

CASE REPORT**A Diagnostic Dilemma:
Catatonia, Serotonin Syndrome and Parkinsonism**

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Abstract

Catatonia may concomitantly occur with other psychiatric diagnoses such as Major Depressive Disorder, however problem in diagnosis may arise due to the overlapping features with other problems such as serotonin syndromes, neuroleptic malignant syndromes and Parkinsonism. Despite the diagnostic dilemma and lack of diagnostic tools, the clinical correlation between the carbon monoxide poisoning and the late-onset development of the Parkinsonian features is the highlight of this report.

Keywords: Catatonia, Parkinsonism, Carbon Monoxide Poisoning

Introduction

Catatonic syndromes are a diagnostic conundrum and always offer a therapeutic challenge as well. Catatonia is an associated feature in major psychiatric diagnoses, as well as a prominent overlapping features in neuroleptic malignant syndrome, akinetic parkinsonism and representing motor features of serotonergic syndromes [1,3]. The list of the possible cause of similar presentations are vast. Among others, carbon monoxide poisoning is known to cause delayed onset Parkinsonism [2].

Among the said differentials, there are few common and similar presentations between them, hence the dilemma. Similarity of the presentations are usually of the motor

symptoms such as stuporous nature of the patient, rigidity, tremor and bradykinesia.

Despite the similarity, the option and response towards treatment are quite different. While electroconvulsive therapy is a known treatment option in catatonic state, it may not be the case for the state of serotonergic syndrome and Parkinsonism.

Case report

Mr LHC, a 58 years old Chinese male, with background history of methamphetamine abuse, presented to the hospital following unconsciousness episode at home. This is his first psychiatric contact. He allegedly attempted to suffocate himself by burning up charcoal in his bedroom. Upon arrival in the

emergency department, he was documented to have Glasgow Coma Scale score of 10/15.

He was previously well until about a month ago when he started to display depressive symptoms which being described by his friend as appearing to be sad, reduced oral intake and lethargic. Since separation from his partner about few months ago, he had been staying alone. He also lost his part-time job recently. He reportedly not able to pay the rental fee of his house for the past few months. His family stated that his partner had used up a large part of his saving and about RM 100,000 had been spent in the past 6 months. No manic or psychotic features were established.

Within a week prior to the admission, Mr LHC performed 3 self-harm attempts. He attempted the first 2 self-harm by hanging himself. He was saved by his friend who accidentally noticed his act but he was never brought to the hospital. His 3rd attempt was by charcoal smoke inhalation while locking himself in his room. He also took methamphetamine pill prior to the self harm attempt. He was later found unconscious by his friend and the duration of exposure to the charcoal smoke was not able to be established.

Upon presentation to the hospital, Mr LHC was drowsy and had impairment of consciousness level. The possibilities of major depressive disorder as well as underlying asphyxiation injury, cervical injury and carbon monoxide poisoning were being considered. Limited physical findings were available at that point. Blood investigations, arterial blood gases test and computerised tomography scan (CT scan) of the brain and cervical were done. Asphyxiation injury, carbon monoxide poisoning and cervical injury had been ruled out by the respective departments.

Unfortunately, no carbon monoxide measurement had been done during his initial ward stay.

Mr LHC was transferred to the psychiatric ward for his persistent depressive features and strong suicidal risk.

In psychiatric ward, examination reveals that he is alert but not forthcoming and guarded on his problems. Presence of psychomotor retardation is noted. He admitted of having low mood, reduced appetite and poor sleep for the past 2 weeks.

He was initiated on Escitalopram 5mg every night and titration of dose up to 15mg every night was done in a period of 10 days.

Clinically, he was deemed to be deteriorating and in a catatonic state as he appear to be stuporous, mute and posturing. A consideration of serotonin toxicity was later being considered due to the recent introduction of serotonergic drug despite the overlapping features of catatonia and the serotonin syndrome. Rigidity and stiffness were also present and the differential of Parkinsonism was also considered. Escitalopram was stopped immediately and symptomatic treatment as well as benzodiazepine therapy were initiated.

Despite ongoing symptomatic treatment for 8 days, patient remain to be in a similar state and clinical features of Parkinsonism became more noticeable. Clinically, patient is found to have cogwheel rigidity of the upper limbs, increased muscle tone, pill-rolling hand movement, resting tremor, bradykinesia, gait instability and masked facial expression.

Discussion

Parkinsonism is a clinical diagnosis that consist the features of tremor, rigidity

bradykinesia and impaired postural reflexes. Parkinsonism can result from several causes and may mimic the presentation of catatonic state in psychiatric patients [3].

Despite the rising incidence of carbon monoxide (CO) poisoning, it is often a challenging diagnosis to be made [3, 4]. The subtle and non-specific symptoms of CO poisoning made it even unrecognizable in the mild state and even more difficult to establish in the presence other concomitant cause. It is often believed that the severity of symptoms are proportionate to the level of CO in the blood. However, the predictability is inaccurate and possibility of delayed sequelae also make the diagnosis even more challenging and may lead to underdiagnosis of such potentially catastrophic but treatable diagnosis [2,4,5,6].

CO poisoning is potentially emerging as threat to health as it is cheap and simple method to commit suicide. It is accounted for 0.22 per 100000 suicides in 1996 and 6.48 of 100000 suicides in 2006 in Hong Kong[6]. Neuropsychiatric sequelae occur in up to 50% of all patients who sustain toxic level of CO [4].

Earlier detection facilitates in the treatment of patient. Lacking of the diagnostic tools often lead to reliance in clinical diagnosis [4,9]. Radiological intervention such as magnetic resonance imaging (MRI) may detect acute as well chronic changes in the brain and findings include necrosis of the globus pallidus, deep white matter oedema and less, frequently oedema of the deep seated nuclei such as putamen, caudate nucleus, thalamus and hippocampus [2,6,7].

Delayed sequelae from CO poisoning is devastating and occurs in 10-43% of person recovering from acute exposure [8]. Treatment options focusing in oxygen

therapy and the respective sequelae. Hyperbaric oxygen therapy is recommended for hastening the removal of CO from the circulation however the efficacy and cost efficiency of such treatment had long been discussed [9]. While the use of anticholinergic drug is ineffective [2], the use of L-dopa also had been disappointing [2,8]. However, a small study with another centrally acting dopaminergic agonist, bromocriptine, displayed improvement [8].

The devastating outcome of delayed sequelae such as Parkinsonism is of variable prognosis, in mild CO poisoning gradual and spontaneous recovery is expected within 1-3 months [2, 8] while a certain percentages still having a residual Parkinsonism features after more than 6 months [8].

Uncommon occurrences in Malaysia and non-specific symptoms made it difficult to recognise but nonetheless effort has to be done in patient who committed suicidal attempts by the mean of charcoal poisoning. They already succumbed to depressive state and lived with a stressful life, lets hope that we manage to treat them right.

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