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Medical Group

Archives of Depression and Anxiety

ISSN: 2455-5460 DOI CC By

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Dates: Received: 18 August, 2017; **Accepted:** 28 August, 2017; **Published:** 29 August, 2017

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Case Report

Hypochondriacal delusion in an elderly man with good response to electroconvulsive therapy but complicated with febrile reaction

Abstract

In this case report, we present an elderly gentleman with prominent somatic delusions associated with depressive features. He was diagnosed to have severe depressive episode with mood congruent psychotic symptoms and was resistant to both pharmacological and psychological intervention. There was a differential diagnosis of delusional disorder, somatic subtype, resulting in a secondary depressive episode in view of his persistent somatic delusions which appeared more distressing and more prominent than his mood symptoms. Chronologically, his depressive symptoms had been there first, followed by more and more somatic complaints therefore he was concluded to have treatment resistant depression. Electroconvulsive therapy (ECT) was started with significant improvement especially for his hypochondriacal delusions. However, the ECT was complicated by recurrent febrile reaction which had close temporal relationship with each ECT session but was noted to be benign in nature. This patient has reminded us that psychotic depression is common in the elderly and may have atypical presentation. ECT is an effective treatment option for those resistant to pharmacological and psychological intervention. It has resulted in a fast and complete recovery for hypochondriacal delusion. Febrile reactions might be associated with ECT and it is important to rule out other causes.

Introduction

Major depressive disorder with psychotic features is more likely found in elderly than adulthood [1]. The delusions are commonly mood-congruent, including delusions of guilt, delusions of deserved punishment for moral or personal inadequacies, delusions of nihilism, somatic delusions and delusions of poverty. The body is often a focus of delusion in psychotic depression [2]. Recent research in four academic centers showed that the diagnosis of depression with psychotic symptoms was missed in about 25% of the cases if the psychotic symptoms were more prominent than a disturbed mood [3]. Hypochondriacal delusions as part of a severe depression might be under-diagnosed as some patients did not have depressed mood or loss of interest as the prominent features [4]. It was stressed that a mood disorder with hypochondriacal delusion especially in the middle age and elderly could mimic several psychiatric conditions like hypochondria, delirium, catatonia and anxiety disorder, depending on the symptoms that were most prominent [5].

In this case report, we present a case with prominent somatic delusions associated with depressive features. There were two interesting points in this case. The first point was about the diagnosis of the patient. There was a period when the delusion became more predominant comparing with the depressive symptoms. This affected the choice of treatment for the patient when he was found to be resistant to the antidepressants and antipsychotics given to him. The second point was about the recurrent benign febrile reactions while he was receiving ECT. The benign febrile reactions had close temporal association with each ECT session.

Case Report

A 67-year-old man who was still working as an insurance agent presented with low mood, low self-esteem, insomnia, anxiety symptoms and fleeting suicidal ideation for four months after an episode of sinusitis with persistent tinnitus. He did not have any significant medical and psychiatric history except a small shadow around his right kidney found during body check. He sought treatment from general practitioner but the symptoms persisted. His family suspected that he had depression and brought him to see private psychiatrist. He was diagnosed to have depression with anxiety symptoms and was put on sertraline 125mg/day, mirtazapine 15mg/day

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and clonazepam 1mg/day. His mental state deteriorated after he had received quadrivalent (IIV4) inactivated influenza vaccine the same month and he presented with lower limb weakness, reduced motivation and body tremors. He presented to the Accident and Emergency Department with sudden onset of escalating anxiety, shortness of breath, palpitations and feeling loss of control the next day. The attacks had a quick onset without particular trigger. The whole duration of attack lasted for about twenty minutes. He was admitted to medical ward subsequently, where there were further attacks. His conscious level remained intact during the attacks but had difficulty controlling himself. Neurologist ruled out seizure. Organic workup including renal, liver and thyroid functions tests, complete blood count, assays of cardiac enzymes and creatine phosphokinase (CPK), electrocardiogram, electroencephalogram and magnetic resonance imaging brain scan were unremarkable. He was transferred to psychogeriatric ward for further management. The provisional diagnosis was moderate depressive episode with anxiety symptoms and panic attacks

He kept ruminating about his poor physical health and other somatic complaints. He described having a gust of air circulating in his body from time to time. He felt himself getting thinner, his muscles eroding, his stomach slowing down and his throat narrowing. He was strongly convinced that there was an obstruction in mid esophagus even though he was told about the normal finding of an oesophagogastroduodenoscope done not long ago. He complained that he was unable to swallow.

Other somatic foci included urinary frequency and constipation that he believed he had sinister illnesses causing all his distress. He firmly believed he would die soon from his current physical illness. He was hopeless and believed that his somatic symptoms were genuine and not due to his depression. His appetite was poor and his sleep was fragmented. He was pervasively anxious but not panicky, and that affected his interest level. There was no psychomotor retardation. Sertraline was gradually stepped up to 150mg/day and mirtazapine to 45mg/day. In view of his unshakable belief regarding his physical condition, they were regarded as delusional and his diagnosis was revised to severe depressive episode with mood congruent psychotic symptoms according to the Tenth Revision of the International Classification of Diseases and Related Health Problems (ICD-10). He was started on quetiapine which was gradually titrated to 200mg/day. We had been cautious regarding the speed of titration of psychotropics in view of his age and high sensitivity to side effects of medications. Improvement was noted in his sleep, appetite, mood and motivation to take part in ward activities. However, his delusional belief persisted. Sertraline was replaced with venlafaxine which was titrated to 225mg/day and quetiapine was replaced with olanzapine increased to 20mg/day. The dosages of the psychotropics used were within recommended dosage for his age and physical state. Psychological intervention was offered to him with a view to boosting insight and acceptance towards his physical complaints. He was still preoccupied with and distressed by his physical complaints albeit able to follow ward routine. He believed his physical condition was deteriorating and he would die soon. He had developed a systematized belief that the gust of air travelling inside his body was causing stomach bloating, vein engorgement, body swelling, muscle wasting and blockage to his intestines and throat. He was worried that the symptoms might further deteriorate and become fatal. He demanded to have further investigations. This had affected his mood and he had fleeting suicidal idea. He was observed to be anxious and worrying all the time though he was still able to befriend with co-patients and watch them play chess in ward. He was able to finish his meal and had no major problem in sleep at night.

There was doubt about the psychiatric diagnosis of this patient. In view of his persistent somatic delusions which appeared more distressing than his mood symptoms, a differential diagnosis of delusional disorder, somatic subtype, resulting in a secondary depressive episode had been considered. His presentation was atypical of psychotic depression at this stage of treatment as his depressive symptoms were not florid other than sense of hopelessness and there was no psychomotor retardation all along. His anxiety symptoms were more prominent and had been protective towards him as he would consciously force himself to eat more to prevent from dying from malnutrition. Chronologically, his depressive symptoms had been there first, followed by more and more somatic complaints. The combination of two antidepressants and augmentation with antipsychotics might have some effect on his depression although they could not bring a remission. Somatization disorder with marked depression and anxiety was unlikely in view of the relatively short duration of physical symptoms. A second opinion confirmed treatment resistant psychotic depression. Major depression with a poor or unsatisfactory response to two adequate (optimal dosage and duration) trials of two different classes of antidepressants has been proposed as an operational definition of treatment-resistant depression. ECT was started after detailed explanation to the patient and his wife. Six sessions were booked for him. The dose titration method was used to estimate the patient's seizure threshold. Bilateral ECT was administered twice weekly. Table 1 shows the details of the treatment parameters of all the ECT sessions.

192 milliCoulombs and 1.0 ms pulse width were used in the first session, resulting in 30 seconds motoric seizure and 30 seconds on the EEG recording. Thiopentone was used as the induction agent and suxamethonium was given as the muscle relaxant for all ECT sessions. There was gradual improvement in his mood and motivation after the first session. 288 mCoulombs was used in the third session. A significant

Table 1: Treatment Parameters and Fever Characteristics.

Treatment no.	1	2	3	4	5	6
Stimulus power (mC)	192	192	288	288	288	432
Thiopentone (mg)	120	120	100	100	100	100
Fentanyl (mcg)	40	40	40	40	40	40
Suxamethonium (mg)	50	50	50	50	50	50
EEG seizure (s)	50	30	46	48	20	32
Fever	/	/	/	39.6	39.6	38
Onset of fever (hour after ECT)	/	/	/	12	5	24
Duration of fever (hours)	/	/	/	24	26	29

ECT Machine: MECTA Spectrum 5000Q Stimulator with ULTRABRIEF 0.3ms Pulse Width Option (Mecta Corp., Lake Oswego, OR)

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reduction in his somatic complaints was noted after the third session.

Hyperthermia after the fourth, fifth and sixth sessions of ECT

After the fourth session of ECT, he was noted to have high fever with tympanic temperature of 39.6 degree Celsius about 12 hours afterwards. The patient only complained of sore throat. Laboratory findings revealed an elevated white blood cell count of 12.9 (reference range 3.7-9.3 10^9/L). The assays for troponin-I and amylase were within normal range. Urine and blood culture found no growth of bacteria. Nasopharyngeal swab was negative for influenza A and B. Chest X-ray showed no signs of infection. We empirically prescribed amoxicillin/clavulanic acid 1g twice daily. The white blood cell count became normal. He had taken paracetamol 1000mg on Day 1 of fever. Fever subsided on Day 2.

The fifth session of ECT took place on scheduled three days after the last session. Five hours after the ECT, the patient developed recurrent fever with a tympanic temperature of 39.6 degree Celsius. Laboratory findings showed a raised white blood cell count of 9.7 (3.7-9.3x10^9/L) and a neutrophil count of 6.7 (1.8-6.2 10^9/L). C-reactive protein was elevated to 95.8 (<5mg/L). Alanine Aminotransferase (ALT) was raised at 159 (<41 IU/L) and alkaline phosphatase (ALP) was mildly elevated at 130 (40-129 IU/L). Renal function tests, CPK, ammonia level and clotting profile were normal. ALT dropped to 76 IU/L and ALP normalized after two days. Hepatitis screen was performed and the results came back to be negative. Findings from ultrasound abdomen were unremarkable. He took paracetamol on Day 1 of fever. Fever subsided completely on Day 2. He received the sixth session of ECT as scheduled. 432 milliCoulombs was used. The patient had a kick of fever at 38.5 degree Celsius the next morning. He took paracetamol on Day 1 of fever. It subsided the next day. There was no other physical complaint.

After a total of six sessions, he recovered completely and his hypochondriacal delusion had disappeared. The scoring of visual analog mood scale [6], increased from 3/10 before ECT to 8/10 after ECT. He was discharged three days after his last ECT session and maintained on olanzapine 20mg daily, venlafaxine 225mg daily, mirtazapine 45mg daily and valium 2mg daily.

Discussion

This case report presented an elderly gentleman who had an atypical presentation of psychotic depression and was resistant to pharmacological and psychological intervention. When ECT was started for him, there was significant improvement especially for his hypochondriacal delusions. However, the ECT was complicated by recurrent febrile reaction and raised liver function test (LFT). The cause of fever was suspected to be infective in origin as the patient complained of sore throat and there was a raised while blood cells noted. However, the fever receded rapidly and reoccurred after another session of ECT. A febrile reaction induced by ECT is rarely reported as an adverse effect. To date, there have only been a few case reports on fever after ECT for the treatment of bipolar disorder [7-9], depression [10,11] and catatonic schizophrenia [12]. Among them, three cases were associated with organic brain conditions, namely cerebral palsy [7,12] and learning disability [9] which was absent in our patient. Another major difference was that all the other patients had fever after the first session of ECT [7-12], but for our patient, it only occurred after the fourth session. The differential diagnoses for febrile reaction in our patient include concomitant infection, neuroleptic malignant syndrome and benign fever induced by antipsychotic agents. Complications associated with the use of general anaesthesia have to be considered. It is possible that anaesthetic agents may induce febrile reactions. Malignant hyperthermia triggered by other anaesthetic agents, suxamethonium in particular, is also possible. However, associated features such as muscle rigidity, rhabdomyolysis or elevated CPK were absent in this patient. The associated transient deranged liver function test required ruling out of acute hepatitis.

The exact mechanism of how ECT induces febrile reactions is unclear. One postulation is derived from reports of febrile reactions after spontaneous seizures [13,14], which may be explained by ictal involvement of the hypothalamus [15], where the thermoregulatory centre is located. Innate immune reaction with production of pyrogens [16], after electrical stimulation could result in febrile reactions. The febrile reaction in our patient was associated with an inflammatory response with raised CRP level. The raised LFT was a transient phenomenon and might be a reaction of the liver as part of the inflammatory response. Neurochemical changes in the thermoregulatory centre associated with electrical stimulation may be a possible mechanism for fever associated with ECT. It was hypothesized that post-ECT hyperthermia may be a correlate of successful stimulation of brain structures (e.g., the hypothalamus) involved in mood and thermal homeostasis which could be associated with symptom improvement [10].

The febrile reaction associated with ECT is benign in nature according to the existing case reports. Therefore ECT was continued in our patient after other causes of fever had been ruled out and followed by resolution of fever. At the same time, continuous improvement of symptoms had been observed after each ECT session. The patient eventually received six session of ECT and had returned to his premorbid state after the last session.

ECT is recommended for depression which had not responded to multiple treatments [17] and it is often effective for severely depressed patients who have not responded to multiple medication trials [18]. There were a number of case reports on patients with hypochondriacal delusion improving after ECT [4,19,20]. ECT was found to play a significant role in the modulation of the immunological system, including psychiatric illness [21,22]. It is believed that the protracted effects of stressors and cytokines may play an important role in the pathophysiology of amplification of somatic complaints [23]. Recognizing this type of depression in the elderly population would speed up treatment and prevent unnecessary, costly test and delay. Many practitioners do not consider referral for ECT before multiple medications have been tried, a process that may span many months or even years and leave the patient seriously ill, suffering, and dysfunctional, for a prolonged period [24]. Longer duration of

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depressive episode has been associated with greater treatment resistance, another reason such delay may be inadvisable [25]. Concern about cognitive effects is the main reason ECT is not more widely prescribed for severe depression [26]. Semkovska and McLoughlin [27], performed a systematic review and meta-analysis of 84 studies (N =2981) that used standardized tests to assess cognition in ECT patients. They concluded that cognitive abnormalities associated with ECT are mainly limited to the first three days posttreatment. Pretreatment functioning levels were subsequently recovered. The cognitive impact of ECT, particularly retrograde amnesia, should therefore be considered an important, but not overriding, tolerability issue.

This patient has reminded us that psychotic depression is common in the elderly and may have atypical presentation. ECT is an effective treatment option for those resistant to pharmacological and psychological intervention. It has resulted in a fast and complete recovery for hypochondriacal delusion. Febrile reactions might be associated with ECT and it is important to rule out other causes.

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