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Case Report

SPONTANEOUS THIRD-DEGREE ATRIOVENTRICULAR BLOCK IN A DIABETIC PATIENT PRESENTING AS RECURRENT SYNCOPE

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ABSTRACT

Chronic, uncontrolled hyperglycemia is associated with an increased incidence of coronary artery disease, cardiac failure, diabetic nephropathy, diabetic retinopathy, and associated mortality. A 72-year-old female presented to the OPD with multiple episodes of loss of consciousness for the past 1 month. The patient also had other comorbidities such as hypertension, diabetes mellitus (DM), and hypothyroidism. On systemic examination, her blood pressure was 124/77 mmHg (supine) and 116/72 mmHg (standing), and her pulse rate was 56 beats/min. She had a loss of vibration sense in both lower limbs up to the ankle. Fundus examination showed non-proliferative diabetic retinopathy. The rest of the systemic examination was clinically normal. Her HbA1C was 8.1%, and other routine investigations were within normal limits. Electrocardiography (ECG) showed sinus bradycardia. Echocardiography showed normal ventricular function with no evidence of ischemic heart disease (IHD). 24-h Holter ECG revealed sinus bradycardia with an intraventricular conduction defect, a third-degree AV block with junctional beats, and rare supraventricular ectopics. This is a case of Type 2 DM with complete heart block (CHB) of spontaneous onset. Other causes of AV block have been ruled out, and it seems that this case of CHB is possibly due to cardiac autonomic neuropathy (CAN). Multiple factors, such as the duration of diabetes, poor glycemic control, metabolic derangements, and genetic factors, determine CAN. This case emphasizes that patients with type 2 diabetes without IHD can develop CHB spontaneously.

Keywords: Complete heart block, Type 2 diabetes mellitus, Cardiac autonomic neuropathy.

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INTRODUCTION

Diabetes Mellitus is rising to an alarming epidemic level in low and middle income nations like India. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels. Cardiac autonomic neuropathy (CAN) associated with DM damages the autonomic fiber's innervation of the heart, resulting in heart rate abnormalities and vascular dynamics. The survival rate in DM is affected by CAN. Several case studies have demonstrated an increased correlation between T2DM and third-degree heart block, brought on by CAN and genetic factors [1,2].

CASE PRESENTATION

A 71-year-old female was hospitalized for complaints of recurrent syncopal episodes, which drastically reduced her quality of life. Syncope started appearing 1 month before, at a rate of three to four episodes a day, each lasting for 5–10 s, followed by spontaneous recovery. There was no history of chest pain, palpitations, abnormal body movements, nausea, or vomiting. Her father had CAD. The patient was a known case of Type 2 DM for the past 20 years, treated with tablet metformin 500 mg BD; hypothyroidism for the past 6 years, treated with tablet thyroxine 75 mcg OD; and systemic hypertension for the past 4 years, treated with tablet telmisartan 40 mg OD. She also had a post-modified radical mastectomy for a carcinoma breast 20 years ago and is currently on tamoxifen (Table 1).

The patient's consent was taken for the publication of information about them in a journal. There were no abnormalities discovered during the physical examination. She was conscious and oriented to time, place, and person. Her height was 170 cm, her weight was 67.5 kg, and her body mass index was 23.4 kg/m^2. Pulse rate was regular and felt in all extremities (56 beats/min), and blood pressure (BP) was 124/74 mmHg in the supine position and 116/72 mmHg in the standing position, with no murmurs on cardiovascular examination. Auscultation of the lung was normal, with a respiratory rate of 18 breaths per minute, a SpO_2 of 99% on room air, and no peripheral edema. The abdominal examination was clinically normal.

Nervous system examination and fundus examination revealed bilateral loss of vibration sense in the lower limbs up to the ankle and bilateral non-proliferative diabetic retinopathy, respectively. The recommended basal evaluation of syncope and transient loss of consciousness (TLOC), including history, systemic examination, BP measurements, and relevant hematological investigations yielded no pathological findings (Table 2). For CAN, autonomic function testing revealed decreased sympathetic and parasympathetic reactivity in the moderate CANS dysfunction study (Table 3). The electrocardiography (ECG) showed bradycardia with no bundle branch block (Fig. 1).

We focused on more thoroughly examining a possible cardiac cause of TLOC. Echocardiography showed no valvular dysfunction or other structural diseases, with a normal ejection fraction. A 24-h Holter ECG revealed sinus bradycardia with an intraventricular conduction defect, third-degree heart block with junctional beats, and rare supraventricular ectopics (Figs. 2 and 3).

As per the ESC guidelines, a permanent pacemaker was inserted, a post-pacemaker insertion ECG was taken, and the patient was discharged (Fig. 4). At a follow-up consultation 1 month later, the patient reported no new episodes of TLOC, which resulted in a substantially improved quality of life [3].

DISCUSSION

It is evident from the above findings that this is a case of DM with an acute onset of complete heart block (CHB). The CHB may be attributed to the development of CAN in this patient. The association between DM and CAN involving the sympathetic and parasympathetic nervous systems is well established and is responsible for higher mean heart rate arrhythmia and death. There is also an increased prevalence of right/left bundle branch block (RBBB/LBBB) and bifascicular block among patients with diabetes. Chronic hyperglycemia leads to increased production of reactive oxygen species and causes oxidative damage to the microvasculature supplying these nerves, resulting in poor vasomotor autoregulation. As a result, sympathetically mediated vasodilation of coronary vessels is affected. The calcium/calmodulin pathway in the cardiac muscles changes due to vasoconstriction, which also causes a fibroblastic response, resulting in fibrosis of the heart's conduction system. Longer refractory periods within the ventricles due to this fibrosis cause inhomogeneity in the refractory period and repolarization, which results in CHB [2]. Krishna *et al*. also found that patients with diabetes have a higher incidence of first-degree heart block and CHB [4].

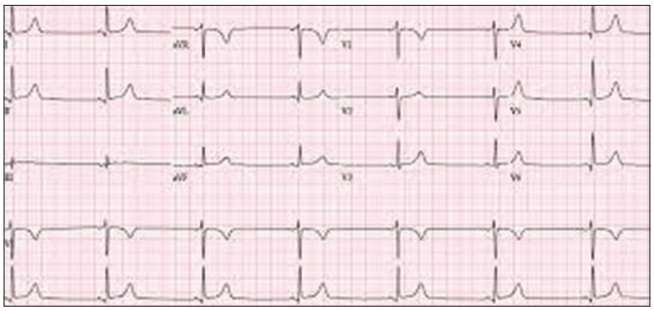
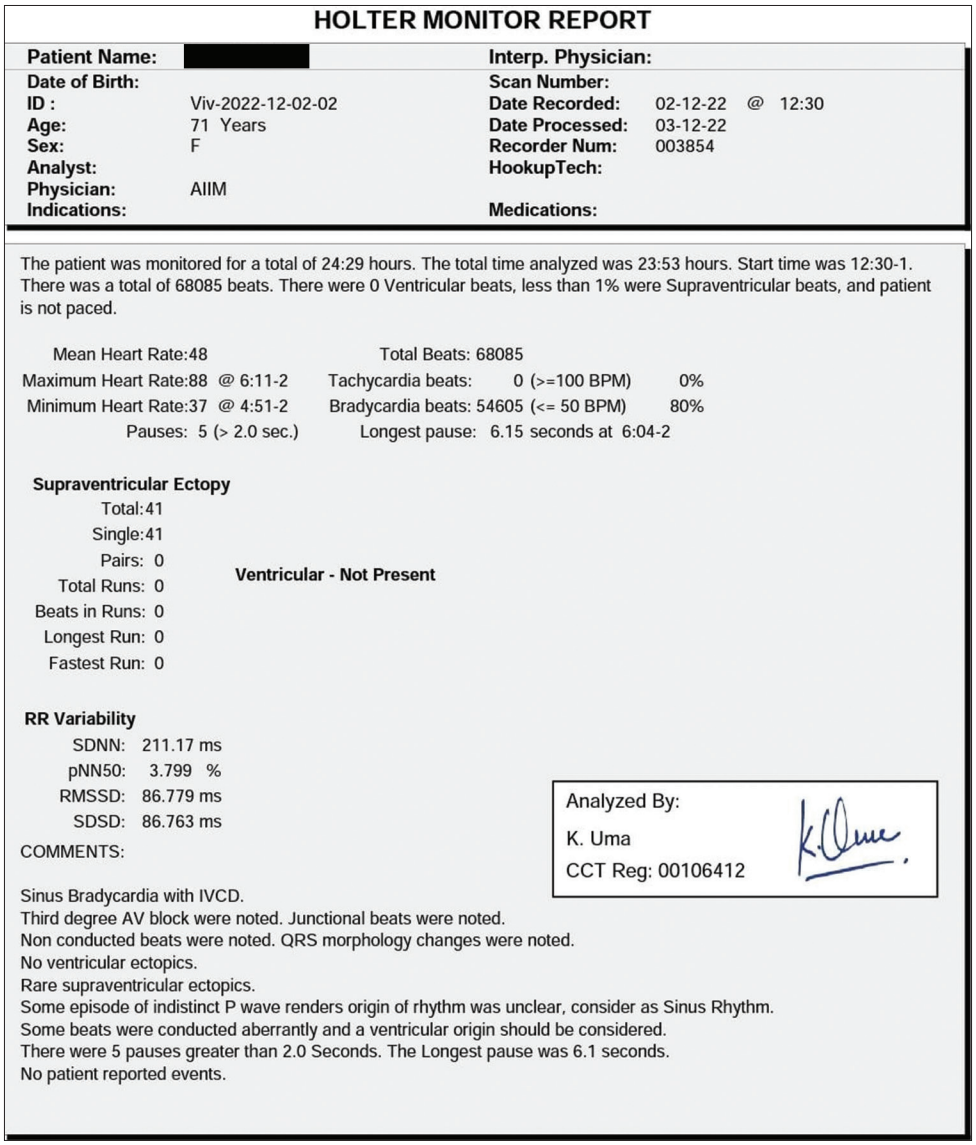


Fig. 1: ECG of the patient-Before pacemaker insertion

This patient had longstanding diabetes and CAN due to DM, manifested in the form of a 3rd-degree AV block. Degenerative AV nodal disease could also cause third-degree AV block, but it can be ruled out as autonomic neuropathy would be absent in such a condition. In addition, degenerative AV nodal disease typically manifests with syncope in



HOLTER MONITOR REPORT

Patient Name:	[redacted]	**Interp. Physician:**	
Date of Birth:		**Scan Number:**	
ID :	Viv-2022-12-02-02	**Date Recorded:**	02-12-22 @ 12:30
Age:	71 Years	**Date Processed:**	03-12-22
Sex:	F	**Recorder Num:**	003854
Analyst:		**HookupTech:**	
Physician:	AIIM		
Indications:		**Medications:**	

The patient was monitored for a total of 24:29 hours. The total time analyzed was 23:53 hours. Start time was 12:30-1. There was a total of 68085 beats. There were 0 Ventricular beats, less than 1% were Supraventricular beats, and patient is not paced.

Mean Heart Rate:	48	Total Beats:	68085
Maximum Heart Rate:	88 @ 6:11-2	Tachycardia beats:	0 (>=100 BPM) 0%
Minimum Heart Rate:	37 @ 4:51-2	Bradycardia beats:	54605 (<= 50 BPM) 80%
Pauses:	5 (> 2.0 sec.)	Longest pause:	6.15 seconds at 6:04-2

Supraventricular Ectopy

Total: 41
Single: 41
Pairs: 0
Total Runs: 0
Beats in Runs: 0
Longest Run: 0
Fastest Run: 0

Ventricular - Not Present

RR Variability

SDNN: 211.17 ms
pNN50: 3.799 %
RMSSD: 86.779 ms
SDSD: 86.763 ms

Analyzed By:
K. Uma
CCT Reg: 00106412

COMMENTS:

Sinus Bradycardia with IVCD.
Third degree AV block were noted. Junctional beats were noted.
Non conducted beats were noted. QRS morphology changes were noted.
No ventricular ectopics.
Rare supraventricular ectopics.
Some episode of indistinct P wave renders origin of rhythm was unclear, consider as Sinus Rhythm.
Some beats were conducted aberrantly and a ventricular origin should be considered.
There were 5 pauses greater than 2.0 Seconds. The Longest pause was 6.1 seconds.
No patient reported events.

Fig. 2: Holter monitor report

Fig. 3: Holter monitor tracings

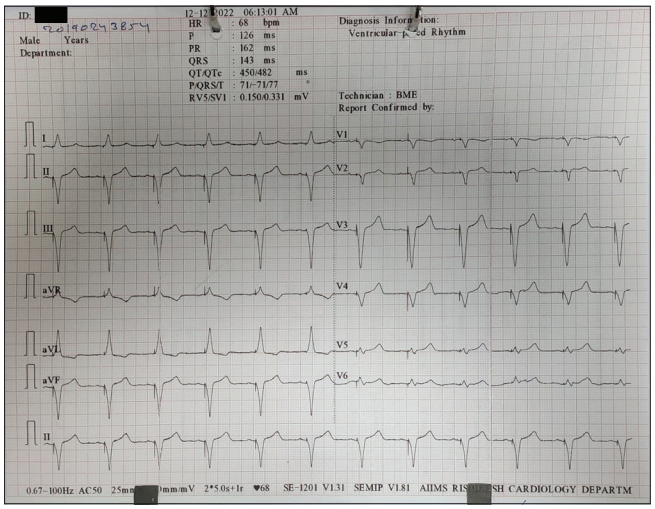


Fig. 4: ECG of the patient-After pacemaker insertion

Table 1: Timeline of events

Timeline	Events
20-year presentation was diagnosed with *type 2 diabetes mellitus*.	On tablet metformin 500 mg BD.
15 years before presentation was diagnosed with *breast cancer*.	Underwent post-modified radical mastectomy.
6 years before presentation was diagnosed with *Hypothyroidism*.	On tablet thyroxine 75 mcg OD.
4 years before presentation was diagnosed with *Systemic hypertension*.	On tablet telmisartan 40 mg OD.

patients above 80 years of age, making this a unique case for discussion. Considering that the patient is on tamoxifen, drug-induced CAN and associated heart disease can also be ruled out. The CHB observed in the 24-h Holter recording is known to cause neurocardiogenic syncope in this patient.

Table 2: Relevant hematological investigations

Investigations	Value
Hemoglobin	11.2 g/dL
Total leukocyte count	6200 cells/mm^3
Platelets	1,96,000 cells/mm^3
Urea/Creatinine	19.6/0.46 mg/dL
TSH	2.24 IU/L
HbA1C	8.2%
Total cholesterol/Triglycerides/ Low-density lipoproteins/ High-density Lipoproteins	168/154/132/32 mg/dL
Total bilirubin/Direct bilirubin	0.9/0.4 mg/dL
Serum sodium	135 meq/L
Serum potassium	4.4 meq/L

Table 3: Cardiac autonomic function testing

Test	Observed value	Inference
Deep breathing test-E/I ratio	6	Grade 2 dysfunction (Decreased parasympathetic reactivity)
Lying to standing test-30:15 ratio	0.98	Grade 2 dysfunction (Decreased parasympathetic reactivity)
Isometric hand grip test-change in diastolic BP	5 mmHg	Grade 2 dysfunction (Decreased sympathetic reactivity)

CONCLUSION

This 72-year-old woman's case illustrates the complexities and challenges of the workup for patients who present with recurrent syncope due to CHB, most likely as a result of CAN. Multiple factors, such as the duration of diabetes, poor glycemic control, metabolic derangements, and genetic factors, determine CAN. This case emphasizes that patients with type 2 diabetes without ischemic heart disease can develop CHB spontaneously. We suggest that patients with DM should be given more attention to the early detection of life-threatening conduction abnormalities that could potentially decrease mortality in this population.

AUTHOR CONTRIBUTION

Rohit Raina- formulation of the case report, Vikram Jain- Abstract formulation, Srikant S- Introduction, Discussion, Conclusion, Mayank Agarwal - Figures and table formulation, Ravi Kant- Overall Supervision.

CONFLICTS OF INTEREST

Nil.

AUTHOR SOURCE OF FUNDING

Nil.

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