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CASE REPORT
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Abrupt Onset Depressive Episode in Patient with Lenticular Nucleus Infarct with Low-Risk Factors

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To the Editor,

Post-stroke depression has been well-researched over the last few decades. Despite advances, it remains a poorly understood entity and till now, a replicable and valid model explaining the etiology of post-stroke depression remains elusive. This is alarming considering the fact that the prevalence of post-stroke depression has been estimated to be around 18 to 33% of patients suffering from stroke in various studies.[1] Post-stroke depression is often associated with changes in the expression of the monoamine receptors and alteration in the receptor sensitivity.[2] A number of studies have attempted to recognize clinical and neuro-anatomical correlates that can predict the development of post-stroke depression in patients suffering from stroke.[3–5] The most replicated risk factor that has been postulated for post-stroke depression include a history of mental disorder, higher stroke severity, higher post-stroke physical disability, higher cognitive impairment, and poorer social support.[3,4] However, here we present a case of post-stroke depression with few of the predictive factors being present.

Mr. M, a 69-year-old male presented to the outpatient clinic with complaints of low mood, crying spells and anhedonia for the last 2 weeks. As corroborated by the family, Mr. M was his usual self until the night before and the change in the presentation was sudden as apparent the next morning. Mr. M appeared sad for no apparent reason, clumsy in carrying out daily activities like self-care and dressing and developed facial deviation to one side. No other symptoms like gait changes, urinary and/or fecal incontinence or sensory changes were noted at the time.

Over the initial days, Mr. M remained aloof and was also found to have decrease in speech productivity and found it challenging to express himself, along with restricted vocabulary. His appetite during this time was reduced by about 75% and total duration of sleep was also decreased by about 50%. During interactions, Mr. M would be tearful and low in mood but denied any self-harm ideations.

In 3 days post onset, CT head was found to be normal, and symptomatic treatment was prescribed. With no noticeable improvement in the following 10 days, he was brought to our outpatient clinic for further management. An eval-

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uation revealed no past personal or family history of psychiatric illnesses. He also had no history of other medical comorbidities like hypertension, diabetes mellitus or dyslipidemia. Mr. M had a history of nicotine dependence for the last 40 years, predominantly using bidis (tobacco rolled in leaf). He was also using alcohol in a non-dependent pattern for the last 15 years, and predominantly using country-made liquor. Since the onset of the symptoms, he had stopped taking either of the substances. Sensory and motor examination of the nervous system was mostly normal, except for deviation of the face to the right side.

The plantar responses on each side were flexor. His cognitive testing revealed features of aphasia. He presented with a depressed mood and ideas of helplessness, though the evaluation was hampered by paucity of speech. The patient scored 15/15 on the Glasgow Coma Scale, and scored 2 on the Modified Rankin Scale, signifying 'Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance'. The National Institutes of Health Stroke Scale (NIHSS) rating was 4 denoting minor stroke. The depressive symptoms of the patient were evaluated using the Hamilton Depression Rating Scale and the score was found to be 12 (denoting mild depression). The patient was tested for his cognitive functioning with Montreal Cognitive Assessment (MoCA) and the score was found to be 15/30, signifying severe cognitive impairment. Due to high degree of suspicion a neurological insult because of abrupt onset of depressive symptoms with focal neurological signs and cognitive symptoms, magnetic resonance imaging (MRI) of the brain was performed, which showed evidence of infarct in the region of the left lenticular nucleus (Figure 1). Based on the obtained information, the diagnosis of major depressive disorder, secondary to infarction of the left lenticular nucleus, was made according to the Diagnostic and Statistical Manual 5th edition. The patient was initiated on Escitalopram (10 mg) for the depressive symptoms and post-stroke measures included initiation on Atorvastatin (10 mg) and Aspirin (75 mg), after consultation with Neurology. The patient was discharged after one week, when the HAM-D score was 10 and the MoCA score was 19.

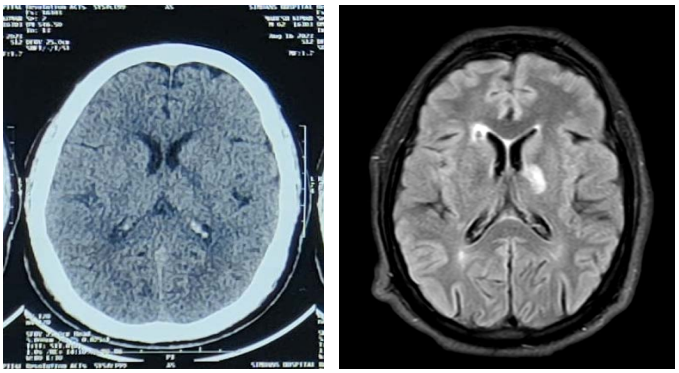

Figure 1: Image on the left is the computed tomographic image of the brain done on the 3rd day of the onset of the symptoms and image on the right is the diffusion weighted image of the brain done on 14th day of the onset of symptoms.

Strokes involving the various regions of basal ganglia have been repeatedly implicated for post-stroke depression in various studies.[1,6] In our patient, we also found that the afflicted region was the lentiform nucleus. Still, we decided to report this case because we felt that there were some critical learning points from this case. Firstly, in this case, the onset of symptoms was abrupt, which is relatively rare even in cases of secondary depression (depression due to other medical causes). Though there was certain overlap with cognitive symptoms, the manifestations of the depressive symptoms were clear (as manifested by depressive cognition, decreased appetite and sleep and low mood) in this case. Previous studies on this topic reported that symptoms are highest within the first 3 months of stroke, but an abrupt onset (<48 hours) is relatively rare.[7] Secondly, most of the existing literature shows a positive correlation with the severity of stroke and degree of disability. In our case, however, the patient had a minimal disability and the severity of the symptoms were mild. Finally, though a suspicion of cerebro-vascular accident was very high during the initial workup, the initial CT images were inconclusive. It was during the later MRI scans that the diagnosis could be established. A valid question that arises in this context is the overlay between post-stroke depression and post-stroke cognitive impairment. Current evidence does suggest that the cognitive profile of patients with post-stroke depression is worse than patients who do not develop depression following stroke.[8–10] Cognitive impair-

ment can be seen in about 35 to 87% patients of with post-stroke depression.[10] There has been attempts to find neurological correlates of depression and cognitive impairments and it was found that cognitive impairment in post-stroke depression is commoner in patients having lower years of education, higher age and left-sided lesions (vs right sided lesions).[8] We believe that this case goes on to show that the importance of detailed clinical evaluation, including emphasis on history taking can avoid many unintended clinical misdiagnoses. In case of high clinical suspicion one can go for regional cerebral blood flow (rCBF) measurement to assess brain function in neuropsychiatric disorders. For example, single photon emission brain CT is a reliable method for the measurement of rCBF. We were not able to go for it due to limited resources. It is an investigation worth considering in such types of cases.

References

1. Medeiros GC, Roy D, Kontos N, et al. Post-stroke depression: A 2020 updated review. *Gen Hosp Psychiatry* 2020; 66: 70–80.
2. Guo LH, Wang S, He R. Poststroke Depression. *Stroke* 1995; 9: 230–231.
3. Chen Y-M, Chen P-C, Lin W-C, et al. Predicting new-onset post-stroke depression from real-world data using machine learning algorithm. *Front Psychiatry* 2023; 14: 1195586.
4. Hackett ML, Anderson CS. Predictors of Depression after Stroke. *Stroke* 2005; 36: 2296–2301.
5. Ladwig S, Werheid K, Südmeyer M, et al. Predictors of post-stroke depression: Validation of established risk factors and introduction of a dynamic perspective in two longitudinal studies. *Front Psychiatry*; 14. Epub ahead of print 2023. DOI: 10.3389/FPSYT.2023.1093918/FULL.
6. Espárrago Llorca G, Castilla-Guerra L, Fernández Moreno MC, et al. Post-stroke depression: an update. *Neurol (English Ed* 2015; 30: 23–31.
7. Robinson RG, Jorge RE. Post-Stroke Depression: A Review. *Am J Psychiatry* 2016; 173: 221–231.
8. Baccaro A, Wang YP, Candido M, et al. Post-stroke depression and cognitive impairment: Study design and preliminary findings in a Brazilian prospective stroke cohort (EMMA study). *J Affect Disord* 2019; 245: 72–81.
9. Tu J, Wang LX, Wen HF, et al. The association of different types of cerebral infarction with post-stroke depression and cognitive impairment. *Medicine (Baltimore)*; 97. Epub ahead of print 1 June 2018. DOI: 10.1097/MD.0000000000010919.
10. Terroni L, Sobreiro MFM, Conforto AB, et al. Association among depression, cognitive impairment and executivedysfunction after stroke. *Dement Neuropsychol* 2012; 6: 152.