



CASE REPORT

DELERIUM: OFTEN OVERLOOKED

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ABSTRACT

Delirium is a neurobehavioral syndrome caused by dysregulation of neuronal activity secondary to systemic disturbances. It occurs in 35 to 80 % of critically ill, hospitalized patients. Recent estimates of in-hospital mortality rates among delirious patients have ranged from 25 to 33%. Based on arousal disturbance and psychomotor behaviour, the following three clinical subtypes of delirium have been described like hyperactive (hyper aroused, hyper alert, or agitated), hypoactive (hypo aroused, hypo alert, or lethargic), and mixed (alternating features of hyperactive and hypoactive types). Antipsychotics have been the medication of choice in the treatment of delirium. We report a case of hyper acute delirium due to parietal lobe lesion without any focal neurological deficit. While other metabolic causes were ruled out. Patient after pharmacological like antipsychotics as well as non pharmacological intervention showed partial improvement in few days. Thus we conclude that delirium is a serious cause and complication of hospitalization in elderly patients and should be considered to be a medical emergency until proven otherwise. For these reasons, prevention, early recognition and effective treatment of delirium are essential. Pathogenesis of delirium is incompletely understood. Right parietal and thalamic lesions have been reported most commonly among neurological causes.

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INTRODUCTION

Delirium is a common and serious disorder with high morbidity and mortality. It occurs in 35 to 80 percent of critically ill, hospitalized patients.¹ Recent estimates of in-hospital mortality rates among delirious patients have ranged from 25 to 33%.² It may lead to mortality if not detected early. Delirium is a common clinical syndrome characterized by inattention and acute cognitive dysfunction. The hallmarks of delirium are waxing and waning signs, symptoms, and sensorium. (Shahid Ali et al., 2011) A diverse range of terms has since emerged to describe delirium, including 'acute confessional state', 'acute brain syndrome', 'acute cerebral insufficiency' and 'toxic-metabolic encephalopathy', but 'delirium' should still be used as the standard term for this syndrome. Over time, the term delirium has evolved to describe a transient, reversible syndrome that is acute and fluctuating, and which occurs in the setting of a medical condition. Delirium has many clinical manifestations, but defined as a relatively acute decline in cognition that fluctuates over hours or days. (Andrew Josephson and Bruce L. Miller, 2015 19th edition)

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Based on arousal disturbance and psychomotor behaviour, the following three clinical subtypes of delirium have been described:

- 1) Hyperactive (hyper aroused, hyper alert, or agitated),
- 2) hypoactive (hypo aroused, hypo alert, or lethargic), and
- 3) mixed (alternating features of hyperactive and hypoactive types). (Shahid Ali et al., 2011)

Common aetiologies of delirium includes toxins (benzodiazepines, narcotics, and drug abuse like alcohol, opiates, bath salts, cocaine, etc.), metabolic conditions (electrolyte disturbances, pulmonary, renal, cardiac and liver failure, dehydration, vitamins deficiencies), infections (systemic and CNS infections like meningitis, and encephalitis), endocrine conditions (thyroid and adrenal insufficiency), cerebrovascular disorders (hypoperfusion states, encephalopathy, focal ischemic strokes, nondominant parietal and thalamic lesions), seizure related disorders (nonconvulsive status epilepticus and prolonged postictal phase with intermittent seizures). (Weisberg and Gracia, 2010)

There is no clear aetiopathogenesis model for delirium. However, two hypotheses have been proposed: neurotransmitter hypothesis and inflammatory hypothesis.

(Surendra Kumar Mattoo *et al.*, 2010) There are a number of neurotransmitters believed to be involved in the pathogenesis of delirium, including acetylcholine, serotonin, dopamine, and gammaaminobutyric acid (GABA). (Michael H. Johnson, 2001) The common differential diagnoses of delirium are dementia, depression and psychosis/schizophrenia. In contrast to delirium, dementia is characterized by insidious onset, and gradually progressive course with no diurnal variation. However, dementia itself being a risk factor for development of delirium. (Surendra Kumar Mattoo *et al.*, 2010)

Prevention is the most effective strategy for minimizing the occurrence of delirium and its adverse outcomes. Non pharmacological strategies are the first-line treatments for all patients with delirium. The non pharmacological approaches available include reorientation and behavioral intervention. (Tamara G. Fong *et al.*, 2009) Appropriate management of the underlying condition (s) and the drugs that the patient is taking, remains the mainstay of delirium treatment. (Gideon Caplan, 2011) Antipsychotics have been the medication of choice in the treatment of delirium. Droperidol, a butyrophenone with a rapid onset of action and relatively short half-life that is more sedating than haloperidol, has also been found to be an effective treatment for hospitalized patients with agitation, although not necessarily delirium. (Paula Trzepacz *et al.*, 2010) Some atypical antipsychotics (for example, risperidone, olanzapine and quetiapine) have been used clinically to treat agitation in patients with delirium, with controlled trials showing efficacy at least comparable to haloperidol.6Few controlled studies have evaluated the efficacy of benzodiazepines (lorazepam, oxazepam) as a monotherapy (i.e., not in combination with other pharmacotherapies) for the treatment of delirium. (Paula Trzepacz *et al.*, 2010)

Case Report

A 46 year old married right handed female presented with continuous three to four episodes of generalised tonic clonic convulsion followed by which she became drowsy. Patient was admitted in intensive care for observation. Patient was a known case of generalised tonic clonic seizure disorder since 10 years and type 2 diabetes mellitus since 1 year on treatment with oral phenytoin 100mg BD and Metformin 500mg BD. She had no other relevant history.

Patient remained drowsy for two to three days. Later over a period of four to five days in intensive care unit, patient had an irrelevant talk, confusion and emotional outburst which were episodic. Over a period of four to five days, patient continued to show episodic agitation and aggressive behaviour. Her neurological examination revealed no obvious sensory and motor deficit except for higher mental functions as already described above. She was afebrile and other systemic examinations were normal. She was evaluated for metabolic causes which remained normal throughout. Her complete blood count and metabolic parameters were all normal. Lumbar puncture was done for CSF analysis which was normal. Further CT Brain was done which revealed right parietal lobe gliotic area. EEG could not be done due to agitation and non availability of bedside facility. The differential diagnostic possibilities in this patient considered were: a) post epileptic

confusion which was unlikely because of long duration of abnormal behaviour. b) CNS infections like encephalitis or meningitis which were also unlikely as there was no meningeal signs and CSF was normal. Hence neurologist opinion was sort and on evaluation, he opined on case as hyper acute delirium. Further on his advice, she was administered injectable phenobarbital sodium and other supportive treatment. Oral Quetiapine was also initiated. Patient after pharmacological as well as non pharmacological intervention showed partial improvement in few days. However due to financial reasons patient was then taken to charitable hospital.

DISCUSSION

This patient showed dichotomous features of agitation as well as drowsiness over a week. Patient partially recovered after prolong stay and with the use of antipsychotics for a while. The only cause in this patient was parietal lobe lesion without any focal neurological deficit. Diagnosis of delirium is clinical and mainly bedside after careful history and clinical examination. Using well validated CAM, diagnosis of delirium was made on following features of acute and fluctuating course, and inattention accompanied by altered level of consciousness.2 Helpful investigations like CBC, renal and liver function tests, systemic infection screen by cultures and chest radiographs, arterial blood gas, and CSF studies did not reveal any causative factors towards her delirium. However except for history of GTC seizure and gliotic area in right parietal lobe were strong indicators for etiology of her present hyper acute delirium state.

Pathogenesis of delirium is incompletely understood. Right parietal and thalamic lesions have been reported most commonly among neurological causes. In a previously healthy individual with no known history of cognitive illness develops delirium, its essential to rule out neurological illness including previous strokes. The etiologies of delirium are diverse and multifactorial and often reflect the pathophysiological consequences of an acute medical illness, medical complication or drug intoxication.

The treatment of patients with delirium begins with an essential array of psychiatric management tasks designed to provide immediate interventions for urgent general medical conditions, identify and treat the etiology of the delirium, ensure safety, and improve the patient's functioning. Environmental and supportive interventions are also generally offered to all patients with delirium and are designed to reduce factors that may exacerbate delirium, to reorient patients, and to provide them with support. Somatic interventions largely consist of pharmacologic treatment with high-potency antipsychotic medications. Delirium is a serious cause and complication of hospitalization in elderly patients and should be considered to be a medical emergency until proven otherwise. For these reasons, prevention, early recognition and effective treatment of delirium are essential. We conclude that in patients with lesion in the parietal lobe, hyper acute delirium is not uncommon. The clinical implications, however, are clear: cerebrovascular disease old or recent should not be dismissed as a diagnostic consideration in patients with agitated confusional states, who lack definite lateralizing signs.

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