Every Syncope is not Benign

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Abstract

Introduction: Syncope is a diagnostic challenge to treating physician. Most of the cases have a benign course. But cardiogenic syncope has a worse outcome unless managed. Here we present a case of a 38 year old presents with recurrent history of syncope since 2 years. Her vitals were normal. ECG and echo cardiogram were normal. 24 hour holter done 2 years back did not show any arrhythmia. We did an extended Holter monitoring which showed 42 episodes of pauses. So we done an Urgent Permanent pacemaker implantation DDD PPI with good results. This emphasizes that patients presenting with syncope but with normal ecg should undergo extensive cardiological evaluation for AV block. Syncope is sudden and transient loss of consciousness and postural tone attributed to inadequate cerebral blood flow. It can be classified as neurally mediated, cardiac and orthostatic hypotension. Neurally mediated syncope is most common and has a benign course but cardiogenic syncope is associated with increased mortality and morbidity.

Keywords: Syncope; paroxysmal AV block; extended holter monitoring; electrophysiological study; pause-dependent phase 4 av block; his-purkinje system.

Abbreviations: AV: Atrio Ventricular; CNS: Central Nervous System; CT: Computed Tomography; DDD: Dual Chamber Sensed, Paced and Response To Sensed Event; ECG: Electrocardiogram; EEG: Electroencephalogram; ENT: Ear Nose Throat; MRI: Magnetic Resonance Imaging.

Case Report

38 year old staff nurse with no prior co-morbidities presented with transient loss of consciousness lasted for 1-2 minutes for the last 2 years associated with blurring of vision. No report of seizure activity or post ictal state. She was relatively asymptomatic till the last 2 months. During the last episode she was driving and fell down from the vehicle and sustained injury to the back of head. 2 years back when she had the first episode, was evaluated from cardiology department where she had normal blood pressure, pulse rate and ecg. 24 hour Holter monitoring did not show any arrhythmia. Neurological evaluation with CT head, MRI brain, EEG studies were normal and ENT evaluation also could not detect any etiology.

After that she had recurrent episodes of syncope lasting for 1 minute once in a week. Within this 2 year she was evaluated by doing ECG, EEG. She had a recent episode of syncope while at home to such an extent that her son had to give CPR. She became conscious immediately. On examination Pulse rate- 62/mt, regular rhythm, normal volume, all peripheral pulses palpable, blood pressure was 120/70mmhg, SPO2-98%, Cardiovascular System Examination-within normal limit, CNS-no focal neurological deficit. On examination Pulse rate- 62/mt, regular rhythm, normal volume, all peripheral pulses palpable, blood pressure was 120/70mmhg, SPO2-98%, Cardiovascular System Examination-within normal limit, CNS-no focal neurological deficit.

Figure 1: On examination Pulse rate- 62/mt, regular rhythm, normal volume, all peripheral pulses palpable, blood pressure was 120/70mmhg, SPO2-98%, Cardiovascular System Examination-within normal limit, CNS-no focal neurological deficit.
Discussion

• Paroxysmal AV block with recurrent syncope

• Type of AV block is defined as a paroxysmal third-degree AV block [1] that exhibits abrupt onset, with no other rhythm disturbances before or during the block, and occurs in patients with a normal ECG and a normal heart

• An abrupt and unexpected complete AV block in a patient with otherwise 1:1 AV conduction, with delayed ventricular escape

• Classically described as pause-dependent Phase 4 AV block,

• Associated with a diseased His-Purkinje system (HPS). Phase 4 (pause-dependent or bradycardia-dependent) aberrancy or block occurs when the supraventricular impulse is unable to conduct in the diseased HPS.

• This is a consequence of partial depolarization in the HPS occurring during the latter part of Phase 4, leading to inactivation of sodium channels, unable to cause a complete depolarization by itself.

• Intrinsic paroxysmal AV block is triggered by a pause following a PVC, a premature atrial complex, or an abrupt change in heart rate when the impulse reaching the HPS finds it inactive and is unable to conduct.

• When a critical membrane potential is reached after a criti-
cally timed pause, the sodium channel continues to be inactive and unavailable for conduction till the membrane potential is reset to an excitable state by another premature depolarization.

Electrophysiological study has limited specificity and sensitivity for detecting alterations in AV conduction.

**CHB-clinical criteria**: CHB seen on ECG or ambulatory monitoring in the presence of symptoms with or without hemodynamic compromise.

**Paroxysmal AV block**: No rhythm disturbances before or during the block, and occurs in patients with a normal ECG and a normal heart

**Conclusions**

When patient presenting with syncope eventhough ECG is normal, they should undergo extended holter monitoring and look for av block and symptom rhythm correlation.

**References**

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