Vol 4 No 2 October 2024
Solo Journal of Anesthesi, Pain and Critical Care

E-ISSN : 2797-0035
P- ISSN : 2776-1770

CASE REPORT

Resolution of Complete AV block to Normal Sinus Rhythm without Cardiac Pacemaker in a Patient with Severe Acidosis: A Case Report

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Article Info :
Submitted :
18-06-2022
Accepted :
11-10-2024
Published :
31-10-2024
https://doi.org/10.20961/sojaV4i2.62234

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ABSTRACT

Background : Complete atrioventricular block (CAVB) is a life-threatening medical emergency and requires an immediate cardiac pacemaker to reduce mortality by up to 87%.

Case Illustration : A 55-year-old woman with an unknown medical history was brought to the emergency room with a Glasgow Coma Scale (GCS) of E2V1M1 (sopor comatose) and Kussmaul breathing. The patient, who was in septic shock, had Multiple Organ Dysfunction Syndrome (MODS) and severe acidosis. The patient was hemodynamically unstable, requiring vasopressor to maintain a Mean Arterial Pressure (MAP) ≥ 65 mm Hg. The analysis of the arterial blood gas revealed a pH of 7.19, pCO_2 of 16 mm Hg, pO_2 of 204 mm Hg, and bicarbonate of 5.9 mmol/L. Hyperkalemia (5.33mmol/L) was discovered during the electrolyte analysis.

Conclusion : An electrocardiogram (ECG) examination showed CAVB with non-malignant ventricular extrasystoles. Without the use of a cardiac pacemaker, the ECG becomes normal sinus rhythm after corrections for severe acidosis and hyperkalemia. This case demonstrates that severe acidosis and hyperkalemia can induce reversible CAVB without the use of a pacemaker. Hence, it should be evaluated as part of the initial assessment and therapy of CAVB before a cardiac pacemaker is implanted.

Keywords: complete atrioventricular block; hyperkalemia; pacemaker; septic shock; severe acidosis.

INTRODUCTION

Complete atrioventricular block (CAVB) or third-degree AV block is a medical condition characterized by the complete dissociation of the P wave and QRS complex on the electrocardiogram between the atrium and ventricle. CAVB is a life-threatening medical emergency and requires an immediate cardiac pacemaker to reduce mortality by up to 87%.[1] However, on rare occasions, this condition can be treated without the use of a cardiac pacemaker The following case demonstrates that severe acidosis and hyperkalemia can induce reversible CAVB without the use of a pacemaker.

CASE ILLUSTRATION

A 55-year-old lady was brought to the hospital in a comatose state, with GCS E2V1M1, Kussmaul breathing (respiratory rate 10-12 times per minute), blood pressure 54/37 mmHg, and a temperature of 35.9^{0}C. The patient's extremities were cold during physical examination, and her pulse was weak and slow (heart rate 67 beats per minute (bpm).

Alloanamnesis data from the patient's relatives stated that the patient had been unable to communicate for several days, was experiencing nausea and vomiting, and was unable to eat or drink. The patient had been complaining of shortness of breath for at least 12 hours before being transported to the hospital. Fever, cough, runny nose, and chest discomfort were all denied. The patient's medical history of hypertension, heart disease, diabetes, and cancer was all denied.

Dehydration was observed from the dry oral mucosa, and concentrated, minimal urine output (20 cc) from the catheter. A stress ulcer is identified by a dark red nasogastric tube product.

Laboratory tests demonstrated multi-organ failure (Table 1) as well as metabolic acidosis (Table 2). CAVB with non-malignant ventricular extrasystoles was discovered on electrocardiography (Figure 1).

The patient's early warning score is 14 points, the patient was diagnosed with septic shock, Multi-Organ Dysfunction Syndrome (MODS), CAVB with non-malignant ventricular extrasystoles, electrolyte imbalance, and severe metabolic acidosis requiring intensive care.

Table 1. Laboratory findings on the first day of treatments.

Parameters	Result
Hemoglobin	10.9 gr/dL
Hematocrit	34%
Leukocytes	16.7 $x10^3$/uL
Platelets	349 $x10^3$/uL
Erythrocytes	3.86 $x10^6$/uL
MCV	87.6 fL
MCH	28.2
AST	258 U/L
ALT	51 U/L
Blood glucose	232 mg/dL
Urea	114.0 mg/dL
Creatinine	3.20mg/dL
Sodium	136 mmol/L
Potassium	5.33 mmol/L
Chloride	96.3
Albumin	2.1 g/dL

Abbreviation: ALT = alanine aminotransferase; AST = aspartate aminotransferase; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin.

Table 2. Blood gas analysis on the first day of treatment.

Parameters	Day 1	Day 2
pH	7.190	7.460
PCO2	16.0 mmHg	21.0 mmHg
PO2	204.0 mmHg	210.0 mmHg
HCO3	5.9 mEq/L	14.6 mEq/L
TCO2	6.4 mmol/L	15.2 mmol/L
Base Excess	-20.3 mEq/L	-7.6 mEq/L
O2 Saturation	99 %	100 %
AaDO2	185.9 mmHg	456.8 mmHg
FIO2	60 %	100 %

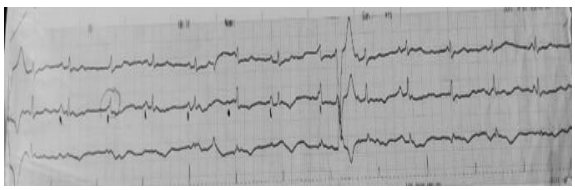

Figure 1. The initial electrocardiographic evaluation (ECG) revealed CAVB with non-malignant ventricular extrasystoles.

The patient's airway was clear, then a non-rebreathing mask with a flow rate of 10 liters per minute was used to raise oxygen saturation from 87 percent to 94-98 percent. After a fluid challenge failed to increase the patient's blood pressure, norepinephrine was titrated to reach the target mean arterial pressure (MAP) of 65 mmHg. Correction of the acidosis and hyperkalemia was then initiated after the patient's vital signs were stabilized. Within 8 hours, the patient is given 2 flasks of bicarbonate in 500cc of D5% solution. Hyperkalemia is treated with a drip of 10% calcium gluconate in 100cc of 0.9 percent NaCl solution and a gradual bolus of short-acting insulin in two flasks of D40 percent solution.

The patient was administered an 80 mg bolus of esomeprazole followed by a 5cc/hour drip of 80 mg esomeprazole in a 50cc syringe pump for stress ulcer conditions. To manage gastrointestinal bleeding, the patient was also given a 500 mg tranexamic acid injection and a 2 mg vitamin K injection. In this case, metronidazole 500 mg injections every 8 hours and ceftriaxone 1-gram injections every 12 hours were chosen as the antibiotics. The patient's vital signs and general condition is

monitored every 15 minutes with the help of a bedside monitor.

On the next day, the patient's condition remained stable, so a follow-up laboratory examination was carried out and found an improvement in the condition of acidosis and hyperkalemia (Table 2). ECG examination revealed spontaneous conversion to sinus tachycardia with a heart rate of 110 beats per minute and non-malignant ventricular extrasystoles (Figure 2).

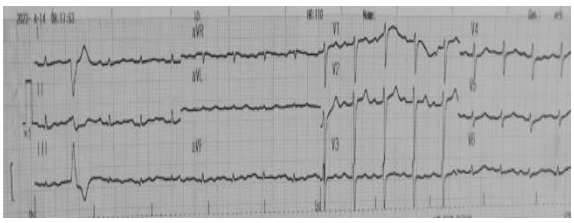

Figure 2. ECG examination after correction of acidosis and hyperkalemia.

Table 3. SOFA SCORE of patients in the first 24 hours in the hospital.

Variable	Points
Vasopressor requirement (norepinephrine > 6 mcg/min)	4
Glasgow coma score < 6	4
Creatinine ≥ 5 mg/dL or UOP < 200	4
PaO_2:FiO_2 ratio 301 - 400	1
Bilirubin < 1.2 mg/dL	0
Platelets > 150 ($x10^3/mm^3$)	0
Total	13

Abbreviations; UOP: Urine Output; PaO_2: Partial Pressure of Oxygen in Arterial Blood; FiO_2: Fraction of Inspired Oxygen.

However, the patient's condition deteriorated during the next few days. The Sequential Organ Failure Assessment (SOFA) score of this patient increased from 13 to 14 on the second day (Figure 3, Table 3). On the 5th day of treatment, the patient was supported using mechanical ventilation and maximum doses of vasopressors. The patient passed away the next day.

DISCUSSION

The atrioventricular block is the loss of the cardiac electroconductive impulse transmission function, which connects the sinoatrial node (SA node) and the ventricles via conduction through the atrioventricular node (AV node). Impaired impulse transmission can be temporary or permanent and may manifest as conduction delay in the AV node (first degree), intermittent conduction failure from the atrium to the ventricles (second degree), or complete AV block (third degree).[2]

Third-degree AV block or often referred to as complete AV block is a condition where communication between the atria and ventricles is completely cut off. Third-degree AV block occurs when no P waves are

transmitted and there is a complete dissociation of the atrial rate with ventricular rate. Complete atrioventricular (AV) block is a medical emergency, which is associated with a heart attack if left untreated.[3,4]

The etiology of AV block varies and is the same for all degrees of the block, namely structural and functional abnormalities. Structural changes consist of coronary artery disease, infection, congenital heart disease, inflammation, and neoplasms which generally cause permanent AV block. Functional disorders consist of autonomic disorders, drug toxicity (e.g., the use of antiarrhythmics or digoxin), and metabolic causes that often lead to reversible AV block conditions. Common metabolic causes of AV block are hypoxemia, hyperkalemia, acidosis, and hypothyroidism.[5,6]

The definitive treatment for CAVB according to the American Heart Association (AHA) guidelines is the implantation of a permanent pacemaker.[7] However, in this case, CAVB converted to sinus rhythm without the implantation of a permanent pacemaker.[7]

In this case report, the patient underwent mild hyperkalemia (K: 5.3 mmol/L) corrected to normal potassium serum (Corrected K: 4.07mmol/L) and CAVB converted into sinus rhythm. This result is similar to the result of the case reported by Kim et al (2005) in a CAVB patient with severe hyperkalemia (initial K: 7.9 mmol/L) decreased to normal potassium level (Corrected K: 5.03 mmol/L), ECG converted into sinus rhythm after insertion of a percutaneous pacemaker and correction of hyperkalemia.[8]

The case report by Kosovali and Yidliz (2018) also reported similar results in which an 84-year-old woman with type II diabetes mellitus, hypotension and CAVB with metabolic acidosis and mild hyperkalemia (K: 6.1 mmol/L) underwent conversion back to a normal rhythm. sinuses without a pacemaker or hemodialysis.[9]

Baratloo et al. reported case of a patient with severe hyperkalemia (initial K: 8 mmol/L) who also had metabolic acidosis (pH: 7.23) and CAVB, who had his cardiac rhythm converted into sinus, after the initial insertion of temporary transcutaneous cardiac pacemaker, correction of hyperkalemia, and hemodialysis.[10]

Severe hyperkalemia is an important etiology of CAVB.

Hyperkalemia is defined as serum potassium concentration >5 mmol/L, moderate (6-7 mmol/L) and severe (>7 mmol/L) are life-threatening and require immediate treatment. Hyperkalemia causes different degrees of electro-conduction block, including bundle Purkinje block and AV block. Electrocardiogram (ECG) findings of hyperkalemia may progress from high and elevated T waves, shortened QT interval to PR interval lengthening and loss of P waves, widening of the QRS complex, culminating in a "sine wave" morphology, and death if not treated. The correlation between the degree of ECG changes and AV block is usually associated with serum potassium levels >6.3 mmol/L.[11]

The effect of acidosis on the cardiac conduction system may be important, for example the excitability of Purkinje fibers is altered by exposure to low pH, whereas the rate of spontaneous sinoatrial nodal cells and small tissue preparations shows marked pH sensitivity. Thus, acidosis can significantly lengthen the AVN delay, which, when combined with short cycle lengths, can result in partial or complete AVN block and is therefore implicated in the development of bradyarrhythmia in conditions of local or systemic acidosis.[12]

This patient also had hyperglycemia of 232 mg/dL, which may have been caused by the septic shock's powerful inflammatory response. Recently, sepsis and systemic inflammatory response (SIRS) have been observed to induce catecholamine release. Previously believed to be mainly released from neuroendocrine cells, catecholamines are now known to be synthesized and released from leukocytes and macrophages. This involvement plays a major role in increasing glucose production during acute inflammatory disease.[13]

While in this case the patient experienced conversion of CAVB to normal sinus rhythm, the patient's general deterioration was probably due to septic shock and MODS condition. The mortality rate was more than 65% in patients with a SOFA score of more than 12.[14] This patient had SOFA SCORE 13 with an estimated mortality of 50-60%. The patient's SOFA SCORE the next day changed to 14 points (Figure 3). In 2001 Belgian research, serial SOFA scores were collected throughout the first 96 hours after admission. According to this study,

patients with a rising or stable SOFA score in the first 48-96 hours had a higher risk of mortality than those with a dropping score (Figure 4).[14]

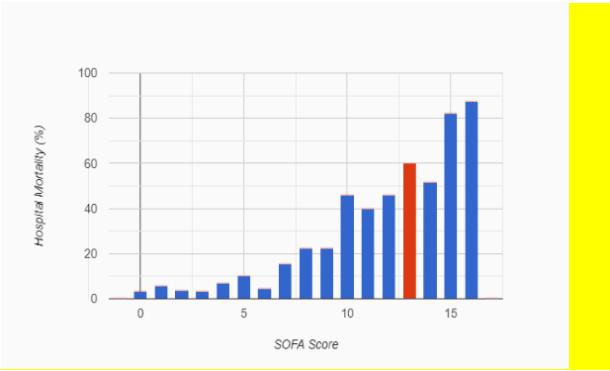

Figure 3. Hospital mortality rate associated with maximum SOFA score.

Table 4. The correlation between the sofa score trend and patient mortality during the initial 48 hours of treatment.[14]

Score Trend (first 48 hrs)	Mortality
Increasing	> 50%
Unchanged	27 - 35%
Decreasing	< 27%

CONCLUSION

This case demonstrates that in rare occasions, severe acidosis and hyperkalemia can cause reversible CAVB. Therefore, before a cardiac pacemaker is implanted, it should be evaluated as part of the initial evaluation and treatment of CAVB.

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