CASE REPORT

Psychosis post corona radiata and lentiform nucleus infarction

Khadijah Hasanah Abang Abdullah,^{1,2} Suriati Mohamed Saini,¹ Shalisah Sharip,¹ Abdul Hamid Abdul Rahman¹

SUMMARY

¹Department of Psychiatry, UKM Medical Centre, Kuala Lumpur, Wilayah Persekutuan, Malaysia ²Universiti Sains Islam Malaysia, Nilai, Malaysia

Correspondence to Professor Suriati Mohamed Saini, suriati.saini@yahoo.com.my

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To cite: Abang Abdullah KH, Mohamed Saini S, Sharip S, et al. BMJ Case Rep Published online: [please include Day Month Year] doi:10.1136/bcr-2014-208954 Complications of stroke can include neuropsychiatric symptoms. However, post-stroke psychosis is rare. We report a case where an acute presentation of psychosis, depression and fluctuating cognitive impairment in a middle-aged man turned out to be related to a silent brain infarction. The patient had a background of poorly controlled type 2 diabetes mellitus with glycated haemoglobin level of 9.0-11.0%, hypertension and ischaemic heart disease. His CT brain results showed multifocal infarct with hypodensities at bilateral lentiform nucleus and bilateral corona radiata. His strong genetic predisposition of psychosis and a history of brief psychotic disorder with complete remission 3 years prior to the current presentation might possibly contribute to his post-stroke atypical neuropsychiatric presentation, and posed diagnostic challenges. He showed marked improvement with risperidone 6 mg nocte, chlorpromazine 50 mg nocte and fluvoxamine of 200 mg nocte. The need of comprehensive treatments to modify his stroke risk factors was addressed.

BACKGROUND

Stroke can lead to many complications including neuropsychiatric problems. Depression is a common complication of stroke with a prevalence of 29% according to a latest meta-analysis in 2013.¹ However, post-stroke psychosis is a rarer condition with an incidence of less than 1%.² This case illustrates a patient who developed depression, cognitive impairment and recurrent psychosis post infarction of corona radiata and lentiform nucleus following a complete remission of psychosis for a year. We also discuss the predisposing factors of developing poststroke psychosis and its management in this patient.

CASE PRESENTATION

A 48-year-old Indian man presented with a history of abnormal behaviour for 3 days prior to admission. He was preoccupied with religious rituals and the colour red, became withdrawn and had poor oral intake. He believed he was dead and needed to drink blood. He had auditory and visual hallucinations. He described seeing a Chinese girl with long hair and a red dress, telling him and gesturing to him to kill his wife. However, no attempt was made.

He also described having depressed mood, anhedonia, reduced concentration, reduced energy, feelings of hopelessness and worthlessness. He had suicidal thoughts of killing himself with a knife, but again, no attempts were made. He showed some fluctuation in his orientation and memories initially, but even after it improved, his psychosis and mood symptoms still continued.

He has a second degree relative with psychotic illness. Apart from that, at presentation he had poorly controlled type 2 diabetes mellitus, hypertension and ischaemic heart disease with angioplasty performed in the past.

Three years earlier, the patient was diagnosed with brief psychotic disorder. He presented with psychosis involving auditory hallucinations and persecutory delusions. CT of the brain during that admission did not reveal any abnormality. The symptoms resolved after 3 weeks of antipsychotic therapy and he was able to work.

A year after he was diagnosed with the brief psychotic disorder, he had a stroke with right corona radiata infarct. He had some facial weakness but otherwise had complete recovery. However, he stopped working and, at presentation, had been unemployed for 2 years. He gradually developed depressive symptoms and was started on antidepressant. His depressive symptoms somewhat improved, but he did not return to work and his family did not object, as he has other medical problems, which they believe make him tire easily.

Mental state examination on admission revealed an adult, Indian man who appeared slightly anxious and perplexed. He was disoriented to time, place and person. His mood was depressed. His affect was restricted. He had commanding auditory hallucination and visual hallucinations. He was under the delusion that he was already dead and needed to drink blood. His vital signs, systemic and neurological examinations were unremarkable.

INVESTIGATIONS

- CT brain scan: CT of the brain showed multifocal infarct with hypodensities at bilateral corona radiata (figure 1) and lentiform nucleus (figure 2)
- ► Glycated haemoglobin level of 9–11%
- Cognitive assessment using Montreal Cognitive Assessment (MOCA)³ showed poor visuospatial/ executive function, poor attention and poor recent memory.

DIFFERENTIAL DIAGNOSIS

- ▶ Psychosis and depressive disorder due to stroke
- Schizophrenia



Figure 1 Hypodensities of bilateral corona radiata in coronal view.

TREATMENT

The patient was treated with risperidone up to 6 mg nocte, chlorpromazine 50 mg nocte and fluvoxamine 200 mg nocte. Acute management of a patient presenting with psychosis and mood symptoms would be the same irrelevant of the stroke factor, but long-term management should include monitoring of psychotropic treatment, as the brain is more vulnerable to develop side effects, and comprehensive treatment to modify risk factors for recurrent stroke and cognitive function monitoring, as these patients are prone to develop dementia.⁴

OUTCOME AND FOLLOW-UP

The patient's psychotic and depressive symptoms improved with the medications. Long-term medications have been indicated in this case, especially because the patient has a history of a previous psychotic episode.

DISCUSSION

Generally, a brief psychotic episode carries a good prognosis, but these patients do have increased risk of developing



Figure 2 Hypodensities of bilateral lentiform nucleus in coronal view.

psychiatric illness.⁵ ⁶ However, in the case described, the patient's condition was complicated by a stroke, which occurred between the two psychotic episodes. The CT brain findings in this patient suggested that he could have had a recurrent stroke without clinical manifestation, which is also known as silent brain infarction (SBI). A systematic review by Fanning *et al*⁷ found a prevalence of SBI between 10% and 20% and it has been known to have a strong association with hypertension, carotid stenosis and metabolic syndrome. Therefore a clinician should have some suspicion of SBI-related neuropsychiatric symptoms in these high-risk patients who present with psychiatric symptoms. Apart from case reports,⁸ research on psychosis and SBI is limited.

The human brain is a complex organ and the link between lesion location and psychiatric symptoms presentation is not straightforward. A systematic review showed no support for the association between post-stroke depression and brain lesion location.⁹ However, some authors suggested post-stroke psychosis is related to right-sided brain lesions.¹⁰

The above case shows the patient had depressive symptoms after right corona radiata infarct and developed psychosis and depressive symptoms after bilateral infarcts of corona radiata and lentiform nucleus.

Corona radiata infarction can present with neglect, constructional apraxia and visuospatial impairment (right-sided lesion); and with aphasia, and verbal memory, higher level naming and verbal abstract impairment (left-sided lesion).^{11 12} While infarction of the lentiform nucleus is associated with micrographia (right-sided lesion); and with aphasia and agraphia (left-sided lesion).^{11 12} Apart from arterial supply to corona radiata and lentiform nucleus, the lenticulostriate arteries also supplies the head of caudate nucleus, which, if infarcted, can present with psychosis, mood and cognitive impairment.¹¹ Furthermore, in this patient, a positive family history of psychotic illness and a previous psychotic episode could have made his brain more vulnerable to neuropsychiatric symptoms post-stroke.

The patient had poor glycaemic and blood pressure control due to non-adherence to medication, and poor diet control. Although glycaemic and blood pressure control is important to prevent long-term microvascular and macrovascular complications, many patients still fail to achieve the target. A meta-analysis found coronary heart disease, non-adherence to diet, exercise and medication are associated with poor glycaemic control.¹³ Adherence to therapy, which includes taking medication, diet control, exercises and lifestyle changes, is lacking, especially in this group of patients with chronic illness.¹⁴ Some of the factors that influence non-adherence are complex dose regimes, age, perception and duration of disease, polytherapy, psychological factors, safety, tolerability and cost;¹⁵ which should be addressed in each patient with chronic illness.

A recent review¹⁶ discussed how type 2 diabetes mellitus or hyperglycaemia causes changes in the blood brain barrier permeability and inflammatory process. In type 2 diabetes mellitus, there is an activation of neuroendocrine and inflammatory response that is responsible for the induction of insulin resistance. Depression has also been suggested to involve inflammatory pathways where there is an increase in proinflammatory cytokines and a relative reduction of anti-inflammatory cytokines in the brain regions.¹⁷ ¹⁸ Thus, poor glycaemic control not only activates the inflammatory process but disrupts the blood brain barrier, which in turn leads to leakage of the inflammatory cytokines into the brain, possibly resulting in neuropsychiatric manifestations. Hypertension has been associated with mental illnesses such as depression, anxiety and dementia.^{19 20} Studies on hypertension and cognitive impairment have suggested few hypotheses to explain the pathophysiology.²¹ Hypertension causes vascular alterations, which results in lacunar infarcts and white matter hyperintensities (leucoaraiosis). It also influences β -amyloid accumulation, which disrupts neuronal synapsis and causes senile plaques.²² Another theory suggested degeneration of cerebral capillary, disruption of mitochondria, and alteration in glucose and ATP, which lead to neuronal death.²³ However, the association and its pathophysiological basis are difficult to assess and requires further research.

Measurement of regional cerebral blood flow (rCBF) has been used for assessing brain function in neuropsychiatric illnesses, especially when it cannot be explained by structural neuroimaging findings. Single photon emission brain CT is a reliable method for the measurement of rCBF. Although rCBF was not measured in the case presented, due to limited resources, it is an investigation worth considering in these types of cases. Studies on patients with schizophrenia have shown significant hypoperfusion in specific areas of prefrontal cortex, temporal lobe and basal ganglia.²⁴ In patients with depression, hypoperfusion is seen in the prefrontal area, temporal lobes, cingulate gyrus, limbic areas and, especially, in the left caudate nucleus.²⁵ It is suggested that specifying brain region involvement will help to target treatment and aid in treatment response monitoring.

Learning points

- Post-stroke psychosis is rare, but it can occur in patients with risk factors of psychotic illness, for example, family history of psychotic illness and history of psychosis.
- Physicians should have high suspicion for stroke-related causes in an acute neuropsychiatric presentation, especially in high-risk patients with stroke, for example, those with uncontrolled type 2 diabetes and cardiovascular disease.
- Management for post-stroke psychosis should involve modification of risk factors for stroke on top of the psychotropic medications targeting the psychiatric symptoms.

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Competing interests None.

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