ELECTRICAL CARDIOVERSION FOR ATRIAL FLUTTER IN A PATIENT WITH SEVERE LEFT VENTRICULAR SYSTOLIC DYSFUNCTION: A CASE REPORT

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Abstract

Background: Patients with chronic heart failure (CHF) and severe left ventricular systolic dysfunction (LVEF <35%) are at elevated risk for life-threatening arrhythmias. Atrial flutter (AFL) can precipitate acute hemodynamic instability in this population, often requiring urgent intervention.

Case Description: A 61-year-old male with ischemic cardiomyopathy (LVEF 31-34%), an implantable cardioverter-defibrillator (ICD), and paroxysmal atrial fibrillation presented with CHF decompensation. He developed typical AFL with 2:1 atrioventricular (AV) conduction, prompting intensive care unit (ICU) transfer due to impending hemodynamic deterioration. Despite optimized guideline-directed medical therapy, synchronized electrical cardioversion with a 100 J biphasic shock successfully restored sinus rhythm without complications.

Conclusion: This case illustrates the safety and efficacy of low-energy electrical cardioversion for acute rhythm control in AFL among patients with advanced heart failure and severe systolic dysfunction. It underscores the value of vigilant monitoring, the limitations of ICD therapy for supraventricular arrhythmias, and the need for multidisciplinary, individualized management to avert decompensation in high-risk patients.

Keywords: Atrial flutter, electrical cardioversion, heart failure, left ventricular dysfunction, implantable cardioverter-defibrillator

ЭЛЕКТРИЧЕСКАЯ КАРДИОВЕРСИЯ ПРИ ТРЕПЕТАНИИ ПРЕДСЕРДИЙ У ПАЦИЕНТА С ТЯЖЁЛОЙ СИСТОЛИЧЕСКОЙ ДИСФУНКЦИЕЙ ЛЕВОГО ЖЕЛУДОЧКА: КЛИНИЧЕСКИЙ СЛУЧАЙ

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Аннотация

Введение: Пациенты с хронической сердечной недостаточностью (ХСН) и тяжёлой систолической дисфункцией левого желудочка (ФВ ЛЖ <35%) имеют повышенный риск развития жизнеугрожающих аритмий. Трепетание предсердий (ТП) может спровоцировать острую гемодинамическую нестабильность у этой категории пациентов, часто требующую неотложного вмешательства.

Описание случая: Мужчина 61 года с ишемической кардиомиопатией (ФВ ЛЖ 31–34%), имплантированным кардиовертером-дефибриллятором (ИКД) и пароксизмальной фибрилляцией предсердий поступил в клинику с декомпенсацией ХСН. У пациента развилась типичная ТП с атриовентрикулярным (АВ) проведением 2:1, что потребовало перевода в отделение интенсивной терапии (ОИТ) в связи с надвигающимся

ухудшением гемодинамики. Несмотря на оптимизированную медикаментозную терапию, синхронизированная электрическая кардиоверсия с бифазным разрядом 100 Дж успешно восстановила синусовый ритм без осложнений.

Заключение: Данный случай иллюстрирует безопасность и эффективность низкоэнергетической электрической кардиоверсии для острого контроля ритма при ТП у пациентов с тяжелой сердечной недостаточностью и тяжелой систолической дисфункцией. Он подчеркивает важность бдительного мониторинга, ограничения терапии ИКД при наджелудочковых аритмиях и необходимость мультидисциплинарного индивидуализированного лечения для предотвращения декомпенсации у пациентов высокого риска.

Ключевые слова: Трепетание предсердий, электрическая кардиоверсия, сердечная недостаточность, дисфункция левого желудочка, имплантируемый кардиовертер-дефибриллятор

Introduction

Chronic heart failure (CHF) with reduced left ventricular ejection fraction (LVEF <35%) confers a substantial risk of malignant arrhythmias due to extensive myocardial remodeling [1]. Atrial flutter (AFL), a supraventricular tachyarrhythmia, can exacerbate hemodynamic instability in these patients, potentially leading to rapid decompensation, cardiogenic shock, or sudden cardiac death [2]. We report a case of typical AFL with 2:1 AV conduction in a patient with advanced ischemic cardiomyopathy and an ICD, necessitating urgent electrical cardioversion despite ongoing medical optimization. This case highlights the diagnostic and therapeutic challenges in managing supraventricular arrhythmias in structurally compromised hearts and the role of low-energy cardioversion as a bridge to advanced therapies.

Case Presentation

A 61-year-old male (Patient S.) was admitted to the 10th Cardiac Surgery Department of the Saint Petersburg State Veterans Hospital on April 8, 2025, for decompensated CHF.

Medical History

The patient had a history of coronary artery disease (CAD) with inferior myocardial infarction in 2016, progressing to ischemic cardiomyopathy (NYHA class II). Echocardiography confirmed LVEF 31-34%, left ventricular (LV) dilation, and moderate mitral regurgitation. An ICD was implanted in October 2024 for primary prevention of sudden cardiac death. Comorbidities included type 2 diabetes mellitus, chronic obstructive pulmonary disease (COPD), paroxysmal atrial fibrillation (AF), and prior episodes of AV re-entry tachycardia (AVRT). He was on guideline-directed medical therapy (GDMT) for CHF, anticoagulation, antiarrhythmic prophylaxis, antidiabetic, hypolipidemic, and gastroprotective agents (Table 1).

• *Table 1. Pre-admission and Hospital Medications*

	Drug name	Dosage	Frequency
Anticoagulation therapy			
1	Tab. Warfarin	2.5 mg	Morning
Antiarrhythmic therapy			
2	Tab. Amiodarone	200 mg	Twice a day
Chronic Heart Failure therapy			
3	Tab. Metoprolol succinate	12.5 mg	Twice a day
4	Tab. Torsemide	10 mg	Morning
5	Tab. Valsartan + Sacubitril	25 mg	Twice a day
Antidiabetic therapy			
6	Tab. Dapagliflazine	10mg	Evening
Hypolipidemic therapy			
7	Tab. Atorvastatin	40 mg	Evening
Gastroprotective therapy			
8	Tab. Esomeprazole	20 mg	Evening

On admission, electrocardiography (ECG) revealed sinus bradycardia with ST elevation in leads III and aVF, and Q waves in inferolateral leads, consistent with prior infarction. Laboratory findings showed normal troponin I (0.005 ng/mL), C-reactive protein (2.05 mg/L), and lipid profile, but elevated triglycerides (2.60 mmol/L), prothrombin time (37.2 sec), and activated partial thromboplastin time (APTT; 41.09 sec), reflecting therapeutic anticoagulation (Table 2). Complete blood count was unremarkable except for mildly elevated erythrocytes (5.6×10^{12}/L).

• *Table 2. Admission Laboratory Results (April 8, 2025)*

Biochemistry Analysis				**Blood analysis**			
Analyst	*Result*		*Date*	*Analyst*	*Result*		*Date*
Troponin I	0.005 ng/ml	Normal	08.04.2025	Hemoglobin	147 gm/l	Normal	08.04.2025
C-reactive protein	2.05 mg	Normal	08.04.2025	Erythrocytes	5.6 (1012/l)	Slightly raised	08.04.2025
APTT	41.09 sec	Raised	08.04.2025	Platelets	247 (109/l)	Normal	08.04.2025
Prothrombin time	37.2 sec	Raised	08.04.2025	Avg. Platelet volume	8.5 fl	Normal	08.04.2025
Prothrombin Index	21.60%	Decreased	08.04.2025	Platelet distribution width	15.70%	Normal	08.04.2025
Trygliceride	2.60 mmol/l	Raised	08.04.2025	Leukocytes	7.7 (109/l)	Normal	08.04.2025
Total Cholesterol	3.90 mmol/l	Normal	08.04.2025	Neutrophils	4.1 (109/l)	Normal	08.04.2025

LDL	1.37 mmol/l	Normal	08.04.2025	Lymphocytes	2.9 (109/l)	Normal	08.04.2025
HDL	1.35 mmol/l	Normal	08.04.2025	Lymphocyte percentage	36.80%	Normal	08.04.2025

Echocardiography demonstrated dilated LV and left atrium, LVEF 34%, moderate mitral regurgitation, and mild pulmonary hypertension.

On April 30, 2025, ECG documented AVRT with QRS duration 0.20 s and ventricular rate 150 bpm. By May 1, 2025, ECG showed typical AFL with 2:1 AV conduction (ventricular rate ~120 bpm), complete right bundle branch block, and ST depression in leads V4-V6 (Figure 1). Given the risk of hemodynamic instability, the patient was transferred to the ICU.

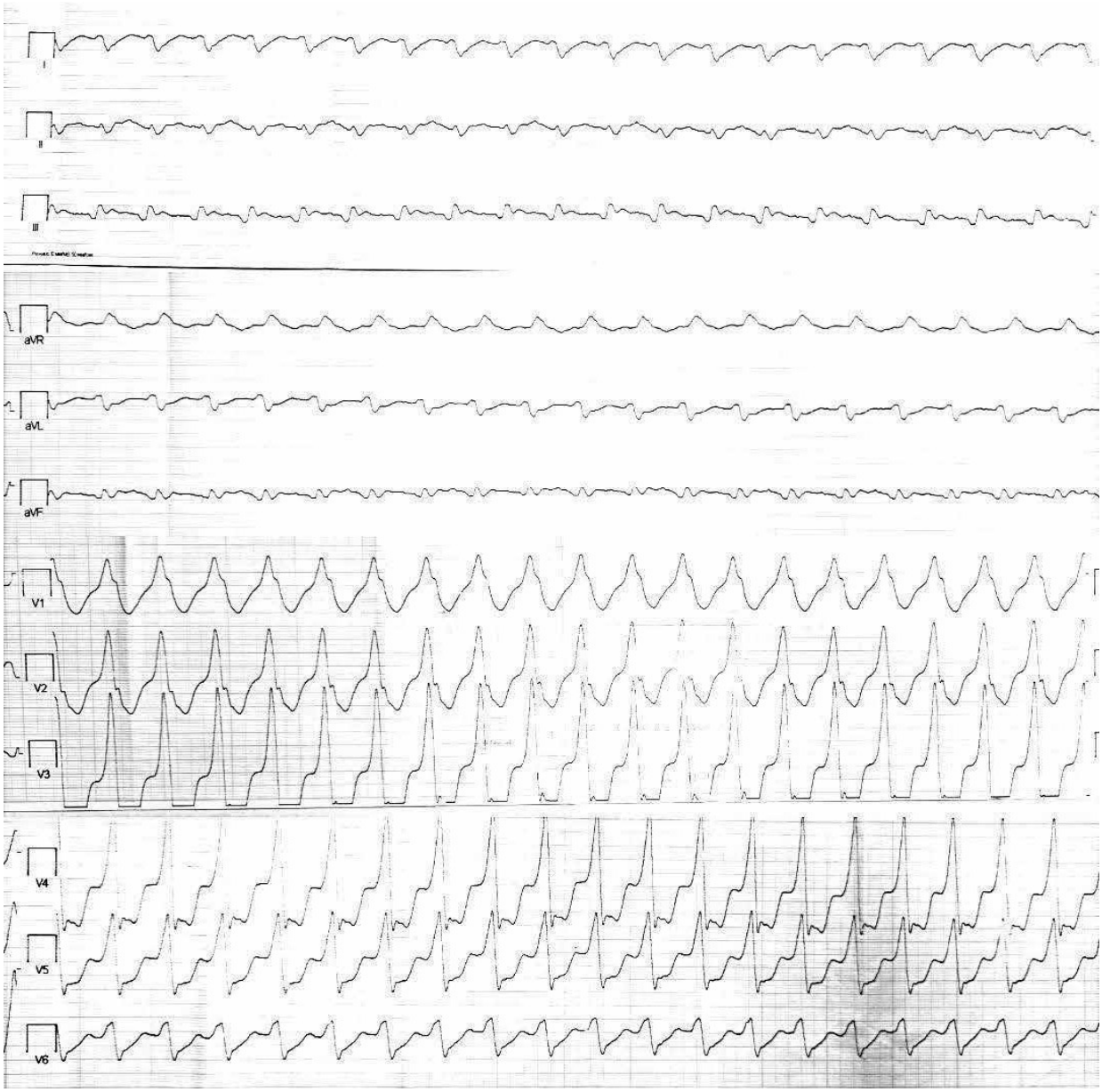
Pre-cardioversion laboratories (May 1, 2025) indicated mildly elevated troponin I (0.226 ng/mL), urea (8.9 mmol/L), creatinine (129.1 μmol/L), prothrombin time (48.5 sec), and APTT (40.9 sec), with leukocytosis (11.5 × 10^9/L) suggesting stress response (Table 3).

• *Table 3. Pre-Cardioversion Laboratory Results (May 1, 2025)*

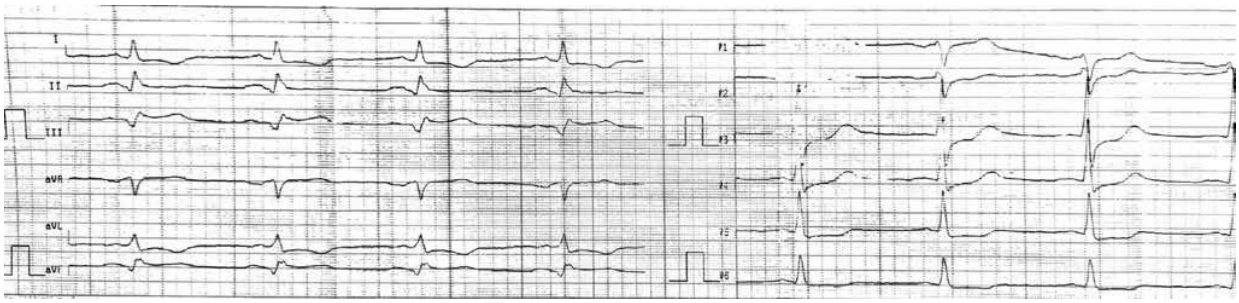
Biochemistry Analysis			**Blood analysis**				
Analyst	*Result*		*Date*	*Analyst*	*Result*		*Date*
Troponin I	0.226 ng/ml	Slightly raised	01.05.2025	Hemoglobin	162 gm/l	Normal	01.05.2025
C-reactive protein	3.18 mg	Normal	01.05.2025	Erythrocytes	5.85 (1012/l)	Slightly raised	01.05.2025
APTT	40.9 sec	Raised	01.05.2025	Platelets	348 (109/l)	Normal	01.05.2025
Prothrombin time	48.5 sec	Raised	01.05.2025	Avg. Platelet volume	9.5 fl	Normal	01.05.2025
Prothrombin Index	17.90%	Decreased	01.05.2025	Platelet distribution width	16.20%	Normal	01.05.2025
Potassium	5.21 mmol/l	Normal	01.05.2025	Leukocytes	11.5 (109/l)	Normal	01.05.2025
Urea	8.9 mmol/l	Raised	01.05.2025	Neutrophils	7.8 (109/l)	Normal	01.05.2025
Creatinine	129.1 mmol/l	Raised	01.05.2025	Lymphocytes	3.0 (109/l)	Normal	01.05.2025
				Lymphocyte percentage	26.20%	Normal	01.05.2025

Intervention

Synchronized electrical cardioversion was performed using a single 100 J biphasic shock. Sinus rhythm was immediately restored (Figure 2). Post-procedure, hemodynamics stabilized without complications or arrhythmia recurrence. The patient remains hospitalized pending heart transplantation evaluation.



• *Figure 1. ECG during AFL with 2:1 AV conduction (May 1, 2025). Sawtooth flutter waves in inferior leads, ventricular rate ~120 bpm.*



• *Figure 2. Post-cardioversion ECG showing restored sinus rhythm.*

Discussion

This case exemplifies the precarious interplay between supraventricular arrhythmias and advanced systolic heart failure. The patient's ischemic cardiomyopathy, compounded by recurrent arrhythmias (AVRT and AFL), illustrates how AFL with 2:1 conduction can precipitate decompensation in remodeled ventricles, reducing cardiac output and worsening ischemia [2,3]. Despite ICD implantation, device therapy failed to address AFL, as ICDs are primarily designed for ventricular tachyarrhythmias [3]. Amiodarone, while continued, proved insufficient for acute rhythm control, aligning with evidence of variable efficacy in AFL [2].

The decision for electrical cardioversion was guided by ESC and AHA guidelines, recommending synchronized DC cardioversion for hemodynamically unstable AFL, starting at 100-200 J biphasic energy [1,4]. The low-energy (100 J) approach minimized risks in this fragile patient, achieving immediate success without peri-procedural complications such as thromboembolism—facilitated by therapeutic anticoagulation—or myocardial stunning. Post-cardioversion troponin elevation was mild and attributable to demand ischemia rather than procedural injury.

Key takeaways include: (1) the imperative for continuous ECG monitoring in advanced CHF, especially pre-transplant, to detect occult arrhythmias [6]; (2) challenges in distinguishing AFL from AVRT in accessory pathway contexts, necessitating electrophysiology consultation; and (3) the superiority of cardioversion over pharmacotherapy in acute, unstable scenarios [5]. Multidisciplinary input optimized outcomes, emphasizing tailored GDMT re-titration post-rhythm control [6]. Limitations of this report include its single-case nature and lack of long-term follow-up; prospective studies are needed to evaluate cardioversion's durability in similar cohorts [7].

Conclusion

In patients with severe LV systolic dysfunction, low-energy synchronized electrical cardioversion offers a safe, effective means of restoring sinus rhythm in acute AFL, preventing hemodynamic collapse and facilitating GDMT optimization. This case reinforces the need for vigilant surveillance, recognition of ICD limitations for supraventricular rhythms, and integrated care models to improve short- and long-term prognosis. Future research should explore prophylactic strategies, including enhanced monitoring and ablation, in high-risk heart failure phenotypes.

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