ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 11 | November - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

SEIZURE AND THE HEART: A RARE COMBINATION

Neurology

Deepika Sagar Associate professor and head, Department of Neurology, L.L.R.M. medical college Meerut.

Dheeraj Kumar Soni* Associate professor and Head, Department of Cardiology, L.L.R.M. medical college Meerut. *Corresponding Author

ABSTRACT

Population studies showed increased evidence of cardiovascular events in patients with seizures. Multiple pathophysiology have been told including accelerated atherosclerosis, myocardial infarction, coronary vasospasm, altered autonomic tone, atrial and ventricular arrythmias. These effects are due to increased catecholamines release and hypoxic damage. The occurrence of ictal and post ictal arrythmias is currently a cause of great scientific debate.

KEYWORDS

INTRODUCTION

Anatomical and functional connections between the brain and heart in both health and disease have long been established. Cardiac arrythmias and ST segment changes have been observed with acute intracerebral events such as subarachnoid hemorrhage or cerebrovascular accidents[1] and more recently, the interaction of the heart and brain in patients with epilepsy has been the subject of intense scrutiny. This has been driven by the publication of a number of important studies that have documented the frequent occurrence of cardiac rhythm changes during epileptic seizures.[2,3]

It is important to note, however that although small series observational studies are helpful in raising awareness of the poorly understood entity, unfortunately they have little capacity to inform the debate about the possible mechanisms, risk factors and preventive measures.

Arrythmias, conduction block and repolarization ECG abnormalities, such as Atrial fibrillation, marked sinus arrythmias, supraventricular tachycardia, atrial and ventricular premature depolarization, bundle branch block, high grade atrioventricular block, ST depression and T wave inversion have been reported in up to 56% of seizures and are more common in nocturnal, prolonged and generalized seizures.[4,5] Sinus rate change is the most common cardiac involvement to ictal discharge with sinus tachycardia reported in 50-100% of patients with seizures.[6]

We are presenting the case series of 4 patients showing association of seizures and cardiac involvement.

Case 1.

A 48 years old male smoker presented in the emergency department with chief complaint of breathlessness, chest pain and swelling in both feet past 10-15 days. There was no history of prior MI, diabetes, hypertension, kidney disease, COPD. On cardiovascular examination, S1, S2 was normal, S3 present. Patient also had a history of orthopnea for past 2 days. Electrocardiograph (ECG) shows left bundle branch block, QRS more than 0.14 sec, with sinus tachycardia. Troponin T was negative.

2D ECHO of the patient shows severe left ventricular systolic dysfunction, global hypokinesia with left ventricular ejection fraction (LVEF) 25%. There was grade III diastolic dysfunction with moderate mitral regurgitation. Patient was admitted and treatment started in the form of diuretics, B-blockers, antiplatelets and sacubitril valsartan. Blood investigations was within normal range.

On day 2, patient developed 2 episodes of generalized tonic-clonic seizures with tongue bite and urinary incontinence. Patient became unconscious. Antiepileptic loading (valproic acid 20mg/kg wt) was given and he kept on lateral position. ECG of the patient shows acute atrial fibrillation so patient was DC cardioverted with 90 J DC shock. (figure 5) Patient regained conscious after 2 hours. Electroencephalograph (EEG) shows generalized interictal epileptiform discharges suggestive of temporal lobe involvement (figure 1). CT scan was normal with mild cerebral oedema. Patient managed conservatively thereafter and discharge in stable condition on antiepileptic and cardiac medications.

Case 2.

A 50 years old female presented In the emergency department with 3 episodes of generalized tonic colonic seizures past 8 hours. She was unconscious with frothing from the mouth, tongue bite and difficulty in breathing. On examination, there was coarse crept in the bilateral lungs, both plantar was extensor. Glasgow Coma Scale (GCS) was E1M3V1, so patient was immediately intubated and kept on mechanical ventilation.

CVS examination shows normal S1 and S2 sound. S3 was present with pansystolic murmur over the apical area. ECG shows irregular wide complex tachycardia with atrial flutter (figure 6). Intravenous antiepileptic and antibiotics with mannitol was given. EEG shows generalized monomorphic interictal epileptiform discharges (figure 2). She regained consciousness on day 2 with mild basal crept and followed command on day 3. GCS was E4M6VT, so she was extubated and kept on oxygen support.

2D ECHO shows severe mitral stenosis, moderate mitral regurgitation, dilated left atrium, LVEF 35% with global hypokinesia of left ventricle. CT scan shows cerebral oedema with old temporal and frontal infarcts. Patient was discharged on cardiac and antiepileptic medications on day 10 and advised mitral valve replacement.

Case 3.

A 21 years old male was presented with 4 episodes of generalized tonic colonic seizures past 4 hours. There was no past history of seizures or any medications. No other significant history was present. He was unconscious with urinary incontinence. Loading was given in the form of intravenous phenytoin (10mg/kg wt) and he regained consciousness in 45 minutes. Cardiovascular examination shows normal heart sound with S3 present. There was a pansystolic murmur present over the tricuspid area. Plantar flexor with no respiratory involvement. ECG shows sinus tachycardia with right ventricular hypertrophy, right axis deviation.

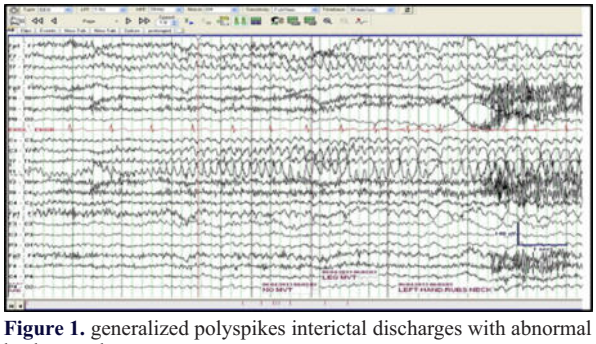

Figure 1. generalized polyspikes interictal discharges with abnormal background

CT scan was normal. EEG shows generalized polyspikes interictal discharges suggestive of frontal lobe involvement (figure 3). 2D ECHO shows normal left ventricular function, severe right ventricular enlargement with systolic dysfunction, severe low pressure tricuspid regurgitation, apical displacement of septal tricuspid leaflet was more than 20mm suggestive of Ebsteins anamoly. Patient was managed on antiepileptics, diuretics, b-blockers and discharged on day 5.

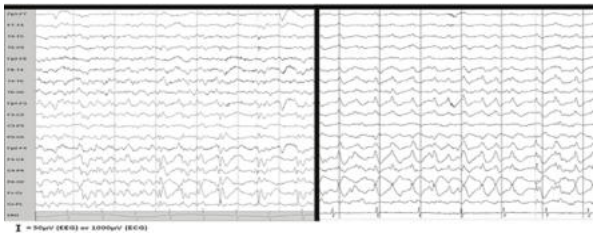

Figure 2 Atypical Generalized Monomorphic Interictal Discharges With Delta Waves.

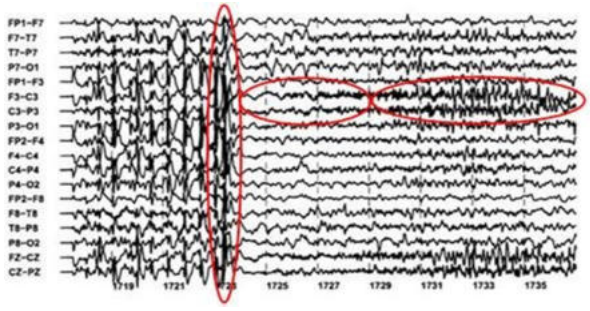

Figure 3 Generalized Polyspike Waves With Intermittent Periodic Epileptic Discharges.

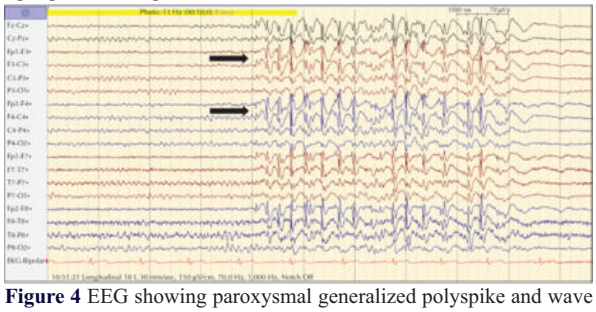

Figure 4 EEG showing paroxysmal generalized polyspike and wave discharges.

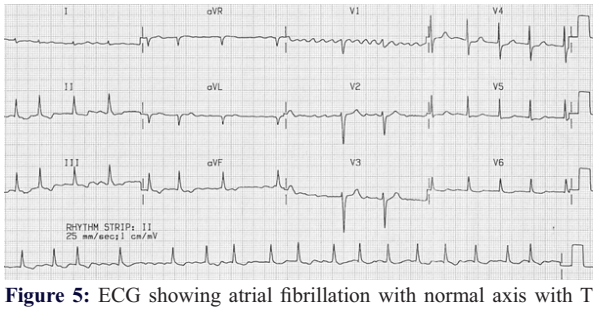

Figure 5: ECG showing atrial fibrillation with normal axis with T inversion lead II,III, aVF.

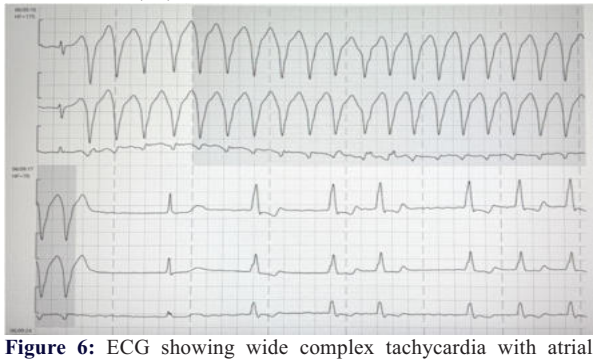

Figure 6: ECG showing wide complex tachycardia with atrial fibrillation, left axis deviation.

Case 4.

A 32 years old male presented with refractory generalized tonic colonic seizures with loss of consciousness past 5 hours. There was past history of seizures and he was taking some irregular antiepileptic medications since last 1 year. There was also history of alcoholism with drug abuse. GCS score was E1M2V2, so he was immediately intubated and kept on mechanical ventilation. Both plantar was extensor, pupil was semidilated. He was started antiepileptic (valproic acid, levetiracetam) and mannitol but seizures did not controlled. So the patient was sedated with intravenous midazolam (0.2mg/kg wt).

After 6 hours patient went into shock with nonrecordable pulse and blood pressure. ECG shows irregular wide complex tachycardia with rate more than 210 per minute. He was cardioverted with 150 joules DC shock. Serum calcium and magnesium level was very low (Ca <2.5mmol, Mg<1mmol) which was supplemented.

Patient regained consciousness, extubated on day 3, seizures controlled and ECG shows normal sinus rhythm. EEG shows generalized interictal epileptiform discharges suggestive of temporal lobe involvement (figure 4). 2D ECHO shows normal left and right ventricular function with normal valves. He was discharged on day 12 in stable condition.

DISCUSSION

Sinus rate change is the most common event with ictal discharge with sinus tachycardia reported in 50-100% of seizures. Although the heart rate in ictal tachycardia is typically 100-120 beats per minute, there are reports of rates exceeding 170 beats per minute even during simple partial seizures.[7]

Ictal tachycardia is most commonly seen in early ictal phase of seizures while bradycardia during late ictal phase. There is some evidence for right sided cerebral lateralization and temporal lobe localization in patients with ictal tachycardia.

A recent literature review by Britton revealed that of 65 cases of ictal bradycardia with sufficient EEG and ECG data, seizure onset was localized to the temporal lobe in 55%, the frontal lobe in 20%, the frontotemporal region in 23% and the occipital lobe in 2%.[8]

Information regarding seizure onset lateralization was available in 56 cases. Seizure onset was lateralized to the left hemisphere in 63%, the right in 34% and bilaterally in 4%.[8,9]

It is possible that patients with epilepsy are predisposed to developing cardiac arrythmias during seizures due to number of factors. These include antiepileptic medications, genetic susceptibility and cardiac channelopathies.

Minor, non specific pathological change like atherosclerosis, conducting system fibrosis and myocyte vacuolization has been the major cardiac changes in the patients with epilepsy. However, the patients with seizures usually have normal coronary arteries. It has been postulated that neurogenic coronary vasospasm may be implicated.[10]

Very few case series are present in the literature showing association between seizures and cardiac disease. In our study we found that patients with seizures may have significant underlying cardiac disease, so in all cases we should rule out cardiac disease for better management.

CONCLUSION

The earliest and best known effect of epilepsy on the cardiovascular system is the increase in the heart rate due to adrenaline release and increased circulatory demand; the latter is especially evident in tonic-clonic seizures. Patients with left temporal lobe epilepsy showed increased heart rate after EEG changes.

Tachycardia and rhythm changes is more frequent and long lasting in patients with temporal lobe epilepsy since propagation of electric discharges is also easier and long lasting than in the case of extratemporal epilepsy.

REFERENCES:

1. Goldstein DS. The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings. *Stroke* 1979;**10**:253-9.
2. Rugg-Gunn FJ, Simister RJ, Squirrell M et al. cardiac arrythmias in focal epilepsy: a

prospective long term study. *Lancet* 2004;**364**:2212-19.
3. Blumhardt LD, Smith PE, Owen L. electrocardiographic accompaniments of temporal lobe epileptic seizures. *Lancet* 1986;1:1051-6.
4. Samuels MA. Neurogenic heart disease: a unifying hypothesis. *Am J Cordiol* 1987;**60**:15J-19J.
5. Altenmuller DM, Zehender M, Schulze-Bonhage. A high grade atrioventricular block triggered by spontaneous and stimulation-induced epileptic activity in the left temporal lobe. *Epilepsia* 2004;**45:**1640-4.
6. Zijlmans M, Flanagan D, Gotman J. heart rate changes and ECG abnormalities during epileptic seizures: prevalence and definition of an objective clinical sign. *Epilepsia* 2002;**43**:847-54.
7. Leutmezer F, Schernthaner C, Lurger S *et al*. Electrocardiographic changes at the onset of epileptic seizures. *Epilepsia* 2003;*44*:348-54.
8. Britton JW, Ghearing GR, Benarroch EE *et al*. The ictal bradycardia syndrome: localization and lateralization. *Epilepsia* 2006;*47*:737-44.
9. Swartz CM, Abrams R, Lane RD *et al*. heart rate differences between right and left unilateral electroconvulsive therapy. *J Neurol Neurosurg Psychiatry* 1994;**57**:97-9.
10. Pirolo JS, Hutchins GM, Moore GW. Myocyte vacuolization in infarct border zone is reversible. *Am J Pathol* 1985;**121**:444-50.