Hong Kong Journal of Psychiatry (1999) 9 (1), 26- 28

CATATONIA MIMICKING NON-CONVULSIVE STATUS EPILEPTICUS: A CASE REPORT

MIAO YIN-KING

Summary

Catatonia is a neuropsychiatric syndrome associated with a host of psychiatric, drug-induced and medical conditions. This report describes a 38 year-old Chinese man who presented with episodic stupor, stereotypic lip smacking and urinary incontinence suggesting temporal lobe seizure. The diagnosis of catatonic schizophrenia was made subsequently with the documentation of first rank symptoms. The stuporous episodes resolved with antipsychotic treatment. The relationship between catatonia syndrome and epilepsy and the use of electroencephalogram in the differential diagnosis are discussed.

Key words: catatonia, epilepsy, electroencephalogram

INTRODUCION

Catatonia has been a syndrome of diverse aetiologies, was first described by Kahlbaum (1874-1974) (Taylor,1990). Up to recent times, the condition had been viewed as a subtype of schizophrenia despite caveats by both Kraepelin and Bleuler that this notion was only justified if other symptoms of schizophrenia were present (Lohr,1987). Contemporary authors view catatonia as a syndrome which occurs in various neurological conditions (e.g. encephalitis, epilepsy, Wernicke's encephalopathy), metabolic conditions (e.g. Lupus erythematosus, uremia, hepatic dysfunction); psychiatric disorders (e.g. bipolar affective disorder, schizophrenia) and can also be the manifestation of drug side effects (e.g. neuroleptics, amphetamine, cortisone). From a neurobiological point of view, it has recently been considered to be a frontal lobe syndrome possibly resulting in an interplay between the frontal lobe, the basal ganglia and the brainstem (Taylor,1990). However the neurobiology of catatonic syndrome is still unclear (Gelenberg,1976).

The clinical presentation of catatonia is as diverse as its aetiologies. The essential features involve cataleptic phenomenon, akinesia, mannerisms, stereotypies, automatic obedience, posturing and mutism (Kahlbaum,1973). Over 40 different signs and symptoms have been described (Lohr,1987). The most common subsyndrome is stupor presenting with immobility, mutism, posturing, rigidity, and staring (Kahlbaum,1973; Rosebush,1990). The patient is inaccessible, withdrawn and retarded, sometimes with stereotyped behaviour resembling automatism.

Nonconvulsive status epilepticus(NCSE) is a clinical phenomenon in which epileptic manifestation range from disturbed motor behaviour, such as motor perseveration, tremulousness, month movements, gait disturbances and bradykinesia to a wide variety of abnormal subjective experience, such as fear, mood fluctuation, flights of ideas and perceptual disturbances. The manifestation replace the fit proper as the ictal manifestation of the epilepsy. Clinical diagnosis can be difficult when an altered mental state or behavioural and psychiatric manifestation are the major ictal manifestation. The seizures can last from minutes to days. All are accompanied by ictal electrical activity of the brain. The subjects usually have varying degree of clouding of consciousness and amnesia for the period of fit. Clinically they may appear slow and confused, occasionally even stuporous(Lishman,1987). NCSE includes generalised and partial forms, nonconvulsive generalised seizure status can be further divided into typical petit mal status, absence seizures with slower or atypical spike-wave or multiple spike-waves discharges, which usually occur in later life, and generalized nonconvulsive seizures secondary to partial epileptic status of temporal or frontal lobe origin (Lee, 1985).

Since catatonic syndrome and epilepsy share certain clinical features (Lee,1985), the differential diagnosis may be difficult. Here we report the case of a catatonic patient whose clinical presentation strongly suggested 'psychomotor seizures' while a diagnostic work-up and longitudinal observation revealed a schizophrenic psychosis.

CASE REPORT

Mr W, a 38 years old single Chinese man, presented to the Accident and Emergency Department for a suspected 'convulsive' episode lasting for a few minutes. He was found by police sitting in the street with a 'stuporous' state, urinary incontinence and limbs twitchings. No focal neurological signs were found, he recovered spontaneously in the A&E Department. Nonetheless, Mr W was admitted into a medical ward, he again lapsed into mutism with a dazed expression. No further limbs twitchings were observed. His movement and vegetative functions were decreased to an extent that tube feeding and urinary catherisation were required. He spontaneously recovered within few hours. Skull and chest X Ray, computerised tomography (CT) scan of brain, complete blood

picture, serum glucose and electrolytes were within normal limits. No electroencephalogram (EEG) was done at that time.

Two days later, Mr W was transferred to the psychiatric unit for further assessment, where no psychotic features, stupor or 'convulsion' were observed. His mood was euthymic, his speech was aprosodic and stilted. Speech mannerisms included the inappropriate use of words and grammatical constraints. Most of the time he appeared aloof and solitary.

In the background, Mr W was brought up by his grandparents. His mother remarried after the death of his father when he was very young. He described himself to be introverted and hardworking. He completed a computer science degree in Canada and subsequently worked as a programmer. He quitted the job a year before the index admission without any particular reason. He lived alone in a rented flat in virtual social isolation.

A review of his past psychiatric history revealed that Mr W had one suicidal attempt in Canada at the age of 21, for which, he received counselling. He returned to Hong Kong when he was 25 years old. After a month, he presented with an episode of hearing non-existent voices associated with involuntary twitching of body. Electroencephalogram (EEG) and computerised tomography (CT) scan of brain performed by private practitioner were normal. He was diagnosed as suffering from schizophrenia and treated with trifluoperazine. Medication was gradually reduced after 2 years and he lost for follow up 3 years later. Mr W reported three similar 'convulsions' in the past ten years, with resulting in hospitalisation. No identifiable causes were found. At the time of the index admission, he was not on any medication.

Mr W had stayed for two weeks in the index admission. The provisional clinical diagnosis were epilepsy and schizoid personality. He was not on any medication upon discharge. He was readmitted 3 months later with a similar episode of mutism, akinesia, stereotypic lip-smacking and urinary incontinence. An EEG and then sleep EEG showed no ictal discharges. During this hospitalisation, carbamazepine 200mg thrice a day was tried for suspected psychomotor seizure. Despite treatment, a further attack was noticed one week later. An ambulatory EEG monitoring for over 3 days did not reveal any evidence of epileptiform activities. Subsequent investigations included magnetic resonance imaging (MRI), single photon emission computed tomography (SPECT) of brain and a further sphenoidal EEG . All results were normal.

During this admission, Mr W was noticed to have disorganised speech, incongruous affect and some vague delusional ideas of religiosity. The working diagnosis was changed to catatonic schizophrenia, carbamazepine was tapered off and trifluoperazine 5mg a day was prescribed. Mr. W responded with satisfactory resolution of symptoms and was subsequently discharged. During outpatient assessments, he recalled the episodes of 'stupor' experienced as 'frozen' state. He claimed to have full awareness of his surroundings during the attacks although he could not response voluntarily or initiated any movements, as though his activities were 'blocked'. He revealed that he had had several past episodes of involuntary utterance, mutism and behavioural disturbances; which appeared to him as 'automatic', i.e., the actions were performed without him being aware of what and why were happening. He also admitted passivity feelings during these attacks. He eventually disclosed the experience of hearing 'non-existent' voices in third person from time to time.

DISCUSSION

In the beginning Mr W's catatonia was unrecognised and the working diagnosis for Mr.W was non-convulsive status epilepticus. On one hand, this resulted from the expectation that patients with catatonia must be totally mute and immobile for considerable time, or that when cataleptic, their posture must be bizarre. In fact most catatonic patients speak and mobile, and when cataleptic, they maintain a mundane posture (Taylor,1990). Besides, the initial episodic attacks of 'stupor', 'limb twitchings', stereotypic lip smacking and urinary incontinence with the apparent absence of psychotic or mood symptoms were suggestive of an organic condition, particularly temporal lobe epilepsy.

The clinical differentiation of non-convulsive status epilepticus (NCSE) from catatonia only on the basis of history and examination may be difficult or even impossible. To made differential diagnosis even more problematic, the conditions may co-occur. As previous case reports documented cases of NCSE with catatonia as ictal manifestation (Drake,1983; Lim,1986). There was also a report of a patient whose catatonic syndrome mimicked NCSE (Louis,1995). The present report documented another case of catatonia masquerading psychomotor epileptic fits. In our case, all investigations including EEG tracings were normal. Past psychiatric history and the subsequent recognition of first rank symptoms suggested a diagnosis of schizophrenia as the psychiatric disorders underlying the catatonic syndrome. The use of electroencephalogram during the attack is pivotal in making the right diagnosis.

Apart from the similarity in clinical manifestations, seizures and catatonia may occur concurrently. Barnes et al(1986) reported 25 cases of catatonia found 10 without identifiable aetiology. Three of their cases had generalised epilepsy, where fits occurred both in catatonic state and during periods of normal motor behaviour. In another patient, classified as idiopathic catatonia, the catatonic episode followed a generalised seizure during pregnancy .

In another study (Primavera,1994) of 29 patients with acute catatonic syndrome, the rate of seizure was found to be higher than in the age matched general population. Four patients had seizures during the course of acute catatonic syndrome. Seizures were more frequent in patients with organic catatonia, and only occasionally occurred in acute catatonia with episodes associated with psychiatric disorders. Seizures in acute catatonic syndrome are likely to have multiple causes such as hyperpyrexia, liver and renal failure, central nervous system infection, or drug treatment. Lim et al (1986) suggested that the 'ictal' catatonia in their patients was resulted from cortical epilepsy with secondary generalisation to the limbic system. Rankel et al (1988) successfully used carbamazepine in two patients of catatonic stupor, one suffering from schizophrenia and other with schizoaffective disorder , based on the theory that carbamazepine suppresses the hypothesized subcortical limbic focus preventing the spread of its excitation to other brain regions, a mechanism proposed for the pathogenesis of catatonia.

CONCLUSION

Catatonia and temporal lobe epilepsy share some common clinical features.The clinical awareness of both conditions and the use of EEG are helpful in the differential diagnosis. Since both conditions constitute a neuro-psychiatric emergency and could be life threatening, an accurate and early diagnostic judgement is essential to provide effective treatment.

ACKNOWLEDGEMENT

The author wishes to acknowledge the contribution of Dr Dunn Lai-wah, Eva (鄧麗華), Dr Gabor S. Ungvari (翁和禮) and Dr Linda C W Lam (林翠華) and Dr SHYEUNG (楊秀香).

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Miao Yin-king MRCPsych *Senior Medical Officer,Department of Psychiatry, Pamela Youde Nethersole Eastern Hospital,3 Lok Man Road, Chai Wan, Hong Kong*