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A Case Report of Diarrhea-Induced Atrial Fibrillation

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Abstract

E.coli-induced diarrheal infection usually presents milder symptoms and recovers with antibiotics. But this case presents with atrial fibrillation, instead of ventricular arrhythmias which is the rarest complication of low potassium. The most prevalent sustained arrhythmia in older adults is atrial fibrillation. A three- to five-fold greater risk of stroke is linked to atrial fibrillation, as well as a greater risk of heart failure, cardiac mortality, and overall mortality. Hypokalemia (3.5 mmol/l), or low serum potassium, is thought to increase the risk of cardiovascular illness, particularly ventricular arrhythmias and cardiac arrest. The relationship between serum potassium levels and the risk of atrial fibrillation has only been briefly studied in the past. When E. coli causes diarrhoea, Low serum potassium, which is connected to a lengthening of P waves, a measure of atrial conduction. Atrial fibrillation risk has been linked to an increase in P-wave duration.

1. Introduction

Invasive, Non-toxigenic Enteropathic *Escherichia coli* and other bacteria like salmonella, shigella, and yersinia and viruses like Rotavirus, and cytomegalovirus can cause diarrhea, producing metabolic acidosis and hypokalemia when severe enough[1-4]. These diarrheal states have a less well-defined pathophysiology, but both increased secretion and absorption appear to be involved[5-10]. Guanylyl cyclase is activated by enterotoxin from pathogenic E. coli, which raises cyclic Guanosine monophosphate levels and stimulates Chloride secretion in intestinal epithelial cells[11]. Furthermore, experimental research revealed that inflammation downregulates the intestinal Chloride/Bicarbonate exchanger and Na+/K+-ATPase while also, non-specifically, increasing intestinal water permeability. In this article, we shall discuss the presentation and treatment of Atrial fibrillation resulted due to hypokalemia in Enteropathic E.coli infection.

2. Case Report

55 years old man presented to casualty with chief complaints of chest discomfort, palpitations, breathlessness, and fatigue since early in the morning. The patient also had chief complaints of lower abdominal pain, loose stools, watery in nature, no mucous present 5 to 6 episodes per day, and nausea for 20 days, patient had a history of anorexia for 7 days. He had a history of outside food intake 2 weeks back following the symptoms started. Patient is N/K/C/O systemic hypertension/ coronary artery disease/ bronchial asthma/stoke. k/c/o type 2 diabetes for 5 years on regular medications. The patient is non smoker, not an alcoholic consumer, mixed diet. On examination, the patient was afebrile[98F], and tachycardia was present, pulse was 175 beats/min, irregularly-irregular, and blood pressure was fluctuating, saturation was 87% on room air, with no pedal edema, pallor, or clubbing.

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Systemic examination- CVS- S1,S2 heard, JVP not raised. RS- bilateral air entry present, no crept, crackles, P/A – soft, epigastric tenderness present, no organomegaly. CNS – no focal neurological deficit

Laboratory investigations was done – ABG revealed severe metabolic Acidosis [PH- 7.2, PCO2- 17mmhg, hco3 -6.6mmol/L], total WBC count(leukocyte) was 18,932 (elevated) other parameters were normal. Renal parameters showed pre-renal AKI picture, serum potassium was 2.6 mmol/L, serum sodium was 130 mmol/L, serum chloride was 97 mmol/L, Fecal complete analysis showed 18 to 20 pus cells, Fecal culture sensitivity showed E.coli growth. Ecg showed fibrillatory wave(Fig 1, Fig 2)

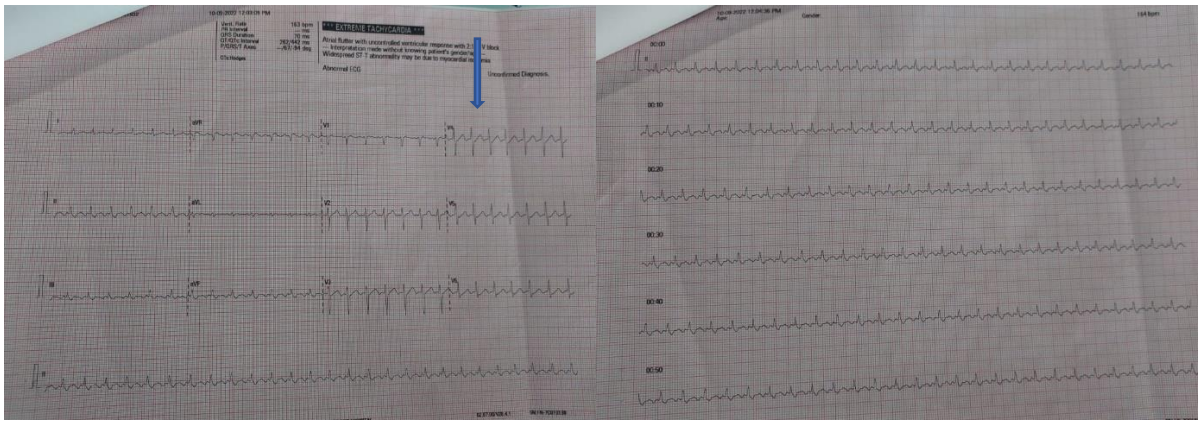

Figure 1 (arrow mark showing fibrillatory waves)

Figure 2 rhythm strip

Cardiac enzymes sent TROP I was ELEVATED. Creatinine Kinase -MB was found to be positive, and ECHO showed normal. CT Brain showed no significant abnormality and age-related atrophy.

The patient's potassium was corrected with IV KCL and metabolic acidosis was corrected by 100mEq/L Sodium Bicarbonate, Rhythm controlled by pharmacological cardioversion IV AMIODARONE regimen loading dose of IV Amiodarone 150mg over 1st 10 minutes (15mg/min) followed by 360 mg over next 6 hours (1mg/min), a maintenance infusion of 540 mg over remaining 18 hrs (0.5mg/min) and loading dose given(CLOPIDOGREL 300MG+ASPIRIN 300MG+ATORVASTATIN 80MG) , repeat ECG reverted to normal sinus rhythm, CHA2DS2-VASc score is 1 so no anticoagulants added, dehydration corrected with adequate IV fluids and treated with higher antibiotics of IV Metronidazole 500mg and IV Ciprofloxacin 500mg BD for 7 days and other supportive management given, patient showed improvement after 5 days of therapy,Renal parameter came to normal, repeat ECG was taken at time of discharge and it was found to be normal. We had follow-up on patient after one month of discharge, patient was perfectly fine with no symptoms and ECG was normal.(Fig 3)

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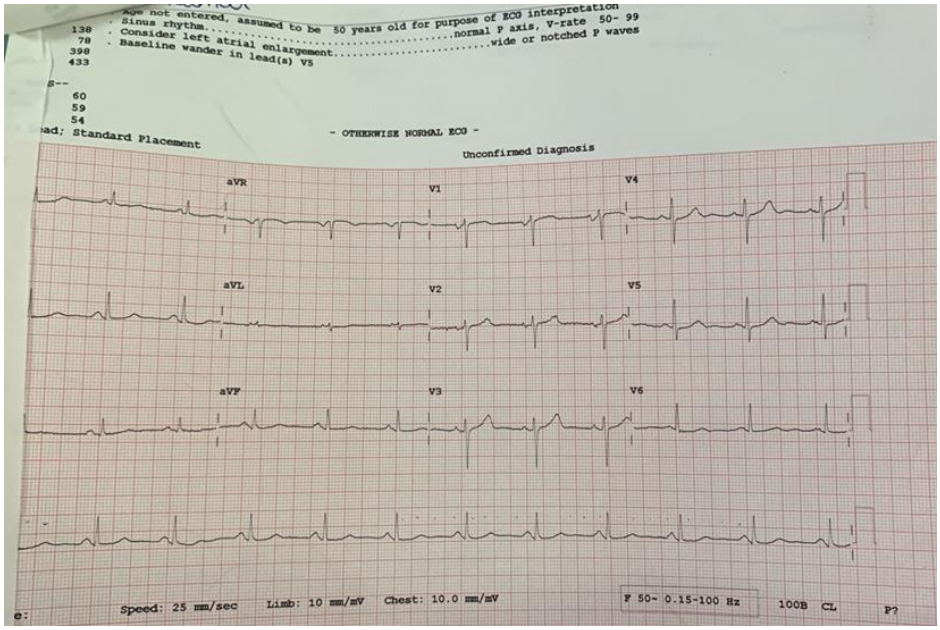

Figure 3 (normal sinus rhythm)

3. Discussion

Up to 20% of hospitalized patients with diarrhea experience hypokalemia (serum potassium <3.5mmol/l),

which is linked to an elevated risk of ventricular and atrial fibrillation. It is uncertain whether these various arrhythmias are a direct outcome of hypokalemia's possible unique effects on cardiomyocytes.[12]. When compared to individuals with normokalemia, patients with hypokalemia had a 5-fold higher incidence of ventricular fibrillation, which is thought to be the cause of the increased mortality. Additionally, patients with hypokalemia are more likely to experience atrial fibrillation; this connection is unaffected by age, gender, serum [Magnesium]levels, or any other potential confounders.[13]. It has been proposed that hypokalemia's arrhythmogenic effects are caused by a predisposition for ectopic (provoked) activity. Despite the fact that hypokalemia is also linked to atrial fibrillation, it is unclear whether or how this condition favors triggered activity in atrial cardiomyocytes. Atrial cells typically display a modest but variable density of t-tubules and related Na+ and Ca2+ handling proteins, which fundamentally differs from ventricular cells' myocyte structure and Ca2+ homeostasis. That atrial cells exposed to hypokalemic circumstances will experience Ca2+ overload in a way similar to ventricular cells is therefore counterintuitive. As a result of delayed repolarization and reactivation of L-type Ca2+ channels, the Action Potential is also considerably shorter in atrial cells, which may prevent the production of Early After Depolarization. But recent research has suggested that cells with shortened Action Potential's may be vulnerable to phase-3 Early After Depolarization triggered by nonequilibrium reactivation of Na+ current (INa).

Atrial fibrillation is one of the rare complications of diarrhea. Only a very few cases have been reported with this complications, symptoms of atrial fibrillation range from chest discomfort to cardiogenic shock and arrest, and diagnosis of Atrial fibrillation can be done using ECG, or ECHO. ECG helps in monitoring of the patient and the prognosis of the patient.

4. Conclusion

Atrial fibrillation following infections of E.coli was found to be rare and if it is not treated appropriately, the patient may go into sudden cardiac arrest and die. ECG changes have always been helpful in diagnosing Atrial fibrillation, and starting appropriate treatment immediately.

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