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Acute Renal Failure and Hemolytic Anemia After Electrical Cardioversion In A Patient With Paroxysmal Atrial Fibrillation.

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Abstract

A 65-year-old man presented progressively worsening shortness of breath due to acute decompensated heart failure with a first episode of atrial fibrillation (Afb) and rapid ventricular response. Due to his symptoms, it was decided to restore the sinus rhythm by applying electrical current cardioversion (EC). However, four days after discharge, the patient presented with anorexia, a recurrence of dyspnea, and weakness. ECG showed sinus rhythm. Laboratory tests revealed elevated serum urea and creatinine, decreased GFR, and severe anemia of hemolytic type. He remained non-oliguric. After a few days, the abnormal finding progressively resolved. On the occasion of this case report, we describe the literature data on the complications of EC in renal function and hematological profile. There is limited data on acute kidney injury occurrence and only one report on hemolytic anemia immediately after successful treatment of Afb by EC.

Keywords: Acute kidney injury, Hemolytic anemia, Atrial fibrillation, Electrical cardioversion.

Introduction

Current guidelines for managing new-onset atrial fibrillation (Afib) include two treatment approaches: heart rate control and rhythm control **[1]**. The choice depends on the patient's clinical status and personal preference. The EAST-AFNET 4 (Early Treatment of Atrial Fibrillation for Stroke Prevention) trial suggested that early rhythm-control therapy was associated with a lower risk of adverse cardiovascular outcomes than usual care of rate control **[2,3]**. Rhythm control included antiarrhythmic drug therapy, catheter ablation, or electrical cardioversion (EC). Furthermore, maintaining the rhythm is highly dependent on managing risk factors such as obesity, physical inactivity, sleep apnea, diabetes mellitus, hypertension, and other modifiable lifestyle-related factors **[4]**.

Direct cardioversion has been performed for Afib for over 40 years. It is a safe, effective, and reliable method for aborting Afib, especially in unstable situations **[5]**. Direct EC is superior to pharmacologic conversion because of its shorter overall procedure duration, higher success rate, and lower risk of proarrhythmic **[6,7]**. Indications for urgent EC in patients with Afib include active myocardial ischemia, significant hypotension, acute heart failure, and accessory pathways with preexcitation. However, it is still being determined what energy setting is necessary for a specific density to reach the myocardium **[6]**. The most often complications associated with EC are thromboembolic events and post-cardioversion arrhythmias **[8]**. Other side effects include temporary low blood pressure, myocardial damage (usually temporary and without symptoms), and skin burns. Acute renal dysfunction post-EC (post-cardioversion renal failure) is an uncommon complication **[9,10]**. Moreover, there is only one report regarding erythrocyte hemolysis after EC **[11]**. On the contrary, Noszczyk-Nowak et al. suggested that EC in dogs did not clinically or statistically affect red blood cells **[12]**.

In this case report, we describe acute kidney injury and marked anemia following cardioversion for atrial fibrillation, highlight their incidence, and describe the possible mechanisms of that unusual complications.

Case presentation

A 65-year-old man presented to the clinic due to severe shortness of breath, orthopnea, easy fatigue, palpitations, and lower leg swelling last week. His medical history reported arterial hypertension, diabetes mellitus, mild heart failure, and mild ankylosing spondylitis. His medication included Nifedipine, Irbesartan, Metformin, and Methotrexate. The ECG in his medical examination before the 15th day had findings of benign hypertrophy of the left ventricle with sinus rhythm **(Figure 1)**.

On admission, his vital signs were

- a body temperature of 36.6 degrees C,
- a pulse rate of 120 beats per minute,
- a respiration rate of 16 breaths per minute,
- blood pressure of 114/75 mmHg, and

- saturating 94 % of room air.

Physical examination revealed bibasilar crepitations, elevated jugular venous pulsation, and bilateral leg edema. Electrocardiogram revealed atrial fibrillation with a rapid ventricular rate of >120 bpm **(Figure 1)**. A chest x-ray revealed bilateral mild pulmonary vascular congestion. An echocardiographic study showed a left ventricular ejection fraction of 48 %, with grade II diastolic dysfunction, moderately dilated left ventricle, normal right ventricle, mildly dilated left atrium, mild mitral regurgitation, and no other structural valve abnormalities, the pericardium was free. Laboratory measurements revealed Na-135 mmol/L, K-5.2 mmol/L, Cl- 119 mmol/L, HCO3- 25 mmol/L, BUN- 47mg/dL, Cr- 1.3 mg/dL, GFR- 49 ml/min/1.82 m2, AST-27U/L, ALT- 29 U/L, HbA1c- 6.4, WBC- 5.9, HGB- 12.7 g/dl, Hct 41%, Troponin - normal, D-dimer- normal, BNP-346, TSH- normal.

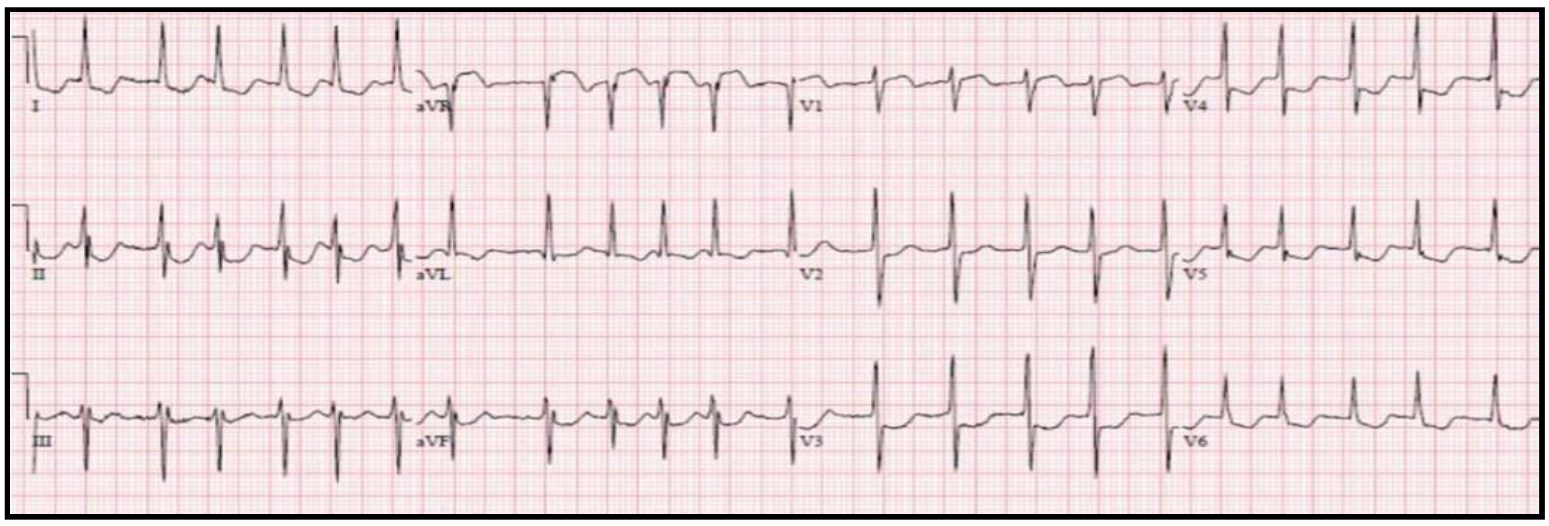

Figure 1: ECG of the patient with rapid atrial fibrillation.

A transesophageal echocardiography was performed, which showed similar findings to the transthoracic echocardiography. Additionally, no evidence of thrombus in the atrial was found. Due to the patient clinical picture, which was attributed to the first-onset atrial fibrillation, it was decided to avert it with electrical cardioversion. The patient was informed about the plan and signed a consent form before the procedure. He was anesthetized with propofol (1 mg/kg IV bolus followed by 0.5 mg/kg). Then, direct current cardioversion of 200 joules was performed without a successful result. We achieved a sinus rhythm with a second EC of 300 joules. The patient was hemodynamically stable, without any hypotensive episodes, before and following the procedure. After successful conversion to sinus rhythm (80 bpm- **figure 2**), the patient was treated with Apixaban, Carvedilol, Amiodarone, Losartan, and Furosemide. His clinical picture improved significantly, and the patient was discharged the next day.

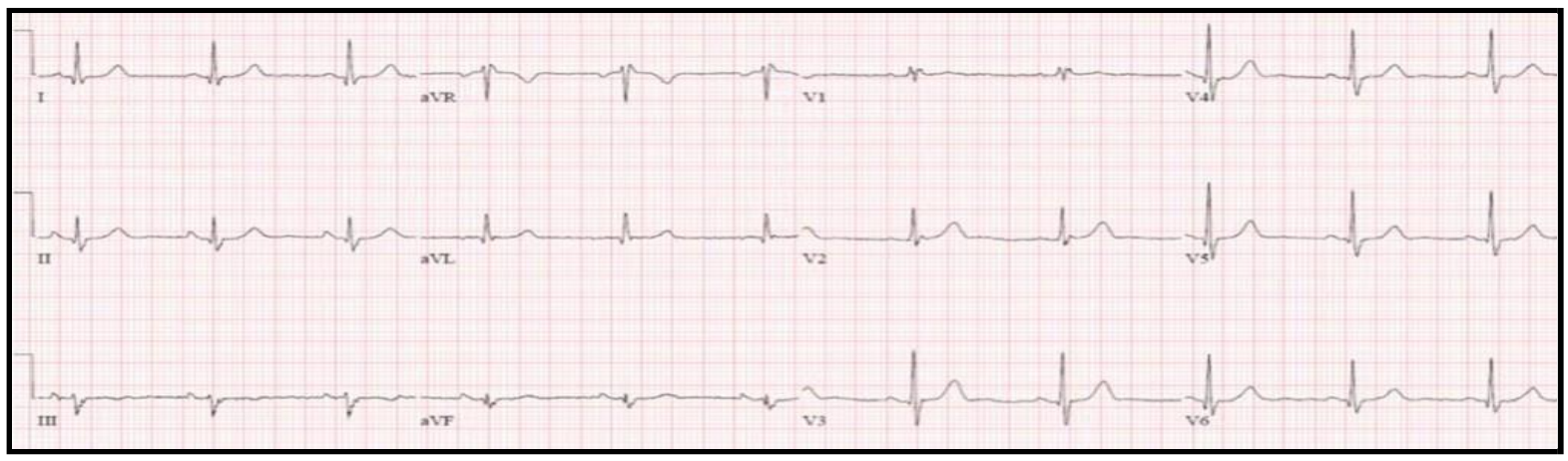

Figure 2: ECG after the restoration of sinus rhythm

While at home, he complained of anorexia, recurrence of dyspnea, and progressive muscle weakness that negatively affected his functional ability. He reported no bleeding manifestations from any organ. He remained non-oliguric. No particularly pathological findings were found from repeating the clinical cardiac evaluation on the 4th day after EC. Also, the ECG revealed sinus rhythm, and the LV EF was estimated at 55% from the echocardiographic study. In addition, laboratory tests revealed Na-130 mmol/L, K-4.8 mmol/L, Cl- 106 mmol/L, HCO3- 20 mmol/L, BUN- 105 mg/dL, Cr- 2.6 mg/dL, GFR- 35 ml/min/1.82 m2, AST-35 U/L, ALT- 40 U/L, Glycose 115 mg/dL, WBC-4.6, HGB- 8.5 g/dl, Hct 30 %, average platelet count, serum total bilirubin 3.2 mg/dL, unconjugated bilirubin 3.8 mg/dL, LDH 380 U/L, CPK 105 mcg/L. Reticulocyte counts 2.8 %, IgM 430 IgG mg/dL, 1800 mg/dL, positive indirect Coombs test. Mild hemoglobinuria was found; negative tests for occult fecal bleeding. Renal ultrasonography was routine.

Then, a gradual trend of urea and creatine downwards to 76 mg/dL and 1.9mg/dL correspondingly was mentioned after a few days. The level of the HGB increased progressively to 9.6 g/dl and of Hct to 34 %. The patient was seen in the clinic three months after heart rhythm restoration. He was in stable clinical status with sinus rhythm. Creatinine level of 1.4 mg/dL showing continuing renal improvement since EC. Also, Hgb was at an average level (12.1 g/dl).

Discussion

According to the current literature, it is the first case report of the combined complication of acute kidney injury and hemolytic anemia due to the side effects of direct EC's application for restoring atrial fibrillation to sinus rhythm.

Cardioversion is a low-risk and standard procedure to restore normal sinus rhythm in patients with persistent Afb or short-term paroxysmal Afb leading to severe symptoms **[5-7]**. The most frequently reported complications are thromboembolic events and the occurrence of arrhythmias. The risk is greater in patients with persistent Afb **[13]**. In new-onset Afb of less than 48 hours, EC can be applied without prior administration of anticoagulants after a patient assessment, including using the CHA2DS2-VASc score and following the screening with transesophageal echocardiography **[8,14]**. Although the risk of thromboembolism is relatively low in these cases, it still exists **[15,16]**. Thus, dislodged blood clots can cause stroke, pulmonary embolism, or other complications. The European guidelines state that all patients undergoing cardioversion for persistent Afb should have a minimum of 4 weeks of anticoagulation **[17]**. Another potential complication of EC is arrhythmias, especially Brady-arrhythmic events **[18]**. They seem to reflect sinus node dysfunction and often result in the later implantation of a permanent pacemaker **[19]**.

Limited data exist regarding kidney dysfunction's incidence and clinical impact following EC for Afb. It was supported that impaired renal function, based on estimated GFR, is relatively common (17 %) and is associated with increased mortality in patients with Afb undergoing successful EC **[21]**. A higher incidence of kidney dysfunction after EC was reported in patients with advanced heart failure and diabetes mellitus **[21]**. Usually, an increase in serum creatinine by 25-50 % compared to baseline was found within 48hrs of EC **[10,22]**. Studies did not clearly define the duration of kidney damage after EC. In our case, on the seventh day after direct EC, we found a significant reduction in GFR; after a month, the renal damage had been restored. Sinus rhythm maintenance was related to improved eGFR in patients with mild or moderate renal insufficiency **[10,23].** However, some cases need to treat by dialysis **[9,21]**. Schmidt et al. suggested that impaired renal function is directly associated with a risk of Afb recurrence after successful EC **[20]**.

Although the pathophysiology of renal dysfunction is not clearly understood, it is reasonable to attribute it to renal hypoperfusion resulting from hemodynamic and fluid balance changes post-cardioversion **[10].** It could be argued that the use of propofol is responsible for hypotension and subsequent renal damage. Propofol is a short-acting anesthetic drug producing conscious sedation during EC. It has a negative inotropic effect and causes vasodilatations in both venous and arterial circulations, reducing systemic blood pressure **[5]**. We did not observe a particular hypotensive episode in our patient after EC and the use of propofol as an anesthetic. Another possible mechanism that could be suggested is the direct tissue injuries of the electrical shock. Evidence shows that natural current flow through the patient's body can cause minimal cell injury, mainly in the myocardium **[24]**. However, others found no cardiac or clinically significant damage assessed using troponin levels **[25]**. As revealed by increased blood creatinine kinase levels, EC also results in skeletal muscle damage and is related to successive energy additions **[26]**. Therefore, rhabdomyolysis may be a cause of renal failure **[26].**In our case, we did not find a significant increase in CPK or myoglobinuria, suggesting that muscle damage may not be present. However, laboratory measurements were made on the fourth day after EC.

An interesting finding in our case was the presence of anemia in the patient after EC, which according to the laboratory findings, was attributed to red blood cell hemolysis. Only one study with a similar result was found in the literature. Makowski et al.**[11]** 30 min after EC with a mean energy of 170 J, red blood cells, HCT, and hemoglobin were significantly lower, whereas direct bilirubin concentration after six h was substantially higher. As mentioned previously, electrocution injuries occur due to a high electrical field strength causing thermal or electrochemical damage to body tissues. It is well known that high electrical field strength causes protein coagulation, coagulation necrosis, hemolysis, thrombosis, muscle or tendon avulsion, acute kidney injury, and dehydration **[27]**. Moroz et al. **[28]** studied the effects of high-voltage single, double unipolar, and double bipolar electric pulses of exponential or sine shape on erythrocyte membranes. Two successive pulses significantly decreased the erythrocyte number. Like defibrillation of the whole heart, the effect of two bipolar pulses on erythrocytes was more pronounced than that of two unipolar pulses **[28]**. Indeed this information refers to the action of high voltage electrical energy, much higher than EC, to restore Afb. Moreover, Noszczyk-Nowak et al. **[12]** suggested that low voltage EC in dogs did not affect red blood cells either clinically or statistically after the successful restoration of Afb. A limitation of their study was that blood sampling was done immediately after the shock, so the EC's effects might differ after a more extended period.

In addition to the toxic effect on the erythrocyte membrane of electrical defibrillation, another possible mechanism that can lead to hemolytic anemia is the damage to the endothelium of the vessels. Hemolysis is directly involved in endothelial injury **[29]**. Freestone et al. **[30]** described vascular endothelial dysfunction four weeks after cardioversion. This injury and shedding of endothelial cells post-cardioversion may contribute to hemolysis and thromboembolic risk. Another explanation of anemia may be that in > 80 % of patients with acute renal failure, anemia is present and shown to be related to a decrease in GFR and the presence of oliguria **[31,32]**. In these cases, anemia may be due to the lack of intrinsic EPO secretion, mainly induced by acute renal tubular interstitial injury **[33]**. Moreover, idiopathic or symptomatic acquired auto-immune hemolytic anemia

with hemoglobinemia and hemoglobinuria is an unusual cause of acute renal failure **[34]**. In addition, intravascular hemolysis caused by mechanical trauma, such as cardiac valvular disease or prosthesis in patients with pre-existing kidney dysfunction, can lead to a further decrease in kidney function **[35]**. Therefore, acute hemolysis is a cause of acute renal failure due to tubular damage caused by the formation of intratubular casts and not the result **[36]**.

A limitation of our case study is that the clinical evaluation and repeated laboratory tests were done a little late, that is, on the fourth day after EC. However, we suggest a bidirectional mechanism of acute kidney injury and hemolytic anemia. Thus, it is most likely that the two pathological entities manifested as separate rare complications of EC.

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