied.

The diagnosis of seizure type(s) relies mainly on a detailed chronological description of ictal signs and symptoms experienced by the patients and other witnesses.[2,4] There is one general rule to be applied to epilepsy—The more unusual experiential or behavioral phenomena, the more likely they are of epileptic origin, provided that the core features (i.e., abrupt onset, short duration, stereotyped

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Received: November 3, 2009 Accepted: May 5, 2010
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sequence of ictal signs and symptoms) of an epileptic seizure are present.[4]

In this case report, we show the identification of simple partial epilepsy in a young man presenting with irresistible crying, irritable rage, episodic nervousness with sensory, motor, and autonomic symptoms. There were also mood, sleep, and anxiety symptoms mixed with these odd clinical characteristics. The purpose of this case report is to highlight the role of detailed history-taking and the importance of familiarity with the symptomatology of partial seizures in differentiating a seizure disorder from primary psychiatric disorders.

## Case Report

In his first psychiatric outpatient visit, Mr. A, a 21-year-old university student, presented with sudden onsets of uncontrollable crying without feeling sad and intense fluctuation in mood between happiness, sadness, anger, and depression for 2-3 months. The crying usually lasted for 1 to 2 minutes in each episode. Besides, he was bothered by sudden feeling of anxiety with gastric upset, chest tightness and shortness of breath, forgetfulness about what he was planning to say and difficulty in concentrating, especially on experiencing dizziness and head heaviness. He also reported problems with emotional control such as physical acting-out while getting frustrated in operating computers and angry fist-clenching and teeth-gritting while being annoyed by surrounding noises.

During the same period, he also experienced low or irritable mood, social withdrawal, decreased interests, sleep disturbance with late onset of sleep after lying in bed for 2-3 hours, frequent sleep disruptions, fatigue, and mental slowness; the above symptoms usually lasted within one week in each episode.

Examination of his past history revealed that he had a drowning episode at age 3, with loss of consciousness for about 2-3 minutes until being brought around by the rescuer's cardiopulmonary resuscitation at the side of a swimming pool. Two episodes of direct head injuries were also recalled and supported by his family: First, he had a head injury while playing on the slide. He then experienced drowsiness for more than 30 minutes. His mother had to give him pain stimulation to keep him partly awake. He received blood transfusion about 2 packs and brain concussion was diagnosed. Second, he fell down with head hitting onto a table corner and bled. A prominent scar was left over the left eyebrow. Both traumas occurred at his age of 4-5. During the period of primary school, he felt difficult to concentrate in classes and had repeated experiences of déjà vu toward places, events, objects, and some conversations. The frequency was higher and duration shorter in childhood than at the present time. In junior high school, he had frequent gooseflesh over both arms and upper legs, lasting for about ten seconds. He felt like being shocked by static electricity and became avoidant to classmates' touch which might trigger such attacks. In senior high school, the gooseflesh symptoms became milder. He began to experience episodic stinging pain, over his back about 2-3 times a day at most. There were also sudden jerks involving the whole body or solely the arms while in clear consciousness or at sleep, which usually startled his classmates.

Since his sophomore year (about two years before his first psychiatric visit), he began to experience involuntary blinking of both eyes or muscle contractions between eyebrows, as well as episodic twitching over right side nasal wing, especially while feeling tired. An ophthalmologist attributed the cause to eye fatigue and one traditional Chinese doctor considered those facial motor symptoms to be related to nasal allergy. He also suffered from episodic involuntary whole body shaking for 1-2 seconds and transient high-tone tinnitus predominantly over the right ear with right side

postaurical discomfort for 2-3 seconds, occurring 1-2 times a week.

His personality was described by roommates and classmates as independent, hypotalkative, cool (passive in interpersonal interaction) and sometimes queer. On careful interview, he denied significant conflicts or stress from interpersonal or intrafamilial interaction. Actually, he felt that the atmosphere of his family had been much improved in recent two years since his parents stopped criticizing and arguing with each other. He attributed the trigger of the symptoms causing his first psychiatric visit to the combined burdens from preparing for three examinations and also his three part-time jobs. The unexplained physical and emotional symptoms as well as poor sleep quality were his main concerns. His episodes of depressed mood and social withdrawal never exceeded one week and there was no significant associated change in appetite, body weight, sleep, idea of worthlessness, hopelessness, or prominent dysfunction in social activities. No well-defined manic/hypomanic attacks or obvious traits of personality problems were detected either. Mr. A performed fairly well in school (ranking at least the top 10 in high school and with an average score of 85 at university).

Scrutinization of his family history of mental illness revealed depressive disorder in his grandmother, mother, and aunt, all of whom had previously received regular psychiatric management. His mother had one previous suicide attempt with drug overdose. Mr. A was often criticized verbally by his father and physically abused by his monther, including foreful blows to his head. Mental examination during first several visits revealed dysphoric mood, mild anxiety, worries regarding his uncontrollable crying, labile emotion, rage and difficulty in falling asleep. Electroencephalography (EEG) showed no cortical dysfunction initially, but rare intermittent, diffuse theta waves indicating mild, diffuse cortical dysfunction 5 months later. His standard brain MRI did not show any significant abnormality.

Mr. A was treated under the impression of "depressive disorder not elsewhere specified" according to the criteria of Diagnostic and Statistic Manual, 4th edition (DSM-IV) and a presumed partial epilepsy. In the first two and a half months, drug response was unsatisfactory with clonazepam, alprazolam, and escitalopram (a serotonin-reuptake inhibitor), and there were still transient episodic queer symptoms such as irresistible crying (1 to 2 minutes each time), right ear tinnitus, right facial twitching, numbness over right posterior head for one minute, sleep talking in Japanese, whole body tremor, involuntary hastening of eating, visual hallucination of black shadow or flash light, and confusing of dream contents with reality. Therefore, valproic acid 200 to 400 mg was added under the suspicion of posttraumatic partial epilepsy. After medical treatment for ten months with clonazepam and antiepileptics (the initial valproic acid was shifted to lamotrigine because of severe weight gain), those troublesome episodic neurological symptoms almost disappeared. He could achieve a fairly stable mental condition by taking only clonazepam 0.5 mg and lamotrigine 200 mg before bedtime. No distressing depression or anxiety recurred after cessation of antidepressant or anxiolytics for 10 months. Mr. A was performing fairly well at the graduate institute. He felt much satisfied with current medications. He could deal with most emotional and somatic distress with a clear insight that he was afflicted with a possible posttraumatic partial epilepsy instead of major primary psychiatric illness.

## Discussion

The illness of Mr. A represents a challenge of distinguishing among primary psychiatric disorders, partial epilepsy, and nonepileptic seizures. As psychiatrists are generally less

sensitive to symptoms characteristic of partial seizure, they tend to pay more attention to the mood or anxiety. Major depressive disorder was excluded according to the DSM-IV criteria, because Mr. A's episodes of depressed mood and social withdrawal never exceeded one week, the other depressive symptoms were limited in number and intensity, and there was no prominent functional decline. In contrast, his queer behaviors, i.e., uncontrollable crying even in happy mood and episodic somatic symptoms in associated with anxiety and anger, were atypical in depressive patients and hence aroused the suspicion of a partial seizure disorder. Together with a detailed history and drug response, pieces of evidence supporting the diagnosis of partial epilepsy emerged, as depicted below.

**1. Presenting symptoms.** Patients with partial epilepsy may present with a wide variety of psychiatric symptoms, including depression, anxiety, psychosis, hypomania, neurovegetation, fatigue, cognitive disorders,[1] and unusual presentations such as crying, singing, episodic dyspnea, and vomiting.[5] Such symptoms may render these patients frequently misdiagnosed as psychiatric illnesses. Mr. A had uncontrollable crying even without internal sadness or external trigger, which he felt inappropriate and irresistible. This finding gave the initial clue to the diagnosis of partial epilepsy.

**2. Associated symptoms.** There were various queer somatic symptoms all of which had the characteristics of abrupt onset and short duration, albeit not obviously stereotypical in sequence. These included episodic sensory symptoms, such as ear pain, tinnitus, head numbness, gooseflesh, stinging sensation, visual illusion, and hallucination; motor symptoms, such as sudden jerks, involuntary blinking and muscle twitching; autonomic symptoms, such as palpitation, chest tightness and forced aspiration; and psychic symptoms such as déjà vu and unexplainable fear and anxiety associated with the above somatic symptoms. Each of the aforementioned conditions can be caused by partial epilepsy.[6] Familiarity with these presentations could help to identify a partial epilepsy.

Besides, since seizures are dynamic and evolving, clinical expression is determined by the area from which the discharge originates and the spreading sequence of cerebral electricity.[2] The various symptoms experienced by Mr. A could be attributed to the variability in the extent and pattern of the cerebral electrical spreading.[2] The corresponding locations in brain could be postcentral gyrus (tingling sensation), sensorimotor cortex (jerky movement of limbs), visual-calcarine cortex (elemental visual hallucinations), mesial temporal lobe or operculum, and occipital region (autonomic symptoms such as gastric upset, chest tightness, shortness of breath and piloerection) as well as temporal and limbic regions (psychic simple partial seizure). Hypoxic or repeated traumatic brain injury may cause diffuse axonal stretching injury and present with various unrelated simple partial symptoms, as in Mr. A.

**3. Past medical history.** Mr. A had multiple head injuries with prolonged loss of consciousness once, which increased the likelihood of posttraumatic epilepsy (PTE). PTE is referred to as a recurrent seizure disorder caused by brain injury, including head trauma and sequela of brain operation.[7] Mr. A was estimated to have a previous moderate head injury with a consciousness impairment longer than 30 minutes. In patients with moderate head injury, the cumulative 5-year probability of seizure is 1.2% and the increased risk may last up to 10 years.[8]

**4. Drug responsiveness.** With a single antiepileptic drug without antidepressants, the patient can still maintain euthymic mood for more than ten months. He experienced regular relapses of sleep disturbances each time he forgot to take clonazepam, as well as

relapses of chest tightness, forced inspiration, gooseflesh and mood fluctuation if not taking valproic acid for one to two days. He considered lamotrigine 200 mg to be the best treatment for his insomnia, labile emotion, fear, anxiety, impulsivity, and other episodic neurological symptoms. The pattern of drug response suggested the diagnosis of partial epilepsy.

The family history of depressive disorder in the patient's grandmother, mother, and aunt may suggest the diagnosis of depression. This significant genetic predisposition and the resultant diathesis in Mr. A deserves long-term follow-up to determine if these unusual presentations are related to the prodromal phase of depressive disorder.

EEG is useful mainly for localizing seizure foci and prognosticating severity. The 24-hour video-EEG monitoring was not arranged for Mr. A because of the low diagnostic yield (about 25% with scalp ictal recordings),[9] complexity, high cost, and relative unavailability in routine outpatient clinic practice. Simple partial seizures seldom reveal themselves in routine scalp EEG studies since they usually present with just nonspecific or unremarkable EEG findings even during attack because the electric discharge of partial seizure may involve only subcortical regions.[9] Therefore, EEG is only supplementary to the diagnosis which is strongly suggested from the clinical picture. We did not change the treatment strategy despite negative EEG findings.

Brain MRI, more sensitive than CT, is the clinical study of choice applied by many clinicians to all patients with posttraumatic seizure.[11] Besides, SPECT (single photon emission computer tomography), PET (positron emission tomography), DTI (diffusion tensor imaging) with MEG (magnetoencephalography) have been shown to be useful in detecting brain abnormalities associated with partial seizures, such as subtle developmental defects, temporal lobe sclerosis, traumatic brain injuries, stroke, and mass lesions. Even so, the negative result of Mr. A's MRI study could not exclude the diagnosis of partial seizures, as suggested from the clinical picture.

Serum prolactin can be measured soon after seizure attack to differentiate pseudoseizure from seizures, though it is more of research interest than of well-established clinical value. The outpatient setting also hampers its use as a timely parameter to correlate with the ictal symptoms.

Patients with psychogenic nonepileptic seizures (PNES) display abnormal behaviors as a consequence of psychological factors, rather than electric dysrhythmia in the brain. Without the aid of video-EEG, the diagnosis of PNES can be challenging even to the experienced clinicians. However, by examining carefully the seizure semiology and certain variables in medical history, PNES was not favored in this case because of onset in younger age, short duration of each spell, and stereotypical activities, which all support the diagnosis of an epileptic seizure. On the other hand, the motions usually more common in PNES attacks, such as pelvic thrusting, preparatory movement, and body stiffening[10], were absent in this patient.

In modern medical practice, over-dependence on instrumentation (such as routine EEG, video-EEG, or MRI) may result in premature exclusion of an epilepsy diagnosis. It should, therefore, be re-emphasized that a detailed chronological history for a patient with presumed epilepsy is the primary way to achieve an accurate diagnosis in up to 90% of cases.[2,4] The first task in the evaluation of epilepsy in psychiatric patients is to establish whether the nonspecific somatic manifestations possess the core features of epilepsy, i.e., unprovoked, sudden-onset, paroxysmal or episodic, and of short duration. Prolonged duration (such as 2-4 hours, or even most of the daytime) of those subjective symptoms cannot rule out the possibility of epilepsy, since they

may be caused by a subclinical nonepileptic status epilepticus. The next step is to establish the type of seizure and epileptic syndrome. For a psychiatrist, this task is not easy and a consultation with a neurologist should be considered. Mr. A had been suggested to undergo a neurological evaluation, but he refused because he felt satisfied with the treatment.

Major medications for PTE include valproate, carbamazepine, and lamotrigine. Lamotrigine is associated with the minor side-effect of weight gain. Caution, however, should be taken about the combination of valproate and lamotrigine because the serum level of the latter can be potentiated and their interaction may increase the risk of Stevens-Johnson's syndrome.

## Conclusions

Several features in the clinical course of Mr. A suggest posttraumatic epilepsy, which was apt to be underdiagnosed. These are 1) queer episodic presentations with associated somatic symptoms in accordance with the core features of epileptic seizures; 2) a history of hypoxic and moderate traumatic brain injuries; and 3) the excellent therapeutic response to muscle relaxant and anticonvulsant. This case report demonstrated the identification of an unusually presented partial epilepsy through detailed history-taking regarding the presenting symptoms and neurological manifestations suggestive of seizure disorders, although the EEG and MRI findings were not supportive of the diagnosis.

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