Case Report

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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 arrythmia. 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 arrythmia. 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.

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						SL	ımm	ary	Report						
Report Numbe	# : 7C/	A1137E3	07121	5		Start	Time			12:29	31 1	M	Total Beats		1464
Test Date	1.11	8/2010				Hour	a Anal	lyzed		23:0	58 :	29	Unknown Deats	16)
rest care	1 1/1	DISAIN				Ante	BCR.			0:	00 : :	27	Other Beats)
Report Date	: 1/11	9/2019											Percent AFIB	:6)
Heart Rates						Ra	te De	pend	ent Events						
Min : 59 I	0PM at	05.33	1.00-2	Br	adyoardia Runs			0					Pausos :	0	
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Max VE/Hour		1	beats		18:00:00-				Max EVE/Hour			1	beats	08.00.00.2	
Mean VE/Hour		0.0							Mean SVE/Hou	ar .		0.0			
VE/1000		0.0							fivE/1000			0.0			

Figure 2: Summary report.



Figure 3: Pulse rate.

		5	Supraventricular	Arrhythmias				
Supraventricular Beats	: 41 (<1%)	MaxHour :	6 (Tue 31, 06.00 PM)	MaxMin	ute: 3 (Tue	31, 04.18 PM)	AverageHour: 1	
Event	Event	SV Beat Count	SV Beats	Total Beats	Per 1000	Max Rate (tipm)	Longest (beats)	
SVT	0	0	0%	0%	0			
SVE Run	0	0	0%	0%	0			
SVE Couplet	1	2	5%	41%	0			
Single SVE Events	40	40	895	+1%	0			
Total duration 48 hours	and 23 minutes, to	tal beats 35128						
Total duration 48 hours The everage heart rate	and 23 minutes, to was 81 bpm, with a	tal beats 35128	8. 12 bpm at 06:24:00-1	2 and a minimu	um of 54 bp	m at 05:49:00-3,		
Total duration 48 hours The average heart rate No Ventricular ectopics.	and 23 minutes, to was 81 bpm, with a	tal beats 35128	8. 12 bpm at 06:24:00-	2 and a minimu	um of 54 bp	m at 05:49:00-3,		
Total duration 48 hours The average heart rate i No Ventricular ectopics. 41 supraventricular ecto	and 23 minutes, to was 81 bpm, with a pice.	tal beats 35128	8. 12 bpm at 06:24:00-	2 and a minimu	um of 54 bp	m at 05:49:00-3,		
Total duration 48 hours The average heart rate of No Ventricular ectopics. 41 supraventricular ecto 5 episodes of Bradycard	and 23 minutes, to was 81 bpm, with r pics. Sa, minimum rate v	tal beats 351280 maximum of 12 vas 9 bpm at 10	8. 12 bpm at 06:24:00- :43:03-2.	2 and a minim	um of 54 bp	m at 05:49:00-3,		

Figure 4: Patient's Echocardiogram, blood and metabolic parametres within normal limits. There were 42 episodes of pauses, the longest was 14.57sec. So we done an Urgent Permanent pacemaker implantation DDD PPI with good results.



Figure 5: Paroxysmal AV block with recurrent syncope.

Features	Intrinsic /W block (I-AVB) Cardiac syncope	Extrinsic vagal AV block (EV-AVB) Reflex syncope	Extrinsic idiopathic AV block (EI-AVB) adenosine syncope
ECG			
Sinus rhythm	BBB frequent	888 infrequent	Narrow QRS
Before AVB	 Sometimes AVB initiated by APB or VPB PR unchanged 	 Progressive sinus rate slowing (P-P cycle increase) Generally progressive PR prolongation 	 Sinus rate unchanged (P-P cycle unchanged) PR unchanged
During asystolic AVB	Sinus rate increase (P-P cycle decrease)	Sinus rate slowing (P-P cycle increase)	Sinus rate unchanged (P-P cycle unchanged)
End of AVB	- Sometimes AVB interrupted by APB or VPB	Sinus rate acceleration (P-P cycle decrease)	Sinus rate unchanged (P-P cycle unchanged)
Follow-up Smoone	Progression to persistent AVB	No progression to persistent AVB	No progression to persistent AVB
History of syncope	Short (mostly < 1 year)	Long (since youth)	Short (average 2 years)
Prodromes	No or very short (< 5 s) prodromes	Always present > 10 s	No or very short (< 5 s) prodromes
Structural heart disease	Mostly present	Mostly absent	Absent
Age on presentation	Elderly	Any age	Any age, mostly over 40 years
Efficacy of pacemaker therapy	Effective	Partially effective	Effective
Efficacy of theophylline therapy Investigations	Ineffective	Partially effective	Effective
Plasma adenosine value	Normal	High	Low or very low
Adenosine (ATP) test	Usually negative	May be positive	Frequently positive (asystolic 3rd degre- block)
Tilt table test	Usually negative	Mostly positive	Mostly negative
Electrophysiological study	Frequently positive	Negative	Negative
Carotid sinus massage	Usually negative	Frequently positive	Negative

Figure 6: Comparison of three forms of paroxysmal AVB.



(*) High risk features: syncope supine or during exertion or preceded by sudden onset palpitations

Figure 7: Distinctive features of the three types of paroxysmal AVB. AVB= Atrioventricular block, BBB= Bundle branch block; CSM= Carotid sinus massage; TT= Tilt testing; SHD= Structural heart disease; EPS= Electrophysiological study.

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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