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CONSERVATIVE MANAGEMENT OF REVERSIBLE TOTAL ATRIOVENTRICULAR BLOCK RESULTING IN SINUS RHYTHM RECOVERY: A CASE REPORT

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Abstract: **Conservative Management of Reversible Total Atrioventricular Block Resulting in Sinus Rhythm Recovery: A Case Report.** *A 49-year-old woman presented with weakness, nausea, and vomiting and was found to have complete atrioventricular (AV) block on electrocardiography. Initial evaluation suggested hypovolemia and electrolyte disturbance due to poor oral intake, with mild hypokalemia identified as a potential reversible contributor to the conduction abnormality. Although temporary pacing was considered because of the severity of the AV block, the patient was managed conservatively with fluid resuscitation and potassium replacement after referral was declined by the family. Progressive clinical and electrocardiographic improvement was observed, with restoration of normal sinus rhythm by the sixth hospital day and complete resolution of symptoms. This case highlights the importance of identifying reversible causes before permanent pacemaker implantation.*

Keywords : *Total AV Block, Hypokalemia, Conservative Treatment, Reversible AV Block*

Abstrak: Manajemen Konservatif pada *Total Atrioventricula Block* Reversibel dengan Pemulihan Irama Sinus: Sebuah Laporan Kasus. Seorang perempuan usia 49 tahun datang dengan keluhan lemas, mual, dan muntah, serta ditemukan mengalami blok atrioventrikular (AV) total pada pemeriksaan elektrokardiografi. Evaluasi awal mengarah pada hipovolemia dan gangguan elektrolit akibat asupan oral yang buruk, dengan hipokalemia ringan yang diduga berkontribusi sebagai faktor reversibel terhadap gangguan konduksi tersebut. Meskipun pemasangan pacu jantung sementara sempat dipertimbangkan karena beratnya blok AV yang terjadi, pasien mendapatkan tata laksana konservatif berupa resusitasi cairan dan koreksi kalium setelah keluarga menolak rujukan. Perbaikan klinis dan elektrokardiografis berlangsung secara bertahap, dengan kembalinya irama sinus normal pada hari keenam perawatan serta resolusi penuh gejala. Kasus ini menegaskan bahwa hipokalemia, meskipun merupakan penyebab yang jarang dari blok AV total, harus dikenali dan dikoreksi secara dini karena penatalaksanaan yang tepat dapat membalikkan gangguan konduksi jantung serta menghindarkan pasien dari tindakan invasif yang tidak diperlukan.

Kata Kunci : Blok AV Total, Hipokalemia, Tatalaksana Konservatif, Etiologi Reversible Blok AV

INTRODUCTION

Atrioventricular block is a disruption of the normal function of the heart's conduction pathway from the sinoatrial (SA) node to the ventricle through the atrioventricular (AV) node. Third-degree AV block indicates a complete cessation of communication between the atrium and ventricle. In the absence of proper conduction through the AV node, the SA node cannot regulate the heart rhythm, resulting in

decreased cardiac output due to the lack of coordination between the atria and ventricles (Knabben et al., 2023).

Various pathological conditions can affect the occurrence of atrioventricular block, whether congenital or acquired. Acquired pathological conditions are more common and include infectious, inflammatory, degenerative, ischemic, and iatrogenic etiologies. Degenerative etiology is the most commonly observed in clinical practice and correlates with advanced age, chronic hypertension, and diabetes mellitus. Infectious etiology, including Lyme carditis, should be considered In appropriate patients, atrioventricular block may be reversible with adequate medical intervention. Ischemic causes should also be investigated, as atrioventricular block resulting from inferior wall ischemia or myocardial infarction can be reversible. Atrioventricular block induced by vagotonic effects is usually temporary and typically does not require pacing. Iatrogenic causes are often clearly evident from the clinical context (Kusumoto et al., 2019).

Based on the cause, AV block can be managed with either invasive or conservative methods. This case report will discuss one type of third-degree AV block that can be managed conservatively, taking into account the possible etiology and risk factors present in the patient. This case is reported due to the rarity of reversible TAVB associated with mild hypokalemia and successful conservative management without cardiac pacing.

CASE REPORT

The patient is a 49-year-old female who came to the emergency department along with her family with a chief complaint of nausea and vomiting for 1 week before hospital admission. The patient also complained of anorexia along with dizziness. Shortness of breath, chest pain, and heart palpitations were denied. The patient also denied having a past medical history of heart disease, hypertension, and diabetes mellitus. The patient only reported suffering from gastritis in the past.

From physical examination, the patient was generally weak and presented with bradycardia (37x/m), tachypnea (24x/m), hypotension (78/58 mmHg), and cold extremities. The ECG test (Figure 1) showed extreme bradycardia with a Grade III total AV block. Laboratory tests (Table 1) showed leukocytosis (14,820), hyperuricemia (6.4), and hypokalemia (3.4). Chest x-ray was within normal limits. The patient was diagnosed with cardiogenic shock due to grade III total AV block, along with low intake + vomiting and hypokalemia. The patient was initially treated with nasal cannula oxygen at 3 lpm, a 500 cc loading dose of crystalloid fluids (continued with a maintenance dose), dopamine at 10 mcg/kg/minute, norepinephrine at 100 ng/kg/minute, 2 ampules of sulfas atropine injection, and 25 meq KCL drip on 500 ml Ringer's lactate. Urine output and signs of lung edema were monitored to observe the potential complications of fluid administration.

The patient was monitored every 12 hours for medication effects and complications. The patient suffered a convulsion while still being awake after 12 hours of initial treatment, possibly due to brain hypoxia caused by the extreme bradycardia. The patient was then treated with 800 mg of phenytoin for the convulsion, and the dose of dopamine was increased to 15 mcg/kg/minute, and the dose of norepinephrine was increased to 100 ng/kg/minute. The phenytoin was stopped on the next day because the patient did not show any additional convulsions, while the other medications were continued. On the second day, the patient was planned to be referred to a higher level of cardiologic care for a temporary pacemaker placement, but the patient’s family refused due to limited resources in taking care of the patient on a hospital outside of town, and the patient was continued with conservative management. On the third day, the patient’s heart rate has risen to

a normal rate (72x/m) and the blood pressure has also normalized (120/60 mmHg). Dopamine and norepinephrine were then tapered down alternately because the patient's hemodynamics had stabilized. On the fifth day, the patient's ECG showed a normal sinus rhythm of 90 beats per minute (Figure 2), and the complaints of weakness were resolved. The patient was then discharged and scheduled for a routine check-up on the cardiology clinic. Upon 1 week follow up at the clinic, the patient was stable and did not have any recurring problem on the cardiovascular system. The patient did not report any complaints upon 3 months of follow up. This lab results were based on the blood sample that was taken from the patient upon the first day of admission in the ER.

Table 1. Laboratorium Results

Lab Test	Results	Normal Levels
Hb	12,7	**Man : 13,5 - 18g/dL Woman : 13 - 18g/dL**
Leucocyte	**14.820**	**4.500-11.000**
Eritrocyte	5,1	**Man : 4,6 – 6,2 10^6 Woman : 4,2 - 5,4 10^6**
Thrombocyte	181.000	**150.000 – 450.000**
Hematocrit	38,3	**35 – 47 %**
MCV	75,2 fl	**82 – 92 fl**
MCH	**24,9 pg**	**27,0 – 31,0 pg**
MCHC	33,1 g/L	**32,0 – 37,0 g/L**
SGOT	56	**<31 IU/L**
SGPT	66	**<35 IU/L**
Ureum	**65**	**10-59**
Serum Creatinin	**1,4**	**0,7-1,2 mg/dL**
Random Blood Glucose	198	**<125 mg/dL**
Natrium	146	**135-148 mmol/l**
Kalium	**3,4**	**3,5-5,3 mmol/l**
Chlorida	100	**98-107 mmol/l**
Calcium	**2,5**	**2,0-2,5 mmol/l**

Table 2. Timeline of The Patient's Condition

Time	Clinical Findings / Events	Investigations	Interventions	Outcome
1 week before admission	Nausea, vomiting, anorexia, dizziness. No chest pain, dyspnea, or palpitations.	–	Symptomatic treatment at home (not specified).	Symptoms progressively worsened.
Day 0 (Emergency Department admission)	Weak appearance, cardiogenic shock with bradycardia (37	ECG: Grade III complete AV block. Laboratory: leukocytosis	Oxygen via nasal cannula (3 L/min), crystalloid loading (500 mL) followed by	Hemodynamic support initiated.

	bpm), hypotension (78/58 mmHg), tachypnea (24 breaths/min), cold extremities.	(14,820/µL), hypokalemia (3.4 mmol/L), hyperuricemia (6.4 mg/dL). Chest X-ray: normal.	maintenance fluids, dopamine (10 µg/kg/min), norepinephrine (100 ng/kg/min), atropine sulfate (2 ampules), potassium chloride (25 mEq in 500 mL Ringer's lactate). Urine output and pulmonary edema monitored.	
12 hours after admission	Conscious generalized convulsion, suspected secondary to cerebral hypoperfusion from profound bradycardia.	Clinical assessment.	Phenytoin 800 mg loading dose. Dopamine increased to 15 µg/kg/min. Norepinephrine continued.	Convulsion resolved.
Day 1	No recurrent seizure. Persistent complete AV block.	Clinical monitoring every 12 hours.	Phenytoin discontinued. Other supportive therapy continued.	Neurological status remained stable.
Day 2	Persistent complete AV block.	Clinical reassessment.	Referral for temporary pacemaker placement was recommended; family declined referral because of financial and logistical limitations. Conservative management continued.	Continued medical management.
Day 3	Hemodynamic improvement: heart rate 72 bpm, blood pressure 120/60 mmHg.	Clinical monitoring.	Dopamine and norepinephrine gradually tapered.	Stable hemodynamics achieved.
Day 5 (Discharge)	Resolution of weakness and stable vital signs.	ECG: Normal sinus rhythm, 90 bpm.	Discharged with scheduled cardiology outpatient follow-up.	Clinical recovery without pacemaker implantation.
1-week follow-up	No recurrent cardiovascular symptoms.	Clinical evaluation.	Routine outpatient follow-up.	Patient remained stable.
3-month follow-up	Asymptomatic.	Clinical evaluation.	Continued observation.	No recurrence of atrioventricular block or cardiovascular complaints.

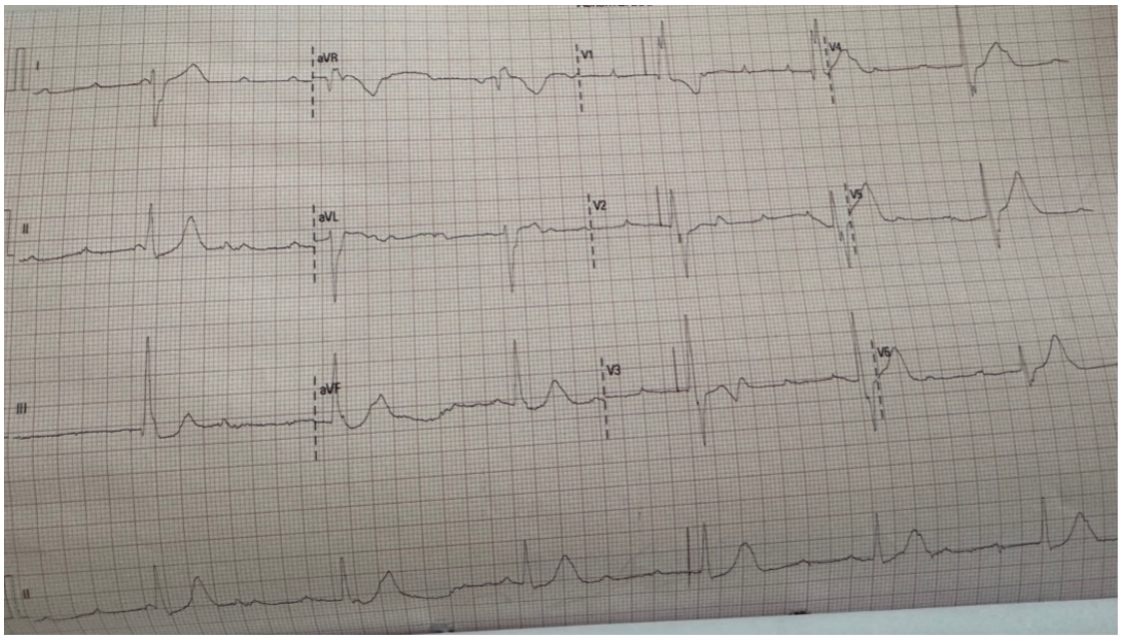

Figure 1. Grade III Total AV Block with Extreme Bradycardia (37 bpm)
This ECG was taken in the emergency department when the patient first arrived at the hospital

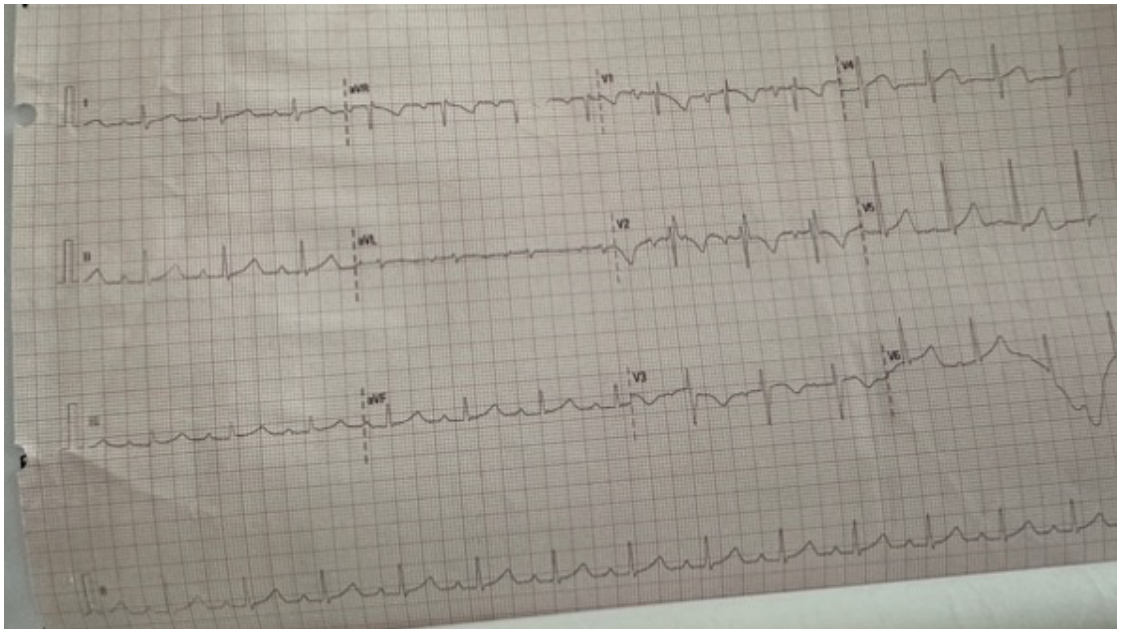

Figure 2. Normal Sinus Rhythm (90 bpm)
This ECG was taken in the general ward after the patient received conservative treatment for five days

DISCUSSION

This patient came with symptoms that seem unrelated to total AV block directly but could have caused the condition indirectly. Atrioventricular blocks may result from several potentially reversible situations, including ischemic heart disease, electrolyte imbalances, pharmacological agents, and viral infections. Such factors must always be excluded to prevent needless pacemaker placement. The care of patients and rates of reversibility are contingent upon the underlying etiology. However, the return of an atrioventricular block after the resolution of the primary reason may require pacemaker installation since reversible situations might reveal a preexisting conduction abnormality (Pavone & Pelargonio, 2021).

As for other etiological possibilities, acute myocardial ischemia remains an important cause of complete AV block, particularly involving the inferior wall and AV nodal artery. In this case, although cardiac biomarkers were not available, the absence of chest pain, ischemic ECG changes, and rapid clinical recovery made ischemia less likely. Myocarditis can also produce transient AV block through inflammatory injury to the conduction system. However, no clinical features suggestive of active myocardial inflammation were identified. Lyme carditis is a recognized reversible cause of complete AV block, although it is uncommon in Indonesia and no epidemiological exposure history was identified. Degenerative conduction disease remains a common cause of AV block in adults. Nevertheless, the rapid restoration of AV conduction after correction of metabolic abnormalities suggests a predominantly reversible mechanism.

In this case, the most likely cause of reversible TAVB is electrolyte imbalance. Hypovolemia, a result of low intake due to the patient's nausea and vomiting, caused the electrolyte imbalance in this case. Dehydration may result in increased electrolyte concentrations, particularly sodium (hypernatremia), while overhydration may dilute electrolytes, leading to diseases such as hyponatremia (Larson et al., 2024). Hypernatremia itself could cause an elevation of intracellular muscle sodium, displacing potassium and resulting in total body potassium deficiency. The primary depletion of serum potassium may result in the buildup of intracellular sodium levels (Torchinsky et al., 2004). This is probably what happened to the patient, as her blood potassium levels were low.

Hypokalemia itself could induce TAVB due to potassium's crucial role in electric conduction regulation of the cardiac muscle. Hypokalemia may result in modifications to cardiac membrane potential and delays in repolarization, increasing the risk of cardiac arrhythmias, including ventricular ectopy, atrial fibrillation, and potentially fatal ventricular tachycardia or fibrillation (Castro & Sharma, 2025). Severe hypokalemia may also induce atrioventricular block, but this occurrence is very rare (Levis, 2012). This case is quite unique in itself, as the hypokalemia that the patient suffered was not severe, and it can still induce TAVB.

Although severe hypokalemia is more commonly associated with high-grade atrioventricular block, the present case demonstrates that even mild hypokalemia may contribute to conduction disturbances when combined with additional physiological stressors such as hypovolemia, prolonged vomiting, sympathetic activation, and possible latent conduction system vulnerability. Therefore, hypokalemia should not necessarily be viewed as the sole etiology of AV block in this patient, but rather as a potentially reversible contributing factor.

The patient was initially planned for a temporary pacemaker to resolve the extreme bradycardia that was caused by the TAVB, as it was still unclear at the start whether the low potassium levels were the cause of the patient's TAVB.

Temporary pacemakers (TP) are used in the urgent management of individuals with severe bradyarrhythmia. They are often used in emergencies and for elderly people in compromised health who exhibit hemodynamic instability (Ayerbe et al., 2004). According to the 2018 ACC/AHA/HRS Guideline on Bradycardia and Cardiac Conduction Delay, temporary pacing is indicated in symptomatic patients with high-grade AV block associated with hemodynamic compromise while the underlying cause is being evaluated and treated. In patients with potentially reversible etiologies, correction of the reversible factor should be prioritized before considering permanent pacing. In the present case, temporary pacing was initially considered because of cardiogenic shock and severe bradycardia (Kusumoto et al., 2019).

However, due to the patient's family not accepting the referral suggestion from our team, the patient was instead treated with conservative treatment with the focus of executing a fluid challenge to resolve the shock along with inotropes, chronotropes, and vasopressors to support cardiac function, with resolving the hypokalemia using KCL infusion. Stabilizing the hemodynamics is essential in cardiogenic shock patients, as rhythm and rate control can only be feasibly treated if the hemodynamics of the patient are stable (Laghlam et al., 2024).

The restoration of sinus rhythm following fluid resuscitation and potassium replacement supports the presence of a functional rather than structural conduction abnormality. Potassium plays a critical role in maintaining transmembrane electrical gradients and impulse propagation through the atrioventricular node. Correction of electrolyte imbalance may normalize conduction velocity and excitability, allowing recovery of AV nodal conduction when irreversible structural damage is absent.

This report has several limitations. Serial electrolyte measurements, cardiac biomarkers, echocardiography, and advanced investigations for myocarditis or infiltrative disease were not available. Therefore, a definitive causal relationship between mild hypokalemia and complete AV block cannot be established. Nevertheless, the temporal association between correction of metabolic abnormalities and restoration of normal AV conduction suggests a potentially reversible functional mechanism.

CONCLUSION

This case describes reversible TAVB occurring in association with electrolyte imbalance and hypovolemia, with recovery following conservative management and correction of metabolic abnormalities. The unusual feature of this case is the occurrence of reversible complete AV block in the setting of only mild hypokalemia. Reversible causes of total AV block should be thoroughly investigated before permanent pacemaker implantation. Conservative management with correction of electrolyte imbalance may restore normal cardiac conduction in selected patients. The correction of metabolic abnormalities associated with hypokalemia was followed by recovery of AV conduction. This case highlights the importance of systematically evaluating reversible causes of complete AV block and supports further investigation into the role of electrolyte disturbances in reversible conduction abnormalities.

Ethical Consideration

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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