

Infective endocarditis presenting as depression: A case report

Keerthy Reji¹, Sivin P Sam², Roy Abraham Kallivayalil^{3*}

¹Senior Resident, ²Junior Resident, ³Professor and HOD, Dept. of Psychiatry, Pushpagiri Institute of Medical Sciences and Research Center, Thiruvalla, Kerala, India

*Corresponding Author: Roy Abraham Kallivayalil

Email: roykalli@gmail.com

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 IE^{1,2} but Streptococcus Viridans alone accounts for 30 to 65% cases of Native Valvular Endocarditis(NVE).^[1] The clinical features are characterized by a triad of heart murmur, fever and splenomegaly.¹ The classical peripheral manifestations includes petechiae, splinter hemorrhages, Osler's Nodes and Janeway's 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.⁶ Studies reports 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 neurological in 20-40% and psychiatric in 10-15% cases.⁴ Depression occurs in 25 - 32% of strokes^[7,8] 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- morbidly 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 depressive 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), Janeway's spot lesions, hypertonia and Brisk DTR on right side, pansystolic murmur in mitral area. MSE revealed dull-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 hypo density 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 Escitalopram 15mg and Clonazepam 1mg 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

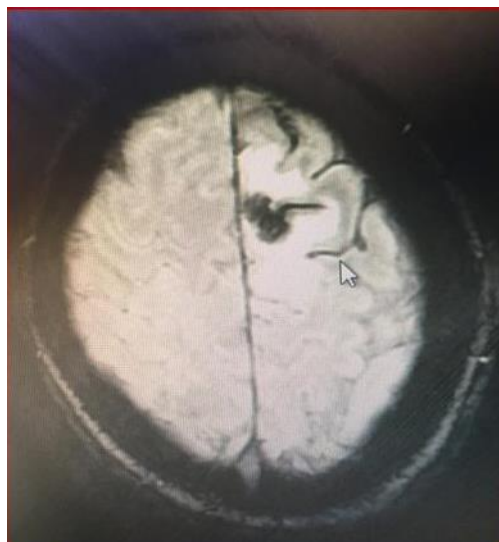


Fig. 2: MRI Brain: A small well defined lesion in left high frontal region



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.³ Studies report high risk of stroke in Infective endocarditis.^{5,6,9} The stroke can be subtle, or the so called Silent brain infarct(SBI).¹⁰ Many studies report SBI to be closely associated with cardiac illness.¹⁰ Prevalence of SBIs is reported to be 10-20%.¹⁰ Studies report that most likely vegetation to embolize are those measuring ≥ 10 mm in size and those located on anterior mitral valve leaflet as in the case of our patient.^{4,6} Diagnosis of infective endocarditis per se may be made only months after the occurrence of emboli⁶ 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.¹⁰ Major depressive disorder has been noted in 20% of patients with cardiovascular disease and 40 to 50% of patients with SBIs.¹⁰ 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.⁷ 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.⁷ 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.⁷ Left dorsolateral prefrontal cortex and left frontal pole has also been implicated in depressive disorders.⁷

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.

Source of Funding

None.

Conflict of Interest

The authors declare that there is no conflict of interest.

References

1. Gupta R. Infective Endocarditis: Indian Scenario. *Index Med Update*. 2013;29:135-41.
2. Abhilash KP, Patole S, Jambugulam M, Sathyendra S, Mitra S, Rebekah G, et al. Changing trends of Infective endocarditis in India: A South Indian experience. *J Cardiovasc Disease Res*. 2017;8(2):56-60.
3. Grecu N, Cristina TI, Terecoasa E, Bajenaru O. Endocarditis and stroke. *Maedica*. 2014 Dec;9(4):375.

4. Sonnevile R, Mirabel M, Hajage D, Tubach F, Vignon P, Perez P, et al. Neurologic complications and outcomes of infective endocarditis in critically ill patients: the END Ocardite en RE Animation prospective multicenter study. *Crit Care Med*. 2011;39(6):1474-81.
5. Cao GF, Liu W, Bi Q. Stroke in patients with infective endocarditis: a 15-year single-center cohort study. *Eur Neurol*. 2018;80(3-4):171-8.
6. Merkler AE, Chu SY, Lerario MP, Navi BB, Kamel H. Temporal relationship between infective endocarditis and stroke. *Neurol*. 2015;85(6):512-6.
7. Grajny K, Pyata H, Spiegel K, Lacey EH, Xing S, Brophy C, Turkeltaub PE. Depression symptoms in chronic left hemisphere stroke are related to dorsolateral prefrontal cortex damage. *J Neuropsychiatry Clin Neurosci*. 2016;28(4):292-8.
8. Hackett ML, Pickles K. Part I: frequency of depression after stroke: an updated systematic review and meta-analysis of observational studies. *Int J Stroke*. 2014;9(8):1017-25.
9. Kamel H, Healey JS. Cardioembolic stroke. *Circ Res*. 2017;120(3):514-26.
10. Indja B, Seco M, Seamark R, Kaplan J, Bannon PG, Grieve SM, Vallely MP. Neurocognitive and psychiatric issues post cardiac surgery. *Heart Lung Circ*. 2017;26(8):779-85.

How to cite this article: Reji K, Sam SP, Kallivayalil RA. Infective endocarditis presenting as depression: A case report. *Telangana J Psychiatry*. 2020;6(2):176-178.