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Case Report

Cardiac Rhythm Management A 65 year old Elephantiasis Male with Hemodynamic Collapse- Role of IV calcium gluconate

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Received date: September 22, 2025; **Accepted date:** October 01, 2025; **Published date:** October 10, 2025

Citation: Ramachandran Muthiah, (2025), Cardiac Rhythm Management A 65 year old Elephantiasis Male with Hemodynamic Collapse- Role of IV calcium gluconate, *Cardiology Research and Reports*, 7(6); **DOI:10.31579/2692-9759/179**

Abstract

Acute myocardial infarction (AMI), defined as ischemic necrosis of the myocardium, has a global prevalence of 3.8% in individuals under 60 years of age and 9.5% in those over 60 years. Various arrhythmias have been reported in AMI, but atrial flutter with variable block has been rarely described in the literature. Among these, bradyarrhythmias, AV block, and various atrial conduction defects are particularly common in inferior wall MI. Atrial flutter is rare during reperfusion and it is reverted to sinus rhythm with intravenous (IV)calcium gluconate.

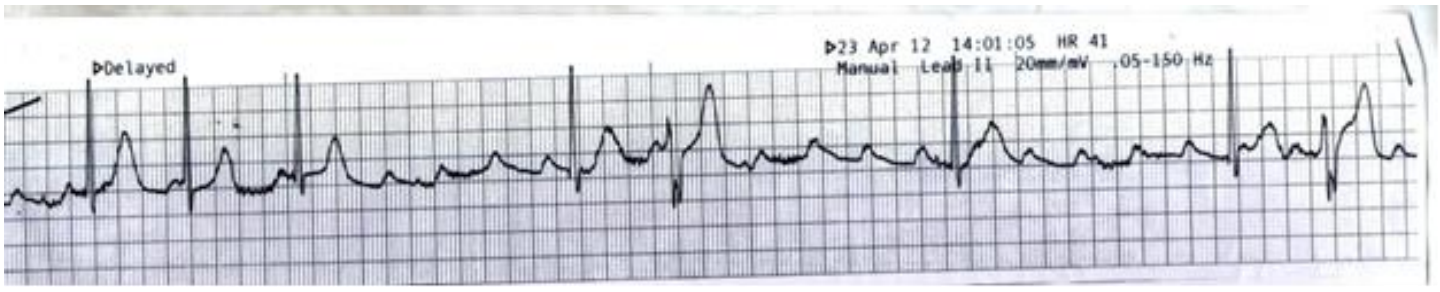
Keywords: elephantiasis, acute inferior wall mi; thrombolysis; atrial flutter; calcium gluconate

Introduction

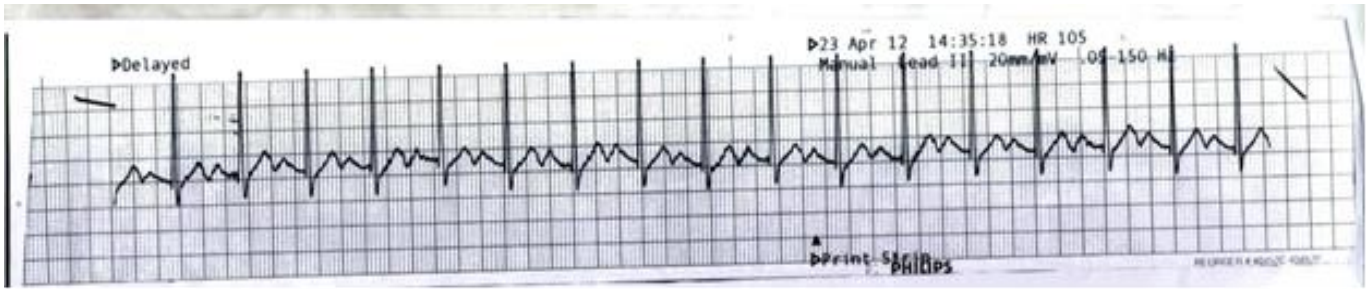
Early reperfusion therapy improves blood flow to the highly arrhythmogenic "penumbra" foci of ischemic myocardium surrounding the infarct area. The presence of atrial conduction defects in MI patients increases morbidity and mortality due to higher oxygen demand and reduced time available for ventricular filling and coronary perfusion [1]. Research and data on atrial flutter in relation to AMI are limited and there are limited data on pharmacological cardioversion of atrial flutter [2].

Case Report

65 years old, a non diabetic, non hypertensive male having elephantiasis of both legs was admitted with sudden onset of chest discomfort of 4 hours duration. He was having Acute Inferior wall Infarction with positive Troponin and elevated CKMB levels and other blood chemistry parameters including Thyroid function tests, serum electrolytes and calcium level were normal. X- ray chest not revealed any COPD features and cardiac contour is normal. Echocardiography revealed hypokinesia of LV posterior wall with moderate LV dysfunction. He was thrombolysed with streptokinase. During Thrombolysis, he developed sudden hemodynamic collapse and the manual Lead II in the defibrillator monitor is shown in ECG 1 as given below and it is reverted into sinus rhythm with Intravenous Calcium Gluconate as shown in ECG (II) and hemodynamically stable. Patient is symptom free, no further recurrances of arrhythmia, LV function improved and discharged from the hospital with further follow up advice accordingly.



ECG I: Rhythm strip in Manual Lead II



ECG II: (Reverted to sinus rhythm)

Etiopathogenesis

In MI, a rise in the extracellular concentration of potassium ions in the myocardial Rnecrotic area contributes to the formation of arrhythmogenic foci [3]. in IWMI (inferior wall myocardial infarction), direct dysfunction of the sinus and AV nodes, leading to improper impulse formation or defective impulse transmission. Improper impulse formation, combined with sympathetic over-stimulation of the atria, can lead to atrial flutter, and simultaneous defective impulse transmission results in variable block [4].

Discussion

Atrial Flutter was first identified as an independent medical condition in 1920 by the British physician Sir Thomas Lewis and colleagues (1881 – 1945). It is caused by a reentrant rhythm and typically initiated by a premature electrical impulse arising in the atria. The impact and symptoms of atrial flutter depend on the heart rate of the patient which is a measure of ventricular rather than atrial activity.

Sudden cardiac death may occur in atrial flutter in those individuals with a preexisting accessory conduction pathway bypassing the AV node with 1:1 conduction with a fast heart rate of 300 bpm [5] which may degenerate into ventricular fibrillation, causing hemodynamic collapse and death. Electrical Direct Current Cardioversion is life saving for this condition as it is more sensitive to a lower energy shock of 20-50 J and later Ablation therapy.

Conversely, atrial flutter is relatively resistant to chemical cardioversion and often deteriorate into atrial fibrillation prior to spontaneous return to sinus rhythm. Pharmacological cardioversion may be attempted if the patient has normal QT and no structural heart disease. Ibutilide is the preferred agent, amiodarone is less effective, class 1C agents and oral sotalol are relative poor efficacy and with significant side effects. 1C agents slow the atrial activity resulting 1:1 conduction and they should be used with AV nodal blocking agents such as beta blockers and calcium channel blockers and 1C drugs are contraindicated in patients with structural heart disease. Dronedarone is more toxic. Atrial overdrive pacing may result in atrial fibrillation. Chronic long term therapy is not required as in 60 % of cases, atrial flutter arises in the setting of a precipitating cause and once that acute process resolves, sinus rhythm is restored.

The ECG (I) reflects the upright "sawtooth " or " picket- fence" waveform pattern of P waves of slow downward slope followed by a fast upward slope as flutter waves described as " Type 1 clockwise atrial flutter" with an atrial rate of 150 bpm and a ventricular rate of 28 bpm with 5:1 AV conduction initiated by an atrial premature beat with aberrancy following the sinus rhythm and occurred during thrombolysis with streptokinase in Acute Inferior wall Infarction in a patient with Elephantiasis at the age of 65 years resulting in acute hemodynamic collapse and corrected with intravenous calcium gluconate.

Conclusion

This is the rarest Reperfusion arrhythmia encountered during Thrombolysis in Acute Myocardial Infarction and calcium Gluconate increases the ventricular contractility and restore sinus rhythm by overdrive suppression and thus, intravenous Calcium Gluconate play a role in atrial arrhythmias with minimal AV conduction with hemodynamic collapse.

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DOI:10.31579/2692-9759/179