**Infected endocarditis presenting as depression: A case report**

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**Abstract**

Psychiatric disorders occur in many neurologic conditions like stroke and may be the only initial presentation, especially if the lesion involve left frontal region. Cardiac conditions like Infective endocarditis can result in embolism, which can lead to neurological complications which are the major prognostic determinants as it causes significant morbidity and mortality. Physical manifestations of the patient can be subtle. Hence caution needs to be taken to conduct a thorough physical examination even in young and seemingly healthy patients who present with psychiatric symptoms.

**Keywords:** Infective endocarditis(IE), Depression.

**Introduction**

Incidence of Infective endocarditis(IE) in western countries is 1.7 to 6.3 per 100000 person years with a 1 year mortality of 40% while definitive incidence is unpredictable in India.1 Among Indian population, younger age and male gender are more prone though mean age of incidence is increasing.1 Streptococci and staphylococci together cause 60-80% of IE1,2 but Streptococcus Viridans alone accounts for 30 to 65% cases of Native Valvular Endocarditis(NVE).10 The clinical features are characterized by a triad of heart murmur, fever and splenomegaly.1 The classical peripheral manifestations includes petechiae, splinter hemmorhages, Osler Nodes and Janeways Lesion.1 Embolic phenomena is a major complication and occurs in 10-35% cases, especially in left sided IE.3 High morbidity & mortality rates are associated with these complications.3-4 Stroke caused by IE accounts for 1.7% of all strokes5 and occurs in 16 to 25% of IE patients.5 Studies report that stroke can occur 1 to 4 months before and peak within 1 month period after making the diagnosis of IE.6 Manifestations can be neurologic in 20-40% and psychiatric in 10-15% cases.4 Depression occurs in 25 - 32% of strokes5,6 and may be the only initial presentation. This case highlights the importance of detailed physical examination even in healthy patients who present with psychiatric symptoms.

**Case Report**

35 year old, 12th completed, house wife, Y, from middle socio-economic status pre- morbidity well-adjusted with no history of medical co-morbidities like diabetes, hypertension, thyroid disorders and not on any regular medications with no precipitating factor presented with complaints of feeling sad, crying spells, decreased talk, decreased interest in daily activities, easy fatigability, death wishes, feeling hopeless and worthless, impaired sleep and weight loss for 4 months. She developed walking difficulty 1½ months after depressive symptoms began. There is no past history of depression, head injury, substance use and premenstrual dysphoria.

There is no history of intellectual impairment, impaired sensorium, self-neglect and violent behaviour.

A plain CT Brain done in another hospital showed normal. She was referred to a psychiatrist by the clinician and was treated for severe depression with Escitalopram 10 mg and Clonazepam 0.5mg for over a month, but symptoms persisted. Later, she developed paraesthesia of limbs, difficulty in getting up and headache and hence was referred to PIMS and got admitted under Neurology. Initial examination revealed depressed symptoms on MSE and detailed physical examination revealed signs and symptoms of IE. Findings on physical examination were raised JVP, Pallor, Clubbing, Mild Bilateral Pitting pedal oedema, Splinter haemorrhages in left great toe (Fig. 1), Janeways spot lesions, hypertension and Brisk DTR on right side, pansystolic murmur in mitral area. MSE revealed-kempt appearance, looking gloomy, crying spells, decreased PMA, decreased speech, preoccupied with walking difficulty, depressive cognition(+), death wishes (+), depressed mood, intact cognitive functions, grade 3 insight. Investigations showed Haemoglobin - 10.7g%, ESR -115 mm/hr, CRP: 9.17 mg /dl. MRI Brain (Fig. 2): A small well defined lesion in left high frontal regions with contrast enhancement suggestive of Cavernoma/ Tuberculoma; A well-defined altered signal intensity lesion in left high frontal region adjacent to above lesion with blooming suggestive of late sub-acute hematoma. CT Brain (Fig. 3): An ill-defined hypodensity in left high frontal regions suggestive of sub-acute infarct with resolving haemorrhagic transformation –? Secondary to septic emboli. ECHO: Dilated LA, MVP with Severe Eccentric MR, TVP with TR, Vegetation (15 x 9 mm) seen on AML, Moderate PAH, No RWMA, Good Biventricular Systolic function. Blood culture revealed Streptococcus viridans. Patient was treated with IV Ampicillin, IV Gentamicin. Mitral valve replacement was done. Patient was diagnosed with severe depression without psychotic symptoms and was treated with Escitalopram15mg and Clonazepam1mg which were continued for 1 ½ months, depressive cognitions and death.
wishes subsided. Patient has no residual focal neurological deficit and is currently maintained on cardiac medications.

**Fig. 1:** Splinter hemorrhages in left great toe

**Fig. 2:** MRI Brain: A small well defined lesion in left high frontal region

**Fig. 3:** CT Brain: An ill-defined hypo density in left high frontal region

**Discussion**
Cardio embolic stroke accounts for a majority of cerebral infarctions and IE is one of the major causes. Studies report high risk of stroke in Infective endocarditis. The stroke can be subtle, or the so called Silent brain infarct (SBI). Many studies report SBI to be closely associated with cardiac illness. Prevalence of SBIs is reported to be 10-20%. Studies report that most likely vegetation to embolize are those measuring ≥10mm in size and those located on anterior mitral valve leaflet as in the case of our patient. Diagnosis of infective endocarditis per se may be made only months after the occurrence of emboli and in some patients the only initial presentation may be depressive symptoms. Numerous psychiatric conditions have been linked to SBIs including mood disorders and dementia. Major depressive disorder has been noted in 20% of patients with cardiovascular disease and 40 to 50% of patients with SBIs. Our patient had a lesion in left high frontal region which concedes with studies that suggests that depression is more common in left frontal strokes which is associated with left hemispheric hypoactivity and hypometabolism. Left hemisphere processes pleasurable experiences and decision making, and its attenuation was reported to lead to anhedonia and indecisiveness because of lower serotonin binding in left hemisphere strokes. Also in left hemisphere stroke, there is relative hyperactivity of right hemisphere which processes negative emotions, pessimistic thoughts and unconstructive thinking styles leading to depressive cognition. Left dorsolateral prefrontal cortex and left frontal pole has also been implicated in depressive disorders.

To conclude, psychiatric disorders like depression occur in many neurologic conditions like stroke and may be the only initial presentation, especially if the lesion involve Left frontal region. Neurologic complications are major prognostic determinants of infective endocarditis as it causes significant morbidity and mortality. Hence caution needs to be taken to conduct a thorough physical examination even in young and seemingly healthy patients. This also highlights the significance of neuroimaging and consultation liaison in psychiatric practice. Timely detection can help save life as well as improve its quality as happened in our patient’s case.

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None.

**Conflict of Interest**
The authors declare that there is no conflict of interest.

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