Irish Journal of Psychological Medicine, (2023), **40**, 292–295. doi:10.1017/ipm.2020.76

CASE REPORT

Manic-like presentation with an organic aetiology

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A 57-year-old man with chronic obstructive pulmonary disease (COPD), obstructive sleep apnoea (OSA) and no prior psychiatric history presented repeatedly over 6 months with mental and behavioural changes. Laboratory tests, chest X-ray and sleep study diagnosed an infective exacerbation of COPD, type II respiratory failure and OSA. Differential diagnoses included delirium, primary mania in bipolar affective disorder or organic pathology causing secondary mania. Oxygen, steroids, bronchodilators, antibiotics and non-invasive ventilation were administered to treat his infection and respiratory failure. However, blood gas analysis showed persistent hypoxia and hypercarbia, aggravating his ongoing mental state disturbance that required security supervision and sedation with antipsychotics and benzodiazepines. Sudden onset of classic manic symptoms and multiple presentations suggested secondary mania, driven by chronic hypoxia in end-stage COPD and OSA. The challenge was establishing a balance between mental state control and treatment of physical illness.

Received 03 October 2019; Revised 31 March 2020; Accepted 05 June 2020; First published online 07 September 2020

Key words: Hypoxia, mania, organic, psychosis, secondary.

Introduction

Mania may be defined as a 'distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased goal-directed activity or energy' (American Psychiatric Association, 2013). Other features include pressured speech, flight of ideas, grandiosity, impaired judgement, decreased need for sleep, increased physical activity, distractibility and increased risk-taking behaviours. Mania is a set of symptoms and signs of both physical and psychological with cognitive, affective and behavioural components. It is a state or syndrome, but not a diagnosis; therefore, it poses a challenge to determine its source and to differentiate between primary and secondary mania. Primary mania occurs in the context of bipolar affective disorder (BPAD) or schizoaffective disorder, whereas secondary or organic mania is a mood disorder due to a medical condition (American Psychiatric Association, 2013), underlying cerebral or systemic pathology (Krauthammer & Klerman, 1978). Organic manic disorder in International Statistical Classification of Diseases, 10th Revision is included under 'disorders characterised by a change in mood or affect, usually accompanied by a change in the overall level of activity' (World Health Organisation, 2009). The cerebral or other physical cause must be demonstrated by appropriate history, physical or laboratory investigations and must meet the criteria for both a manic episode and a disorder of organic aetiology. Idiopathic BPAD should be diagnosed only after all organic causes have been ruled out (Larson & Richelson, 1988). It can be difficult to determine whether symptoms of mania are driven by a psychiatric or underlying organic disorder. The first presentation of mania in a person greater than 35 or 40 years of age, atypical symptoms and signs such as disorientation, altered consciousness, visual or olfactory hallucinations and focal neurology, and prolonged or unremitting mania with poor response to treatments all prompt suspicion of secondary or organic causes (Krauthammer & Klerman, 1978; Satzer & Bond, 2016).

Case report

This case describes the first presentation of manic-like symptoms in a 57-year-old man with no prior history of mental illness. He was admitted three times over the course of 5 months with similar mental and behavioural changes each time. He had a background of chronic obstructive pulmonary disease (COPD), obesity, 37-pack-year smoking history and alcohol abuse. He was unemployed and lived with his wife for whom he was the carer and 18-year-old stepson. He first presented with an infective exacerbation of COPD, type II respiratory failure and an acute change in behaviour noticed by his family. This included increasing confusion, sleeplessness, irritability and threats of self-harm. He was preoccupied with money he had received as a carer's allowance and then spent on a trailer, claiming he was owed a lump sum. On presentation in the emergency department, he was

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aggressive and required IV lorazepam for sedation. No formal thought disorder or hallucinations were elicited. The computerised tomography (CT) brain demonstrated no abnormality and routine laboratory tests were unremarkable. He was treated with intravenous antibiotics, steroids, nebulised bronchodilators and non-invasive ventilation (BiPAP). While admitted, he also underwent a sleep study and was diagnosed with obstructive sleep apnoea (OPA) with plans for home continuous positive airway pressure (CPAP) put in place. He was discharged after 3 days on oral antibiotics and steroids. His mental and behavioural symptoms, during his admission, were attributed to hyperactive delirium secondary to infection.

However, he remained agitated and paranoid at home with ongoing poor sleep and displaying irritability towards his family. He was presented for the second time 1 week later in a similar fashion and was again physically aggressive and uncooperative in the emergency department. He was found to be orientated with circumstantiality, mood lability and distractibility. He had pressure of speech and flight of ideas. This time he did not have delusions and did not exhibit first-rank symptoms of psychosis. He scored 22/30 on Mini-Mental State Examination with deficits in attention and delayed recall. A C-reactive protein (CRP) was raised on this admission at 41 mg/l (0–5 mg/l). Acutely, he required sedation with haloperidol and lorazepam. His mental state was so convincingly mood related that he was assumed to have co-morbid mania and he was admitted to the psychiatric ward for ongoing management. Olanzapine 10 mg p.m./5 mg a.m., pro re nata (PRN) lorazepam and tapering steroids were commenced. He was discharged 3 days later with a CRP of < 5 mg/l. However, his rapid improvement prompted his symptoms to be attributed once more to hyperactive delirium or possibly a steroid-induced psychiatric disturbance/mania.

His third presentation was 5 months later with delusions involving winning money in the lottery and starting a new business with a similar theme to the first episode regarding finances. He exhibited pressure of speech, flight of ideas, mood lability, marked disinhibition and distractibility. His compliance had been poor on home CPAP for OSA as well as continued cigarette smoking. He was admitted to the psychiatric ward but became acutely agitated and aggressive, requiring sedation with IM lorazepam and haloperidol which lead to respiratory depression, a Glasgow coma scale (GCS) of 4/15 and oxygen saturation of 60%. This necessitated flumazenil for benzodiazepine reversal. He was transferred urgently to the care of the medical team. He was commenced on BiPAP 14/7 in the high-dependency unit and his GCS improved. He was subsequently transferred to a medical ward and treated for hospital-acquired pneumonia with acute on chronic type II respiratory failure. He was treated with oxygen, intravenous antibiotics and steroids, nebulised bronchodilators and showed some improvement medically. Chest X-ray was clear and BiPAP was stopped. However, he experienced further episodes of increased agitation and aggression while still in the hospital. He attempted to self-discharge and seriously injured the security staff. Olanzapine 10 mg p.m./5 mg a.m. was recommenced as well as PRN haloperidol. A psychiatric nurse special was also necessary, given his ongoing manic-like symptoms and the expertise required to behaviourally manage them.

A balance was required between the treatment of his mental state with sedation and maintenance of his respiratory function. Arterial blood gases demonstrated persistent hypoxia and hypercarbia without acidosis. However, it was not considered possible to optimise his respiratory function, as home oxygen was contraindicated due to active smoking, and he had ongoing non-compliance with home CPAP. His steroids were tapered to nil and he was continued on olanzapine and oral antibiotics. Discharge to home was arranged 8 days later. Although features of mania remained he had improved on olanzapine and with respiratory treatment. An impression was formed that he had secondary mania precipitated by chronic hypoxia in end-stage COPD and OSA.

Discussion

Due to the sudden onset of classic manic and behavioural symptoms and repeated presentations over a short time period, in a man aged 57 with no personal or family history of BPAD or other mental illness, an underlying organic disorder was suspected. New-onset BPAD had not formally been diagnosed. Steroid-induced psychosis and hyperactive delirium had also been considered as differentials or coexisting states, also causing changes in affect, cognition and behaviour. However, the prescription of steroids was not chronologically linked to the onset of his symptoms in any of the three admissions, but may have maintained them when the dosages were increased. Hyperactive delirium can cause various psychiatric symptoms but did not solely explain the florid manic-like presentation in this case. During his admission, he continued to show features of hyperactive delirium as well as of mania, including fluctuating symptoms, sleep–wake disturbance, confusion and inattention. Therefore, the challenge was in identifying and treating the cause of his mental state change while also managing his co-morbidities.

Distinguishing primary from secondary mania is essential, as treatment requires either mania prophylaxis or elimination of underlying organic pathology

https://doi.org/10.1017/ipm.2020.76 Published online by Cambridge University Press

Table 1. *Causes of organic mania*

Endocrine	Hyperthyroidism
	Premenstrual psychosis
	Cushing's syndrome
	Post-partum psychosis
Infection	Q fever
	Infectious mononucleosis
	Viral encephalitis
	Cryptococcal meningoencephalitis
	AIDS
	Creutzfeld–Jacob disease
	Neurosyphilis
Malignancy	Brain tumour (hypothalamic, diencephalic and frontal)
	Carcinoid syndrome
Metabolic	Uraemia
	Vitamin B12-deficiency
	Vitamin B3-deficiency
	Wilson's disease
	Haemodialysis
Neurodegenerative	Multiple sclerosis
	Huntington's disease
	Frontotemporal dementia
	Temporal lobe epilepsy
	Idiopathic calcification of basal ganglia post-encephalitic parkinsonism
Trauma	Post-traumatic encephalopathy
	Neurosurgery
	Post-operative state
Toxic/drugs	Alprazolam and benzodiazepines
	Amphetamines and psychostimulants
	Antidepressants
	Baclofen
	Bromides
	Bromocriptine and dopamine agonists
	Cimetidine
	Cocaine and sympathomimetics
	Codeine and paracetamol
	Corticosteroids
	Donepezil
	Cyclobenzaprine
	Guanfacine
	Hydralazine
	Isoniazid
	Interferon alpha
	Levodopa
	Metoclopramide
	Metrizamide
	Mirtazapine
	Phencyclidine
	Procainamide
	Procarbazine
	Thyroid preparations
	Yohimbine
Vascular	Ischaemic stroke
	Intracerebral haemorrhage
	Cerebrovascular lesions (temporal and hemispheric)

Krauthammer & Klerman (1978), Cummings (1986), Larson & Richelson (1988), Mendez (2000), Satzer & Bond (2016).

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and addressing physical alongside psychiatric symptoms (Krauthammer & Klerman, 1978).

Acute treatment in both primary and secondary mania includes de-escalation, isolation, supervision and sedation with antipsychotics, short-acting benzodiazepines or mood stabilisers such as haloperidol, lorazepam and lithium. In the case of secondary mania, the treatment also includes targeting the underlying organic cause. Treatment of both mental and physical state is necessary whether the cause is reversible or irreversible. Treatment of the underlying cause, as opposed to mania prophylaxis, is what differentiates primary from secondary mania.

There are numerous secondary causes of mania (Table 1). Investigations should be undertaken to uncover the precise pathology, during or after stabilisation of the patient's mental and physical state. This includes a thorough clinical history and examination, as well as biochemical, radiological and other relevant investigations. A full history should be taken, paying attention to current medical symptoms, recent infections, use of medications, alcohol or drugs of abuse, as well as a personal or family history of BPAD and a personal history of other chronic illness. Mental state examination, physical examination and a collateral history should also be taken. Relevant biochemical and radiological investigations should then be undertaken, including a full blood count, CRP, serum glucose, serum folate and vitamin B12, thyroid function, renal function and electrolytes, liver function, urine toxicology, cerebrospinal fluid analysis, electroencephalogram, CT and magnetic resonance imaging brain (World Health Organisation, 2009; Satzer & Bond, 2016). No further investigation may be appropriate or a likely cause assumed such as in this case.

The challenge was to identify and treat the cause of the patient's psychiatric symptoms, while managing his co-morbidities.

Conclusion

Mania presenting for the first time in an older person without a history of previous mania or mental illness may be primary or it may be secondary to an underlying organic pathology. A person may present with classic symptoms and signs of a manic episode; however, the difficulty arises in uncovering the underlying physical or metabolic abnormality and thereby treating the cause. It is imperative to target both the psychiatric symptoms and physical illness in order to successfully treat organic mania. In this case, a definitive diagnosis was not established, therefore, a combined approach was necessary to optimise symptoms, function and quality of life for this patient to effectively treat and ameliorate his physical and mental state.

Conflict of interest

K.K., C.C. and E.N. have no conflicts of interest to disclose.

Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008. The authors assert that ethical approval for publication of this case report was not required by their local ethics committee. A written informed consent was obtained from the patient for publication of this case report.

Financial Support

This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

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