Mania, as defined in DSM-IV, is more than a week-long history of abnormally and persistently elevated, expansive or irritable mood (less if hospitalisation is required). It is often associated with additional symptoms such as inflated self-esteem, flight of ideas, decreased need for sleep, distractibility, psychomotor agitation, psychotic features and excessive involvement in pleasurable activities, leading to painful consequences and impairment in social or occupational functioning.

Mania occurring for the first time in later life is an uncommon, heterogeneous condition with an often atypical presentation showing a mixture of manic, dysphoric and cognitive symptoms, and less euphoria than in younger, more typical cases. Studies in the elderly have shown that increased cerebral vulnerability due to organic insults, eg stroke or head trauma, play a stronger role than life events alone in precipitating late-onset mania.

Secondary mania is defined as elevated or irritable mood caused by neurological, metabolic or toxic disorders with the presence of at least two other symptoms such as hyperactivity, pressured speech, flight of ideas, grandiosity, decreased sleep, distractibility and lack of judgement.

It is not essential (according to the DSM-IV criteria for secondary mania) for the full diagnostic criteria of manic illness to be met; however, evidence from the history, physical examination or laboratory findings that the disturbance is the direct physiological consequence of a general medical condition must be present and not accounted for by any other mental disorder or during the course of delirium.

Post-stroke mania

Neuropsychiatric syndromes are common in the setting of stroke disease. For example, there is high incidence of depression after an acute brain injury. Other syndromes that can occur after stroke include cognitive impairment, dementia, personality change, psychosis, apathy and anxiety. However, a systematic review found only 74 reported cases of adult stroke patients with mania symptoms in the last 50 years. Thus mania is a rare consequence of stroke and right-sided cerebrovascular lesions involving regions connected to the limbic cortex have been implicated in late-onset mania.

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Here, we describe a patient with no past psychiatric history, who presented with manic illness in the context of a recent stroke.

Presentation

An 83-year-old man was referred to old age psychiatry services from a stroke rehabilitation unit. Initially the man presented to the general hospital with a left-sided weakness in his leg, arm and face. A CT scan of his brain revealed a haemorrhagic stroke involving the right posterior thalamus with ventricular extension.

Within two weeks of his admission, his behaviour became very erratic, unpredictable and aggressive. He assaulted various members of staff and on one occasion bit a nurse on the breast. He seemed overfamiliar in his manner, disinhibited in his speech and extremely difficult to manage in the stroke rehabilitation ward due to his increasing aggression and other challenging behaviours, such as repeatedly throwing himself onto the floor. He was otherwise alert but disoriented in time and place. Due to his ongoing aggression, which did not settle with regular use of benzodiazepines, he was transferred to the local old age organic psychiatry unit.

On admission to the organic psychiatric unit, the patient was found to be irritable and unpredictably aggressive. He required full assistance with personal care. All blood tests, including a delirium screen, were normal. He remained unsettled with inappropriate behaviour such as removing clothes in front of visitors, urinating in the ward and being sexually disinhibited towards

Rajdeep Routh MBBS, MRCPsych, Andrew Hill MBChB, DGM
the nursing staff. His mood was labile, with occasional tearful outbursts. He was increasingly talkative with occasional pressure of speech and evidence of flight of ideas. He started misidentifying staff as guards in a concentration camp and appeared very distressed by the fact that the guards were refusing to give even water to ‘wounded soldiers’, meaning other patients in the ward.

His behaviour continued to deteriorate along with evidence of poor sleep and increased distractibility. He remained agitated with paranoid delusions, now taking the form of the IRA being on the ward. He felt threatened, terrified and was very distressed. He was commenced on haloperidol starting at 0.5mg once daily titrated up to 0.5mg three times daily; however, this did not alleviate the mania symptoms. Due to his worsening behaviour requiring physical restraint, he was detained under the Mental Health Act.

From the patient’s presentation, it soon became clear that he was manic and most likely suffering from a post-stroke mania in the absence of any past psychiatric history or family history of mental illness. There was no history of substance misuse and his self-reported alcohol excess at the time of referral was later confirmed by his family to be influenced by his inflated self-esteem. He was subsequently started on olanzapine 5mg daily, which was well tolerated and improved his behaviour significantly. Over several weeks his behaviour settled, initially during the day, and subsequently through the night. His detention was subsequently revoked.

He was ultimately discharged to the care of a local residential home according to his own wishes and for better management of his post-stroke residual disability. Olanzapine 5mg daily was continued after discharge with a plan of follow up and review in the community.

Discussion
We suspect that post-stroke mania, like other psychiatric features following a stroke, is under-recognised due to its atypical presentation. Early recognition of post-stroke mania in patients such as ours helps direct appropriate management and minimise risk and suffering. A typical patient with post-stroke mania would be a male, without a personal or family history of psychiatric disorder, with at least one vascular risk factor, without evidence of any subcortical atrophy and who had suffered a right cerebral infarct. The temporal relationship between stroke and mania ranges from immediately post-stroke to up to two years thereafter. However, the majority of mania cases seem to appear within the first month following a stroke, as in our patient.

Optimum management of concurrent vascular disease as well as mania is indicated, preferably in a psychiatric setting, with continuation of stroke rehabilitation. Previous case reports have shown various agents such as mood stabilisers; typical antipsychotics like haloperidol; atypical antipsychotics like olanzapine or risperidone; and even benzodiazepines being used in the treatment of post-stroke mania. Both typical and atypical antipsychotic drugs are associated with an increased risk of stroke. The risk of stroke is even greater in the first weeks of treatment and in the presence of other risk factors like older age, cognitive impairment and vascular illness.

In our patient, the risks to self and others were extremely high due to the level of his distress and aggression. He showed no signs of improvement with either benzodiazepines or haloperidol but responded very well to olanzapine without any major side-effects. Olanzapine was started only when other options, including appropriate placement and enhanced nursing care, had failed. In this case, clearly the benefits of treatment outweighed the risks of harm. However, a very cautious trial of olanzapine in patients with similar post-stroke manic presentation is advised because of the associated risks. In the future, double-blind, placebo-controlled studies will be useful in order to clarify the safety of antipsychotics in the treatment of post-stroke mania.

Declaration of interests
None declared.

Dr Routh is a ST5 Trainee in Old Age Psychiatry and Dr Hill is a GPST2 Trainee, Leverndale Hospital, Glasgow

References