Int. J. Curr. Res. Med. Sci. (2025). 11(8): 40-48

International Journal of Current Research in Medical Sciences

ISSN: 3107-3743 (Print), ISSN: 2454-5716 (Online)
(A Peer Reviewed, Indexed and Open Access Journal)
www.ijcrims.com

Case Report

Volume 11, Issue 8 -2025

DOI: http://dx.doi.org/10.22192/ijcrms.2025.11.08.005

Bisoprolol-induced complete heart block: A Case report

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Abstract

Background: Beta-blockers are prescribed in the management of cardiovascular diseases yet may rarely precipitate life-threatening conduction abnormalities like complete heart block (CHB).

Case Presentation: A 68-year-old male with a history of hypertension, diabetes mellitus, CAD post-PTCA, ischemic stroke, and dyslipidemia presented with syncope, bradycardia, and hypotension. ECG showed a third-degree AV block with AV dissociation. Laboratory investigation revealed hyperkalemia, acute kidney injury, diabetic ketoacidosis, sepsis secondary to urinary tract infection, and elevation in troponin I. Bisoprolol was the implicated cause. He was immediately managed with the cessation of bisoprolol, vasopressors and chronotropes, temporary cardiac pacing, insulin therapy, antibiotics, and correction of surrounding metabolic derangements. The patient's rhythm stabilized, renal function improved, and the pacemaker was removed safely prior to discharge.

Conclusion: This case underscores bisoprolol-induced CHB occurring in a high-risk patient and accentuates the utmost importance of early detection, prompt withdrawal of the offending drug, and multidisciplinary management for better outcomes.

Keywords: Bisoprolol, Complete heart block, Beta-blocker induced bradycardia, Adverse drug reaction, Cardiogenic shock, Clinical pharmacist intervention

Introduction

A complete heart block (CHB), otherwise known as third-degree atrioventricular (AV) block, is a dangerous cardiac conduction abnormality that occurs when an atrial impulse fails to reach the ventricles, causing AV dissociation. Syncope, dizziness, hypotension, fatigue, and sometimes sudden cardiac arrest when untreated are some of the presenting symptoms. CHB may occur due to ischemic heart disease, structural abnormalities, myocarditis, infections, electrolyte disturbances, or drug-induced causes. Being a potentially fatal and cardiogenic shock condition, it needs early recognition and treatment. Temporary pacing and support of the hemodynamics with drugs may be necessary in the acute setting, while permanent pacemaker implantation is the answer for chronic cases.[1,2]

Beta-blockers are commonly encountered in the cardiovascular domain for high blood pressure, ischemic heart disease, arrhythmias, and heart failure because they lessen the oxygen demand of the myocardium and improve survival and arrhythmia prevention. However, these drugs have dose-related adverse effects such as bradycardia, hypotension, and conduction disturbances. While Bisoprolol, being a cardioselective β1-adrenergic receptor blocker, is regarded as safer than non-selective agents, AV nodal suppression can still occur. Drug-induced CHB, though rare, is of clinical importance as it is commonly reversed once the causative drug is ceased along with supportive management. Elderly patients with multiple diseases, electrolyte imbalances, or existing conduction disease are at greater risk.[3,4]

This case involves bisoprolol-induced complete heart block in a 68-year-old man with multiple comorbidities, including coronary artery disease, diabetes mellitus, hypertension, dyslipidemia, and ischemic stroke. He further developed diabetic ketoacidosis with acute kidney injury and sepsis, all exacerbating the clinical scenario. The case warrants emphasis on the precipitated conduction disturbance drugs should be recognized in high-risk populations. Furthermore, care regarding beta-blockers, timely removal of the offending agent, and multidisciplinary management including clinical pharmacists will go a long way in preventing morbidity. With this case, pressure was exerted to alert clinicians on the risk of beta-blocker therapy in fragile patients, stressing that early diagnosis and prompt treatment can avert morbidities and mortalities.[5,6]

Case Presentation

A 68-year-old male presented with sudden loss of consciousness, dizziness, generalized weakness, and shortness of breath. Past medical history was significant for:

- Hypertension (15 years, on telmisartan 40 mg daily)
- Type 2 diabetes mellitus (12 years, on metformin and later glycomet GP1)
- Post-PTCA with CAD (on clopidogrel 75mg, atorvastatin 40 mg and bisoprolol 5 mg daily)
- Minor ischemic stroke (2 years prior, on aspirin initially, later switched to clopidogrel due to aspirin-induced gastritis).

On Admission Vitals

- **BP:** 100/60 mmHg
- **HR:** 35 bpm (bradycardia)
- **Temp:** Normal
- **RR:** 18/min
- **GRBS:** 401 mg/dL

Electrocardiogram (ECG)

- Complete Heart Block (Third-degree AV block)
- AV dissociation with independent P waves and QRS complexes.
- Subsequent tracings showed sinus rhythm with prolonged PR interval and transient T wave abnormalities.

Echocardiography (2D ECHO)

- **Left Ventricular Ejection Fraction (LVEF): 40%**
- **Regional Wall Motion Abnormalities (RWMA): Present**
- **Left Ventricular Dysfunction: Moderate systolic dysfunction**
- **Pulmonary Artery Hypertension (PAH): Mild**

Lab Investigation

Parameter	Patient Value	Normal Range	Interpretation
Hematology			
RBC (cells/mm³)	4.6	3.5-5.0	Nromal
Hemoglobin (g/dL)	13.3	12 – 17	Normal
WBC (cells/mm³)	24,100	4,000 – 11,000	Leukocytosis ↑
Neutrophils (%)	93	40 – 80	Neutrophilia ↑
Lymphocytes (%)	4	20 – 40	Low
Platelets (lakhs/cumm)	4.3	1.5 – 4.5	Normal
Renal Function Tests			
Urea (mg/dL)	98	15 – 40	Elevated ↑
Creatinine (mg/dL)	2.8	0.5 – 1.5	Elevated ↑
Uric Acid (mg/dL)	12.4	3.5 – 7.0	Elevated ↑
Electrolytes			
Sodium (mmol/L)	137	135 – 145	Normal
Potassium (mmol/L)	5.6	3.5 – 5.0	Hyperkalemia ↑
Chloride (mmol/L)	102	95 – 105	Normal
Liver Function Tests			
SGOT (U/L)	88	< 40	Elevated ↑
SGPT (U/L)	93	< 40	Elevated ↑
Alkaline Phosphatase (U/L)	144	40 – 125	Mildly Elevated ↑
Cardiac Markers			
Troponin I	31 (Positive)	Negative	Positive ↑
Pro-BNP (pg/mL)	1475	< 200	Elevated ↑
ABG			
pH	7.36	7.35 – 7.45	Normal (low-normal)
PCO_2 (mmHg)	22	35 – 45	Low ↓
PO_2 (mmHg)	81	80 – 100	Normal
Coagulation Profile			
PT (sec)	17	13 (control)	Prolonged ↑
INR	1.4	0.8 – 1.2	Mildly ↑
aPTT (sec)	38	34 (control)	Slightly ↑
Lipid Profile			
Total Cholesterol (mg/dL)	181	125 – 200	Normal
LDL (mg/dL)	112	< 100	Elevated ↑
HDL (mg/dL)	45	> 40	Normal
Triglycerides (mg/dL)	120	< 150	Normal
Urine Examination			
Albumin	Positive	Negative	Abnormal ↑
Sugar	+++	Negative	Abnormal ↑
Ketones	Positive	Negative	Abnormal ↑
Pus Cells (/HPF)	5–6	0–2	Elevated ↑

Differential Diagnosis

Based on syncope, bradycardia, hypotension, and altered sensorium in the patient, several differential diagnoses were considered. Acute coronary syndrome (myocardial infarction) was suspected with raised troponin I and ECG changes. Sepsis with a urinary tract infection was suspected in light of leukocytosis, history of fever, and urine findings. Diabetic ketoacidosis (DKA) could have been there owing to hyperglycemia, metabolic acidosis, and ketonuria. Electrolyte disturbances, especially hyperkalemia, might have contributed to conduction abnormalities. Finally, bisoprolol drug-induced complete heart block was strongly suspected, as the patient gave a history of going on beta-blockers for a long time, and symptoms abated on discontinuing the drug.

Final Diagnosis

Based on the clinical examination and investigations, the patient was diagnosed with complete heart block (third-degree AV block) likely induced by bisoprolol, presenting with profound bradycardia and hypotension. ECG revealed AV dissociation with independent P waves and QRS complexes, confirming complete heart block. Echocardiography showed left ventricular ejection fraction of 40%, regional wall motion abnormalities, and moderate LV systolic dysfunction, suggestive of underlying ischemic cardiomyopathy. Laboratory results demonstrated hyperkalemia, elevated creatinine and urea indicating acute kidney injury, metabolic acidosis suggestive of diabetic ketoacidosis, leukocytosis with neutrophilia supporting sepsis (UTI-related), and positive troponin I, consistent with myocardial injury. Collectively, these findings confirmed bisoprolol-induced CHB complicated by cardiogenic shock, DKA, AKI, and sepsis.

Treatment and hospital course

Day 1: On admission, the patient was hemodynamically unstable with bradycardia (HR 35 bpm), hypotension, and ECG evidence of complete heart block with AV dissociation. Immediate management included oxygen supplementation, intravenous fluids, and initiation of vasopressor and chronotropic support with continuous infusions of noradrenaline (20 ml/hr), dopamine (5 ml/hr), and isoprenaline (6 ml/hr). A temporary transcutaneous pacemaker was placed to stabilize the rhythm. Anticoagulation was initiated with intravenous heparin 5000 units QID, while atorvastatin 40 mg was continued for lipid control. Loop diuretic therapy with intravenous furosemide (20 mg OD) was also started. Symptomatic management included antiemetics, nebulization, and pain relief.

Day 2 : The following day, the patient showed improvement in rhythm with sinus rhythm and prolonged PR interval on ECG. Hemodynamics improved (BP 110/70 mmHg, HR 79 bpm), but blood glucose remained elevated at 470 mg/dL and creatinine increased to 2.9 mg/dL. Glycemic control was achieved with insulin therapy—subcutaneous Lantus 10 units at night and intravenous Actrapid on a sliding scale. Broad-spectrum antibiotic therapy was initiated with piperacillin-tazobactam 4.5 g IV BD after an initial stat dose. For renal protection, potassium binders and Nefrosave were introduced, while gastroprotection was provided with sucralfate suspension TID. Clopitab A (Clopidogrel + Aspirin) was restarted for antiplatelet coverage. Vasopressors were gradually tapered as blood pressure stabilized.

Day 3: On the third day, the patient complained of gastric discomfort and palpitations. Laboratory parameters showed improved renal function (creatinine 2.2 mg/dL) and a reduction in WBC count, though leukocytosis persisted. ECG demonstrated sinus rhythm with T-wave abnormalities, suggestive of possible inferior wall ischemic changes. Management included continuation of IV fluids, escalation of diuretic therapy with furosemide 20 mg BD, and addition of gastrointestinal protection with Ranitidine 150 mg OD. Ceftriaxone (Inj Meaxon) was added to broaden antibiotic coverage. Respiratory symptoms were addressed with Levolin nebulization..

Day 4: By the fourth day, the patient's vitals stabilized (BP 130/90 mmHg, HR 98 bpm), and creatinine improved to 1.6 mg/dL. ECG findings showed normalization of T waves with transient inferior wall MI changes. As the patient reported constipation and cough, supportive therapy was initiated with Cremaffin syrup at night and Pulmoclear syrup. Loop diuretic therapy was discontinued, and Lasilactone (spironolactone + furosemide) was introduced for preload reduction and better fluid balance. Nitrofurantoin 100 mg OD was started for confirmed urinary tract infection. Blood glucose levels improved, and Lantus insulin was discontinued.

Day 5: On the day of discharge, the patient's condition had significantly improved. Vitals were stable (BP 130/80 mmHg, HR 94 bpm, SpO_2 98% on room air), and renal function normalized with creatinine reduced to 1.2 mg/dL. ECG demonstrated normal sinus rhythm, and the temporary pacemaker was safely removed. Coronary angiography showed 30% stenosis of OM1 with patent stents. The patient was discharged on a stable medication regimen, including Clopitab A, Atorvastatin, Telmisartan, Lasilactone, Metformin, Pantoprazole, Sucralfate, and probiotics. Bisoprolol and all beta-blockers were permanently discontinued. He was counselled regarding strict medication adherence, lifestyle modifications, and follow-up with cardiology and nephrology.

Day	Clinical Status	Treatment Provided
Day 1 (Admission)	HR 35 bpm, BP 100/60 mmHg, lethargic, ECG: Complete heart block	- Oxygen therapy, IV fluids - Vasopressors: Noradrenaline infusion (20 ml/hr), Dopamine infusion (5 ml/hr) - Chronotrope: Isoprenaline infusion (6 ml/hr) - Anticoagulation: Inj Heparin 5000 U IV QID - Temporary transcutaneous pacing - Inj Lasix 20 mg OD - Atorvastatin 40 mg OD - Supportive: Ondansetron IV, nebulization (Asthalin)
Day 2	BP 110/70 mmHg, HR 79 bpm, GRBS 470 mg/dL, Creatinine 2.9 mg/dL, ECG: Sinus rhythm with prolonged PR interval	- Insulin therapy: Lantus 10 U SC HS + Human Actrapid sliding scale - Antibiotics: Piperacillin-tazobactam 4.5 g IV BD (after stat dose) - Renal protection: Potassium binders, Tab Nefrosave BD - Gastroprotection: Sucralfate suspension TID - Antiplatelet: Clopitab A (Clopidogrel + Aspirin) - Vasopressors tapered
Day 3	GRBS 210 mg/dL, Creatinine 2.2 mg/dL, ECG: Sinus rhythm with T-wave changes	- IV fluids (Normal saline) - Inj Lasix 20 mg BD - Antibiotic escalation: Inj Ceftriaxone (Meaxon) - GI protection: Tab Rantac 150 mg OD - Nebulization: Levolin
Day 4	BP 130/90 mmHg, HR 98 bpm, Creatinine 1.6 mg/dL, ECG: Sinus rhythm, transient inferior MI changes	- Diuretic modification: Stopped Lasix, started Tab Lasilactone BD - Antibiotic: Tab Nitrofurantoin 100 mg OD (for UTI) - Symptomatic: Syp Cremaffin 10 ml HS (constipation), Syp Pulmoclear (cough) - Stopped Lantus

Day 5 (Discharge)	BP 130/80 mmHg, HR 94 bpm, SpO_2 98% RA, ECG: Sinus rhythm, Creatinine 1.2 mg/dL, CAG: OM1 stenosis 30%	- Temporary pacemaker removed - Discharge medications: Clopitab A, Atorvastatin 40 mg, Telmisartan 40 mg, Lasilactone, Metformin, Pantoprazole, Sucralfate, Probiotics - Calcium + Vitamin D supplements - Beta-blockers permanently discontinued

Discharge Plan

The patient was discharged in stable condition after his cardiac rhythm had been stabilized, the acute kidney injury had resolved, and the sepsis and diabetic ketoacidosis were controlled. Removal of the temporary pacemaker was done while sinus rhythm was restored and maintained. The patient was counselled to avoid beta-blockers for good, because bisoprolol caused him to have complete heart block. Lifestyle modifications suggested were low salt and fat diet, strict glycemic control, adequate hydration, and some regular exercises. The patient was informed about the signs and symptoms of bradycardia, including dizziness, syncope, or fatigue, and advised to seek immediate evaluation should the symptoms recur. Regular follow-up with cardiology and nephrology was advised for device check-ups, renal function monitoring, and prevention of secondary ischemic events.

Generic Name	Dose & Frequency	Indication/Remarks
Clopidogrel + Aspirin (Clopitab A)	PO OD	Secondary prevention (CAD, stroke)
Atorvastatin	40 mg PO OD	Dyslipidemia, CAD protection
Dapagliflozin (Udapa)	10 mg PO OD	Diabetes, cardiorenal protection
Furosemide + Spironolactone (Lasilactone)	20 mg PO OD	Preload reduction, LV dysfunction
Pantoprazole	40 mg PO OD	Acid peptic disorder (APD), gastroprotection
Probiotic (Biospan)	PO OD	GI health
Calcium + Vitamin D supplement	PO OD	Bone health
Sucralfate + Oxetacaine (Sucral O)	10 ml PO TID	Anti-ulcer, APD
Telmisartan	40 mg PO OD	Hypertension
Metformin	500 mg PO OD (night)	Type 2 Diabetes Mellitus

Outcome

The patient responded well to the discontinuation of bisoprolol, initiation of inotropes and chronotropes, and temporary pacing. Within 2 days, the cardiac rhythm was stabilized from complete heart block to sinus rhythm with a prolonged PR interval, and by day 5, the patient maintained sinus rhythm without pacing. Hyperkalemia, diabetic ketoacidosis, and acute kidney injury were treated with insulin therapy, correction of electrolytes, and renal protective measures. Infection complications were treated with a broad-spectrum antibiotic which were then de-escalated to oral nitrofurantoin, culminating in normal white blood cell counts and resolution of the urinary tract infection. Renal function recovered slowly, with serum creatinine improving from 2.8 mg/dL at admission to 1.2 mg/dL at discharge. At the time of discharge, the patient was hemodynamically stable, and conduction abnormalities had resolved, and he was continued on optimized therapy without beta-blockers. He was advised about stringent adherence to medications, lifestyle modifications, and follow-up with cardiology and nephrology.

Pharmacist Interventions

- The pharmacist pinpointed bisoprolol as the cause of complete heart block and advised its immediate withdrawal.
- Inotropes and chronotropes (noradrenaline, dopamine, and isoprenaline) were recommended for maintaining the heart rate and blood pressure.
- A temporary pacemaker was suggested until stabilization of the rhythm.
- Helped manage diabetic ketoacidosis by starting insulin therapy using Actrapid sliding scale and Lantus and keeping an eye on glucose levels.
- Advised correcting hyperkalemia using potassium binders and an insulin-glucose infusion.
- Prevented further kidney injury by adjusting drug dosages based on renal function.
- Recommended antibiotics appropriate for urinary tract infection-initially piperacillin-tazobactam and later nitrofurantoin.
- Proctected from gastrointestinal complications using pantoprazole, sucralfate and probiotics during treatment.
- Educated the patient and family on permanent avoidance of beta-blockers, recognizing signs of bradycardia, and the need for routine follow-ups.

Discussion

A rare but clinically relevant diagnosis of bisoprolol-induced complete heart block (CHB) in a 68-year-old male with several comorbidities like diabetes, hypertension, coronary artery disease, and ischemic stroke is discussed in this study. The patient had been taking bisoprolol for a long time after PTCA, thus possibly placing him at increased risk for conduction abnormalities. The patient presented with profound bradycardia (HR 35 bpm), hypotension, and AV dissociation on ECG, consistent with the diagnosis of third-degree AV block. Importantly, withdrawal of bisoprolol, supportive therapy, and temporary pacing resulted in recovery of the sinus rhythm, the temporal association strongly implicating bisoprolol as the causative agent. This case is a testimony to the need for close attention while prescribing any beta-blocker to an elderly patient with structural heart disease and impaired renal function, as such patients are predisposed to drug-induced conduction disturbances.[7,8]

In addition to lowering heart rate, myocardial contractility, and conduction through the AV node, bisoprolol is cardioselective for the β1-adrenergic receptor. Although this mechanism is beneficial for ischemic heart disease and atrial and ventricular arrhythmias, it can also affect AV nodal suppression. The elderly and middle-aged are most at risk when preexisting conduction disease, LV dysfunction, electrolyte abnormalities, or concomitant AV-nodal blocking drugs exist. There were many aggravating factors present in this patient-moderate LV systolic dysfunction (EF 40%), renal impairment with hyperkalemia, metabolic acidosis secondary to DKA, and systemic infection. These factors may understandably have interacted with bisoprolol to produce CHB. After the withdrawal of bisoprolol and supportive treatment, the resolution of the heart block entailed that the relation was truly causal. There are thus situations under which clinicians need to weigh the risks and benefits of continuing use of a beta blocker in a complicated patient and observe closely for conduction abnormalities.[9,10]

The remarkable characteristic of this case was the coexistence in a plethora of systemic complications at presentation. Severe hyperglycemia, ketonuria, and acidosis confirmed DKA; leukocytosis, pyuria, and elevated inflammatory markers, however, pointed towards sepsis secondary to a urinary tract infection. Then came the impaired renal function, with increased creatinine and urea levels, which was consistent with AKI. All these could have been potential perpetrators in worsening cardiac conduction and myocardial function. Hyperkalemia and metabolic acidosis are known to depress cardiac tissue excitability, thereby precipitating arrhythmias and AV block. Elevated troponin I provided further evidence for myocardial injury, whether ischemic or sepsis-related, which may have further exacerbated the conduction disturbances. This

multifactorial interaction presents a scenario where a drug-induced adverse reaction can be aggravated by systemic illness; this hence portrays why a full assessment of the acutely ill patient is imperative.[11,12]

The management was comprehensive and addressed control of immediate arrhythmic emergencies alongside systemic complications. Hemodynamic stabilization was maintained with vasopressors and chronotropic agents (noradrenaline, dopamine, isoprenaline), while temporary pacing gave life-saving rhythm support. Metabolic derangements were promptly corrected with insulin for DKA and potassium binders with insulin–glucose infusion for hyperkalemia. The patient received broad-spectrum antibiotics for sepsis and was later switched to oral nitrofurantoin for UTI. The renal parameters were closely monitored with protective measures put in place, with renal dose adjustments till normalization. Gastrointestinal protection was added with pantoprazole and sucralfate to avoid ulcer recurrence. Gradual improvement in clinical and laboratory parameters allowed the removal of temporary pacemaker and discharge on optimized medications except for beta-blockers. This case exhibited how drug-induced life-threatening arrhythmias could be reversed with coordinated, multidisciplinary intervention to restore stability.[13,14]

There are several key clinical lessons warranted from this case. Firstly, within the elderly, beta blocker therapy though safe, can induce severe conduction disturbances when structural and metabolic factors predispose to it. Secondly, in cases of unexplained bradycardia or syncope, drug-induced CHB should always be suspected, particularly if the patient is taking any sort of AV nodal blocking agent. Third, systemic ailments such as infections, electrolyte imbalances, and renal dysfunction can magnify drug effects to worsen outcomes. The clinical pharmacists were, however, very critical in identifying this ADR and recommending immediate discontinuation of bisoprolol, helping to choose antibiotics, and guiding renal dose adjustments-all while counseling the patient on adherence and medications to avoid such as beta-blockers. This collaborative care model further emphasizes the importance of integrating pharmacists into the healthcare team to promote patient safety. Finally, early identification with rapid withdrawal of the offending agent and aggressive supportive management go a long way in getting a good outcome in bisoprolol-induced CHB.[15]

Conclusion

This case report represents a very rare yet very serious manifestation of bisoprolol-induced third-degree heart block in an elderly patient with multiple comorbidities. Beta-blockers are indispensable drugs in treating coronary artery disease, hypertension, and heart failure; however, they may cause conduction abnormalities, especially in certain patients with pre-existing structural heart defects, electrolyte abnormalities, or renal dysfunction. For this particular patient, left ventricular dysfunction, hyperkalemia, diabetic ketoacidosis, and sepsis were some factors that would have made him very susceptible to any adverse drug effect. Early recognition of bisoprolol as the offending agent and immediate discontinuation of the drug; supportive management with inotropic support, insulin therapy, antibiotics, correction of metabolic derangements, and temporary pacing led to full stabilization and recovery. This case emphasizes the particular necessity to exercise caution when prescribing AV nodal blocking agents in high-risk patients. It also highlights the critical role of clinical pharmacists in recognizing adverse drug reactions, directing therapy, and ensuring patient safety through education and follow-up.

References

1. Dhobe S, Singh S, Sharma R, Ankar R. A Case Report of Complete Heart Block. J. Pharm. Res. 2021;33(46B):546-50.
2. Friedman D, Duncanson Lj, Glickstein J, Buyon J. A review of congenital heart block. Images Paediatr Cardiol. 2003 Jul;5(3):36-48
3. Johri N, Matreja PS, Maurya A, Varshney S, Smritigandha. Role of β-blockers in Preventing Heart Failure and Major Adverse

Cardiac Events Post Myocardial Infarction. Curr Cardiol Rev. 2023;19(4):e110123212591
4. Bustea C, Radu A-F, Vesa CM, Radu A, Bodog TM, Bodog RF, Maghiar PB, Maghiar AM. Drug-Induced Complete Atrioventricular Block in an Elderly Patient: A Case Report Highlighting Digoxin-Beta Blocker Interactions and a Paradoxical State. Life. 2025; 15(2):215
5. Bustea C, Radu AF, Vesa CM, Radu A, Bodog TM, Bodog RF, Maghiar PB, Maghiar AM. Drug-Induced Complete Atrioventricular Block in an Elderly Patient: A Case Report Highlighting Digoxin-Beta Blocker Interactions and a Paradoxical State. Life (Basel). 2025 Jan 31;15(2):215
6. Zeltser D, Justo D, Halkin A, Rosso R, Ish-Shalom M, Hochenberg M, Viskin S. Drug-induced atrioventricular block: prognosis after discontinuation of the culprit drug. J Am Coll Cardiol. 2004 Jul 7;44(1):105-8.
7. Nakayama M, Fuse K, Sato M, Okabe M, Komatsu Y, Yamashina A, Aizawa Y. Cardiogenic shock induced by bisoprolol in a patient with CYP2D6 variant. A case report. J Cardiol Cases. 2015 Jan 19;11(4):109-112.
8. Baxter AJ, Spensley A, Hildreth A, Karimova G, O'Connell JE, Gray CS. Beta blockers in older persons with heart failure: tolerability and impact on quality of life. Heart. 2002 Dec;88(6):611-4.
9. Wołowiec Ł, Grześk G, Osiak J, Wijata A, Mędlewska M, Gaborek P, Banach J, Wołowiec A, Głowacka M. Beta-blockers in cardiac arrhythmias-Clinical pharmacologist's point of view. Front Pharmacol. 2023 Jan 9;13:1043714
10. Parsova KE, Hayiroglu MI, Pay L, Cinier G, Gurkan K. Long-term follow-up of patients with drug-related atrioventricular block without a need of permanent pacemaker during index hospitalization. Egypt Heart J. 2022 Aug 1;74(1):56.
11. Marcu DTM, Adam CA, Dorobanțu DM, Șalaru DL, Sascău RA, Balasanian MO, Macovei L, Arsenescu-Georgescu C, Stătescu C. Beta-Blocker-Related Atrioventricular Conduction Disorders-A Single Tertiary Referral Center Experience. Medicina (Kaunas). 2022 Feb 20;58(2):320.
12. Zochios V, Valchanov K. Raised cardiac troponin in intensive care patients with sepsis, in the absence of angiographically documented coronary artery disease: A systematic review. J Intensive Care Soc. 2015 Feb;16(1):52-57.
13. Kingma J, Simard C, Drolet B. Overview of Cardiac Arrhythmias and Treatment Strategies. Pharmaceuticals (Basel). 2023 Jun 6;16(6):844.
14. Kaneko M, Kasao M, Obikane H, Ueda K. Critical heart failure associated with beta-blocker-induced cardiac phospholipidosis: a case report. Eur Heart J Case Rep. 2024 Nov 14;8(12):ytae608.
15. Bangalore S, Sawhney S, Messerli FH. Relation of beta-blocker-induced heart rate lowering and cardioprotection in hypertension. J Am Coll Cardiol. 2008 Oct 28;52(18):1482-9.

How to cite this article:
Asma Anjum, Ayesha Batool, Fouzia Khan, Mujeeb Ur Rahman. (2025). Bisoprolol-induced complete heart block: A Case report. Int. J. Curr. Res. Med. Sci. 11(8): 40-48.
DOI: http://dx.doi.org/10.22192/ijcrms.2025.11.08.005

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Website: www.ijcrims.com

Subject: Cardiology

Quick Response Code

DOI: 10.22192/ijcrms.2025.11.08.005