Case Report

A temporoparietal infarct lesion presented as the first-episode mania in an elderly male

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ABSTRACT

The occurrence of late-onset mania after 50 years of age for the 1st time is generally rare and is generally, but not necessarily, secondary to an organic lesion. Various underlying causes include neurological lesions, tumors, thyroid-related disorders, parathyroid abnormalities, electrolyte imbalances, and vascular lesions. After 75 years of age, the incidence of reported mania was 2/100,000, which is very rare. The main concern for such cases is to rule out secondary causes in assessment, diagnosis, and perspectives related to clinical management. We present a case where the first manic episode occurred at the age of 77 years with underlying infarcts in the temporal and parietal regions as seen in MRI brain contrast images.

Key words: Late-onset, Parietal, Temporal, Vascular lesions

The occurrence of the symptoms of mania or any hypomanic episode for the 1st time after 50 years of age is defined as late-onset mania. As per previous studies, approximately 6% of older patients (over 50 years) suffered from the symptoms of mania. Usually, patients with vascular insults and neurological infarcts present with late-onset manic presentations [1]. The occurrence of old age mania after 58 years for the 1st time also has cognitive impairments which have reversible causes such as infarction and vascular lesions [2]. A review analysis by Tohen *et al*. concluded that there were 50 consecutive admissions for old age mania over 65 years, of which 12 were associated with stroke or CNS infections [3]. A study by Berlin *et al*. found that patients with neurological lesions in orbitofrontal areas and basal ganglia areas may also present with old age mania [4]. Another study done by Sperber and Karnath showed that right-sided lesions are more frequently associated with mania, although the exact mechanism of how brain results lead to manic symptoms is not so clear [5]. Hain and Peter concluded that the lesions in subcortical areas of the brain result as the first-episode mania in a 65-year-old male [6].

CASE REPORT

A 77-year-old married male came to Geriatric Mental Health, OPD, KGMU, Lucknow, with chief complaints of non-sensible talks for 20 days, irritability, overtalkativeness, sleep disturbance, big talks from 2 weeks, and over-religious activities from the last 1.5 weeks. The patient used to talk excessively, he was overfamiliar with others, and sometimes strangers used to claim that he was excessively knowledgeable. He also said that he could see the image of a helicopter in clear consciousness with both eyes open; he could explain its speed, color, size, and also told a red flag on it. The patient started to talk with high self-esteem content that he would become the Prime Minister soon, and lots of political meetings he had to arrange. He also explained that he could hear voices from stations or places far from his house, mainly personal voices or train whistles. The patient also reported that he had expertise in knowing 2 or 3 languages and also knew a lot about defense exams and he could take interviews related to national defense. The patient also started to visit nearby temples which initially he never visited. Seeing the severity of symptoms, family members prescribed some sedatives as advised by a local physician on the phone and the patient's sleep improved.

A computed tomography scan head plain revealed left-sided parietal hematoma and diffuse cerebral atrophy. Complete blood count was normal. Treatment was started with tablet haloperidol 2.5 mg HS, tablet donepezil 5 mg, and tablet Ginkoba OD. After 7 days, there was a 30% improvement in sleep and the treating psychiatrist suggested to visit our hospital.

General examination was normal and systemic examination revealed mild swelling in the right inguinal area. Upon respiratory

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examination, there were no added sounds, and cardiac S1 and S2 were normal. Cranial nerves and reflexes were intact and motor tone, bulk, and bilateral coordination were normal. The power was +5 bilaterally, and the sensory touch and pain were intact. Cortical sensations such as graphesthesia, stereognosis, and two-point discrimination were not intact. There were a bent posture and a short stepped gait. Upon mental status examination, he was oriented to time, place, and person, and hallucinatory behavior was present. There were increased psychomotor activity, speech tone, volume, rate, and pressure of speech. The mood was cheerful, elevated affect, increased reactivity, and appropriateness. Thought form had tangentiality. Content had delusions of grandiosity and religiosity, and auditory and visual hallucinations were present. Impaired judgment and insight was 1/6.

Upon investigations, hemoglobin was 11.8 mg/dl (12-16), Vitamin B12 was 166 Pg/ml (187-833), serum sodium was 132 meq./L (135-145), serum calcium was 4.03 mg/dl (4.5-10), and serum thyroid-stimulating hormone was 0.94 IU/l (0.45-4.94). Rest all reports were within normal range. A neurosurgery reference was taken on 18th September (3rd day) where they advised magnetic resonance imaging (MRI) of the brain with contrast and a neurology opinion was taken. MRI report showed a few changes and neurology consultation was taken.

They prescribed tablet amlodipine 2.5 mg OD and also neurosurgery reference was taken and advised no active intervention. Scale scores were as follows: YMRS-15/60, HMSE-25/30, Frontal Assessment Battery-10 BPRS, 2/126. MRI report showed late subacute bleed in the left parahippocampal region, left internal capsule, acute infarct in central semioval, and chronic infarct in the left temporal lobe. Our treatment during discharge was injection of Vitamin B12 1500 µg deep IM in the back 7 days, tablet nitrofurantoin 100 mg BD 5 days, tablet amlodipine 2.5 mg OD, tablet melatonin 10 mg HS, and tablet sodium valproate 200 mg BD. The patient did not maintain follow-up after that discharge.

DISCUSSION

Previous studies by Gafoor and O'Keane [7] showed that manic symptoms were mainly associated with the right-sided lesion of the brain and rarely with the left side, the main areas involved for the manic symptoms were mainly temporal and orbitofrontal areas of the right side. In our case, the left-sided parietal and temporal areas were involved which have not been associated with such symptoms [8]. In older cases, mania may be accompanied by the compromise of cognitive functions, which suspects in favor of dementia. In dementia, cognitive functions decline initially, and later on, psychosis and irritability occur, as well as, sundowning symptoms start to worse in the evening as reported in a study done by Schneider *et al*. [9], unlike in our case, where memory was intact and irritability and psychosis were the initial symptoms going in favor of organic mania.

Mendez in a study reported that mainly right-sided lesions involving the right temporal areas, medial temporal side, and basal ganglia are more commonly associated with manic symptoms, but in our case, the left-sided lesions were prominent, and also the parietal area associated which as per literature, is less associated with affective symptoms [10]. Depp and Lebowitz reported that the lesions in parahippocampal areas lead to some agnosia mainly word, topographical, and some visual defects associated with headaches but unlikely in our patient, no such symptoms were reported even having acute infarct lesions.

Cerebellar infarcts are generally shown as white matter hyperintensities on MRI symptoms such as unsteadiness, vomiting, dizziness, and vision problems may occur but such findings were not present in the patient, if damage occurs in the vermis of the cerebellum, the patient may present as psychosis as shown by a previous study which is similar to our patient having frank psychotic symptoms and MRI changes also in cerebellum bilaterally [11].

A previous study by Evans *et al*. [12] showed that structural causes of mania may respond better to mood stabilizers such as valproate or topiramate, so we plan to start the patient on sodium valproate with a low dose of 200 mg OD dose and plan to increase slowly, as liver function tests were normal too. Fenn and George [13] reported a rare case report in which the left-sided temporal infarction led to the first-episode mania in a 78-year-old male, similar to our case, where the left-sided chronic infarct was present and presented as mania. Our patient had psychotic symptoms which are similar to a study conducted by Masalha *et al*., which showed that Vitamin B12 deficiency in the elderly may remain hidden and cause episodic psychosis in 5.3% of elderly patients [14].

CONCLUSION

In late-life mania, a thorough workup that includes neuroimaging, vitamin profiles, thyroid profiles, urine examinations, and cardiac workups should be performed if an older person experiences mania for the 1st time in their life and has no prior medical history or substance use history. These tests all contribute to the affective symptoms that older people experience. New-onset mania in the geriatric population should raise an opinion to rule out vascular insult tumors or any CNS infection. The use of mood stabilizers such as valproate or topiramate in low doses is useful in the elderly mainly in structural lesions. Antipsychotics should be used very judiciously and more focus should be on treating the underlying organic etiology in such rare cases.

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