



Successful Electroconvulsive therapy and three year follow-up in a Bipolar I depressed patient with comorbid conversion disorder

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ABSTRACT

Background: In the Diagnostic Statistical Manual -5 (DSM-5) Conversion disorder is defined as the occurrence of neurological-like symptoms or deficits that are neither intentionally produced nor feigned. While it cannot be explained by organic pathology, it is often related to psychological events. The first case with comorbid conversion disorder and underlying long-term bipolar I depressive disorder is reported after successful electroconvulsive therapy (ECT).

Case presentation: The case reported is that of a 44-year-old housewife diagnosed with bipolar I depressed disorder that is regularly treated with Lithium, Quetiapine and Mirtazapine after 4-weeks hospitalization at a psychiatric ward in July 2009. However, she developed an episode of severe general weakness with mutism and indifference to pain or voice stimulant after a verbal dispute with her son in July 2013. She was sent to an emergency room where brain computed tomography (CT) revealed negative finding for any brain lesion. One week later, she had recurrent and worsening motor paralysis with retention of urine, dysphagia, poor personal hygiene and intermittent mutism. The patient was rehospitalized on a psychiatric ward with first time where nine ECT were administered. The symptoms and signs were dramatically improved with her walking again without assistance and resumption of independent self-care functioning. Her medications at this time were Lithium, Quetiapine, Mirtazapine, Clonazepam and Zaleplon. This psychiatric medication prophylaxis was continued for next three years.

Conclusions: Although the physiologic effects of ECT are not as yet known, it could be speculated to have an impact on consciousness by means of some dissolution and reorganization phenomenon, like is well-known treatment for catatonia. This rare case report reminds us of the role of ECT in severe motoric conversion disorders.

Keywords

Conversion disorder, Motor paralysis, Electroconvulsive therapy, Bipolar I depressed disorder, psychiatric comorbidity

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Introduction

Conversion disorder (CD), previously known as hysteria, refers to the occurrence of neurological-like symptoms or deficits, such as weakness, epileptic-type attacks, abnormal movements or sensory disturbance that are not attributable to a structural damage to the nervous system or to feigning [1]. Initially it is often mistaken for a medical condition initially. While it cannot be explained by an organic causality by our current and physical and psychological examinations, it is often related to psychological events or psychological precipitant [2]. Psychotherapy is favored if no other specific biological treatment exists [3]. However, these patients are acknowledged to be difficult to treat and exhibit a substantial hospital revisit rate to hospital [4]. The application of ECT was ignored or described as not supported in treatment of CD [5,6]. This article reports a case of a 44-year-old woman with underlying bipolar I affective disorder and clinical manifestations of a CD.

Case Report

Mrs. A was a 36-year-old senior high school graduate housewife who had complained of progressive general weakness and numbness of her four limbs after a motorcycle traffic accident in 2005. But she did not have walking disability, head injury or loss of consciousness initially. She first visited neurologist for low back pain in December 2007. However, in 2008, the patient experienced abrupt subjective declination in her muscle strength (objective grade 3 muscle power: active movement against gravity) and concomitant loss of sensation of her four limbs after she argued with her husband about their poor financial condition. Two days later, she was hospitalized at the neurological ward where laboratory examination, radiography of lumbar spine, brain Magnetic Resonance Imaging (MRI), electroencephalography, nerve conduction velocity, F wave, H reflex studies, and motor evoked potential examination all revealed negative findings. Only C-Spine MRI displayed minimal discs bulging at C5/6/7 without thecal sac narrowing. Because of the incompatibility between the symptom and recognized neurologic or general medical conditions, the psychiatrist consultant suspected that she had a major depressive disorder with psychogenic paraplegia, e.g. CD. She was referred by the neurologist to the psychiatric outpatient clinic for regular follow-up.

In August 2009, she suffered from worse depression, markedly diminished activity, poor appetite, insomnia, psychomotor retardation, loss of energy, diminished concentration, recurrent thoughts of death, delusional misidentification for objects size enlargement, nihilistic delusion during the 4-weeks of her first psychiatric hospitalization. The diagnosis of major depressive disorder was changed to bipolar I disorder based on manic symptoms of irritable mood, decreased need for sleep, pressure to keep talking, flight of ideas (easy change of speech topic), distractibility, and psychomotor agitation during the 23-day hospitalization. The symptoms meet DSM-IV TR criteria for a manic episode despite no obvious inflated self-esteem or grandiosity (criteria 1) and no excessive involvement in pleasurable activities (criteria 7). The antidepressant Bupropion was changed to lithium 1200 mg/day.

During next four-year outpatient follow-up period, she had episodic irritable mood, psychomotor agitation, hypertalkativity, racing thoughts, distractibility, insomnia, difficulties in walking often requiring a wheelchair, nihilistic delusion with complaints “no sensation or extremities numbness (anesthesia or paresthesia)”. The antipsychotic drug of Quetiapine was therefore added.

At age 44 years in July 2013, she developed an episode of severe general weakness with mutism and indifference to pain or voice stimulant after a several verbal disputes with her son. Mrs. A was sent to emergency room where brain CT revealed negative finding for brain pathophysiology. One week later, she developed severe psychomotor retardation, motor paralysis, inability to walk or eat independently, retention of urine, poor sleep, poor hygiene, and intermittent progressive mutism. The patient was hospitalized a second time with the diagnosis of bipolar I disorder, most recent episode depressed, severe with mood congruent psychotic features. It is important to note that the chief symptoms could not be explained by a medical condition, substance abuse, other psychiatric disorder, culturally sanctioned behavior, or catatonia. The psychiatrist added the additional diagnosis of comorbid motor CD with symptoms of impaired coordination, paralysis, dysphagia, aphonia, and urine retention. She did not have fever, tachycardia, or high CPK levels at that time.

Urinary and intravenous catheters were inserted. Standard bitemporal ECT was emergently

performed because of her inability to eat or to consume drugs. ECT was performed using the Thymatron System IV machine (Somatics, Inc., Lake Bluff, IL, USA) with a bipolar brief pulse square wave. The stimulus parameter of ECT was as follows: a pulse width of 0.5 ms, a frequency of 60 Hz, and a constant current of 0.9 A. Seizure duration lasted over 25 second. She was treated with nine sessions of ECT. Mrs. A's CD symptoms were all dramatically improved to allow her to comply with treatment again. The patient had ongoing and regular follow up at the psychiatric outpatient department with medication prophylaxis of lithium 1200 mg/day, Quetiapine 500 mg/day, Mirtazapine 30 mg/day, Clonazepam 4mg/day, and Zaleplon 10 mg/day for the next three years. She could walk without any assistance and regained independent housewife functional capacity. Because of recurrent manic attacks at 28 months and 36 months respectively, the patient was hospitalized on a psychiatric ward.

Discussion

To our knowledge, few cases of successful ECT treatment of motor CD have been reported. The first case was that of a 61-year-old man with a severe CD that included paralysis of his right hand and consequent disuse atrophy for 11 months. He was treated with 19 sessions ECT for prevention of loss of his hand and was followed up for one year [7]. The second case was that of a 33-year-old patient with a fluctuating hysterical tetraplegia and an uncontrollable muscular hypertonia, which had started three years earlier. Treatment with a total of 35 ECTs improved the patient's consciousness and walking ability. However, this patient obtained only partial remission with fluctuation and recurrence later [8].

Mrs. A met DSM-IV TR CD criteria A: The patient has ≥ 1 symptoms of altered voluntary motor or sensory function that suggest a neurological or other general medical condition, criteria B: Psychological factors are judged to be associated with the symptom or deficit because the initiation or exacerbation of the symptom or deficit is preceded by conflicts or other stressors, criteria C: The symptom or deficit is not intentionally produced or feigned, criteria D: The symptom or deficit cannot, after appropriate investigation, be fully explained by a general medical condition, or by the direct effects of a substance, or as a culturally sanctioned behavior

or experience, criteria E: The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation, and criteria F: The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of somatization disorder, and is not better accounted for by another mental disorder.

Mrs. A also had symptoms of paralysis, gait disorder, dysphagia, dysphonia, indifference to stimuli, and clouded consciousness which were all temporally related to a psychological stressor. Mrs. A presented severe regression after she vividly expressed her distress. Regression is a ubiquitous phenomenon of CD in psychodynamic psychotherapy and psychoanalysis. In CD patients with adequate character structure, this regression, when handled effectively by the psychotherapist, might ultimately lead to verbalized thoughts and feelings and get a gradually strengthening alternative [9]. The author speculates that the circumscribed amnesia induced by ECT further provides a 'escape' opportunity for this stress and that the therapist successful handling of her regression may allow the reorganization by cognitive psychotherapy after ECT to reform an adequate and manageable approach to life situations.

Mrs. A also had obvious temporal related psychological stressors, motor paralysis, and altered consciousness. In contrast to the two cases in the literature, Mrs. A experienced an exacerbated episode of chronic CD, previous psychiatric history of bipolar I affective disorder, and two recurrent manic episode in 3-year follow-up course.

Some cases of successful ECT treatment of "possible" motor CD have been described. The first report was that of a 29-year-old woman with a hysterical paralysis of the lower extremities after two car collisions for two months [10]. The second case was that of a 31-year-old woman who had been confined to a wheelchair with violent, grossly bizarre, psychogenic movement for almost ten years. Treatment with total of 16 ECTs improved the patient's walking ability sufficiently that she could return to work and travel over the 2.5 years follow-up period [11]. These two patients were marked as "possible" motor CD because they had history of head injury with experienced traumatic confusion after car accidents. Retrospectively it is difficult

to assess the relatively etiological contributions made by head injuries or psychosocial problems.

The traditional label CD refers to a hypothesis based on a psychological etiology. Historically, psychological and emotional factors, such as psychological trauma, conflict or distress, have been suggested as causal factors of CD. DSM-IV TR diagnostic criteria of CD includes the importance of the temporal relationship that psychological factors precede the initiation or the exacerbation of the symptoms [2]. However, the criterion B has been omitted in the DSM-5 [12,13]. The DSM-5 provided the additional denomination of “functional neurological symptom disorders (FNSD)”. The once essential diagnostic criterion for the identification of stressors around the time of symptom onset has been downgraded from an essential to a supportive criterion and has been replaced by criteria emphasizing the need for positive diagnostic symptoms and signs [14,15]. In the case series report by Regny, *et al.* a relevant psychogenic factor was explicitly mentioned in only 43% of the CD cases. It is felt that the DSM-IV TR criterion of “psychogenicity” is highly problematic in clinical practice [16]. A pain complaint was associated in half of the cases. They suggest a close relationship between conversion disorder and unexplained chronic pain. In Nicholson, *et al.* study [17], they used the Life Events and Difficulties Schedule in the year before symptom onset to support the Freudian theory that physical symptoms of CD could provide ‘escape’ from stressors in only some patients. They found that minority (10%) of CD patients had no identifiable severe life events. Merkler, *et al.* suggest that CD is not an acute, time-limited response to stress, but rather that CD is a manifestation of a broader pattern of chronic neuropsychiatric disease [4].

The case reported herein illustrates the necessity of a multidisciplinary approach to the diagnosis and management of severe CD based on the clinical manifestations [18]. The patient presenting with a CD very often receives multiple examinations and most of them are initially admitted to the neurology ward, rarely to the psychiatric ward [19]. Combined consultation (medicine and psychiatry) is a useful first step to understand patients. In the emergency situation of patients with suspected CD, doctors always try to identify a possible organic disease that could be attributable to a structural damage to the body. After the failure of biological examinations and the exhaustion of psychotherapeutic strategies,

the management starts with reassuring the patient and families that the patient’s major physical complaint might have a psychological cause [3]. Next the staff initiates to coordinate multiple disciplinary treatments for the patients, including physical, neurological, rehabilitational, psychological, occupational, recreational, and family therapy [20].

The cause of conversion symptoms in this patient doesn’t relate to drugs or physical problems, but it is a CD diagnosis which needs to be differentiated from catatonia. Catatonia is a state of apparent unresponsiveness to external stimuli in a person who is apparently awake. Core features of catatonia are stupor/motoric immobility/catalepsy/waxy flexibility, excitement, negativism/mutism, posturing and echolalia/echopraxia, Mrs. A had the signs of motoric immobility and mutism, and had dissimilar signs of motor paralysis and weakness versus waxy flexibility, negativism, posturing, excitement or echopraxia.

ECT is very well established treatment for patients with major depression, delusional depression, bipolar disorder, schizophrenia, catatonia [21-23]. However, CD is not an accepted indication for the use of ECT, reflecting the previous concept of “psychogenicity” as an etiological factor in CD [5, 6]. As electrical treatment was strongly criticized after World War I, ECT has not been used frequently to treat hysterical patients in contrast to depressive states. However, a few case reports or small series reveal some interest of ECT in CD either presenting with motor deficit or pseudoepileptic seizures [24]. Although gross neurostructural deficits do not account for the patients’ deficits or symptoms, studies focusing on potential neurobiological (i.e. functional neuroanatomic/neurophysiological) findings among individuals with various forms of FNSD imply that neural networks and neurophysiologic mechanisms may mediate “functional” symptoms in the era of DSM-5 [25,26]. Aybek, *et al.* suggests a mechanism linking emotions to motor dysfunction by event related fMRI task [27]. Neuronal areas seem to be involved in the pathogenesis, maintenance or as a result of motor CD as evidence on imaging studies [28]. Some case studies of transcranial magnetic stimulation in the treatment of FNSD are critically reviewed but evidence controversy [29]. Suzuki, *et al.* reported a case with hysteria presenting as a prodrome to catatonic stupor in a depressive patient that resolved with ECT [30]. The author agrees that “ a prodrome to

catatonic stupor “ would be more acceptable than “CD” in the era of DSM-IV TR. By the large, ECT resolved Mrs. A’s depression and CD which might be also taken as “ a prodrome of catatonic stupor”. This report of the CD patient with successful outcome from ECT might enhance the role of psychiatrists in diagnosing and managing CD [31].

Conclusion

Although the physiologic effects of ECT are not as yet known, it could be speculated to

have an impact on consciousness by means of some dissolution and reorganization phenomenon, like is well-known treatment for catatonia [23,32]. ECT could be a rapid and effective treatment for the patient with severe motoric CD, even in an individual with a previous diagnosis of bipolar I depression. Consultant psychiatrists need to be aware of this therapeutic option.

Declaration of interests

The authors report no conflicts of interest.

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