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A Case of Ventricular Tachycardia Revealing Myocardial Infarction at Amirou Boubacar Diallo National Hospital in Niamey

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Abstract

Ventricular tachycardia (VT) is one of the most common arrhythmias during the acute phase of myocardial infarction (MI). It is a regular wide-QRS tachycardia with atrioventricular dissociation and often carries a guarded prognosis. Few studies in Niger have addressed arrhythmias in the acute phase of MI. We report a case of sustained VT revealing MI at Amirou Boubacar Diallo National Hospital (HNABD) in Niamey. **Case report**: A 63-year-old male patient with cardiovascular risk factors (age, male sex, poorly controlled diabetes mellitus on metformin for >10 years) was admitted to our department of internal medicine and cardiology for palpitations and exertional dyspnea. Clinical and complementary investigations established the diagnosis of MI complicated by sustained VT and an apical thrombus. After failure of properly conducted chemical cardioversion, the patient finally underwent electrical cardioversion with a 100-joule shock, restoring sinus rhythm. Immediate outcome was marked by hemodynamic and respiratory instability requiring intubation. The patient was urgently transferred to Morocco, where coronary angiography demonstrated occlusion of the left anterior descending artery (LAD), which was successfully stented with favorable outcome. **Conclusion**: Ventricular tachycardia is a severe complication of acute coronary syndrome (ACS) and is generally terminated by electrical cardioversion.

Keywords

Ventricular Tachycardia, External Electrical Cardioversion, Acute Coronary

Syndrome, HNABD

1. Introduction

Ventricular arrhythmias are common during the acute phase of ACS and are related to ischemia. The prognostic significance of an arrhythmia is not always correlated with the extent of MI or the occurrence of late arrhythmias. The reflex is always to look for hypokalemia, acidosis, hypomagnesemia, or hypoxemia [1]. Arrhythmic complications are frequently the presenting feature of MI; they most often consist of ventricular extrasystoles, which are extremely common during the first 2 days [2].

Primary ventricular tachycardia include non-ischemic dilated cardiomyopathy, adult and congenital structural heart disease, inherited cardiac channelopathies, infiltrative cardiomyopathy, electrolyte imbalances, illicit drugs such as cocaine or methamphetamine, and digitalis toxicity [3]. Infiltrative cardiomyopathy can result from systemic lupus erythematosus, sarcoidosis, amyloidosis, rheumatoid arthritis, and hemochromatosis [4]. In the presence of a substrate, ventricular tachycardia is triggered by multiple factors; the most common triggers include myocardial ischemia, hypokalemia, hypomagnesemia, hypocalcemia, sepsis, and metabolic acidosis.

Hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and myocarditis are responsible for ventricular tachycardia and cardiac arrest/sudden cardiac death. It is generally accepted that ventricular arrhythmias, including sustained ventricular tachycardia and fibrillation, occurring within 24 to 48 hours after the onset of myocardial infarction are not associated with a continued risk of cardiac arrest recurrence during subsequent follow-up. Thus, early arrhythmias in acute myocardial infarction are considered transient risk factors that do not predict future risks. However, recent studies have suggested that cardiac arrest during the early phase of acute myocardial infarction [5] or associated with other transient risk factors [6] may indeed indicate a high long-term risk when other variables are included in the risk profile. Whether this is contributed to by the combination of remodeling and individual susceptibility to the expression of cardiac arrest on a genetic basis or the evolution of post-myocardial infarction changes in vascular disease or recurrent ischemia over time remains unresolved. Clinical studies have not yet determined whether deterioration in ejection fraction (weeks or months after the event) alters the benign risk prediction associated with ventricular tachyarrhythmia during the acute phase of myocardial infarction [7] [8].

Ventricular tachycardia (VT): sustained, regular, wide-QRS rhythm with a ventricular rate >130 bpm.

Ventricular fibrillation, responsible for cardiac arrest with apparent death while the EKG shows chaotic, rapid QRS activity [9] [10].

For non-sustained VT (<30 seconds):

If hemodynamically stable: beta-blockers are preferred.

If pulmonary edema or cardiogenic shock: amiodarone or sotalol.

For sustained VT (>30 seconds) or ventricular fibrillation that is poorly tolerated, external electrical cardioversion is required; however, first-line therapy consists of amiodarone 300 mg IV over 30 minutes followed by continuous infusion over 24 hours.

Accelerated idioventricular rhythm (AIVR) is a form of slow VT with a heart rate of 80 - 120 bpm (wide QRS with atrioventricular dissociation) that occurs immediately after coronary reperfusion (angioplasty or thrombolysis) and is a marker of residual myocardial viability and good prognosis. No treatment is required unless the MI is extensive or AIVR is poorly tolerated; termination is achieved by accelerating the atrial rate with atropine, isoprenaline, or temporary pacing [11] [12].

Few studies in Niger have examined arrhythmias following MI. We report a case of MI revealed by sustained VT in the cardiology department of Amirou Boubacar Diallo National Hospital in Niamey.

2. Case Report

Mr A. ALM, a 63-year-old man with cardiovascular risk factors (age, male sex, poorly controlled diabetes mellitus on metformin for 10 years before), was admitted for palpitations and exertional dyspnea.

Symptoms began approximately one week earlier with sudden oppressive retrosternal chest pain of variable intensity and duration, radiating to the left shoulder and worsened by exertion. The patient self-medicated without relief.

Forty-eight hours before admission, dyspnea and palpitations worsened, prompting consultation at a local health facility where he received intravenous fluids and was sent home. Persistent symptoms led to referral to our department.

2.1. Physical Examination on Admission

- Conscious, Glasgow Coma Scale 15/15, WHO performance status grade III.
- Blood pressure 97/84 mmHg (left arm) and 100/69 mmHg (right arm); heart rate 186 bpm.
- SpO_2 99% on room air; respiratory rate 20 cycles/min.
- Weight 90 kg; height 1.76 m; BMI 29 kg/m^2; waist circumference 110 cm.
- Cardiovascular examination: rapid regular heart sounds with left-sided S3 gallop.
- Pulmonary examination: bilateral basal crackles.
- The remainder of the physical examination was normal.

An emergency EKG showed monomorphic ventricular tachycardia (**Figure 1**). Emergency echocardiography revealed extensive akinesia (apical, anteroapical, septoapical, and anterolateral) with a large apical thrombus and LVEF 30% (**Figure 2**).

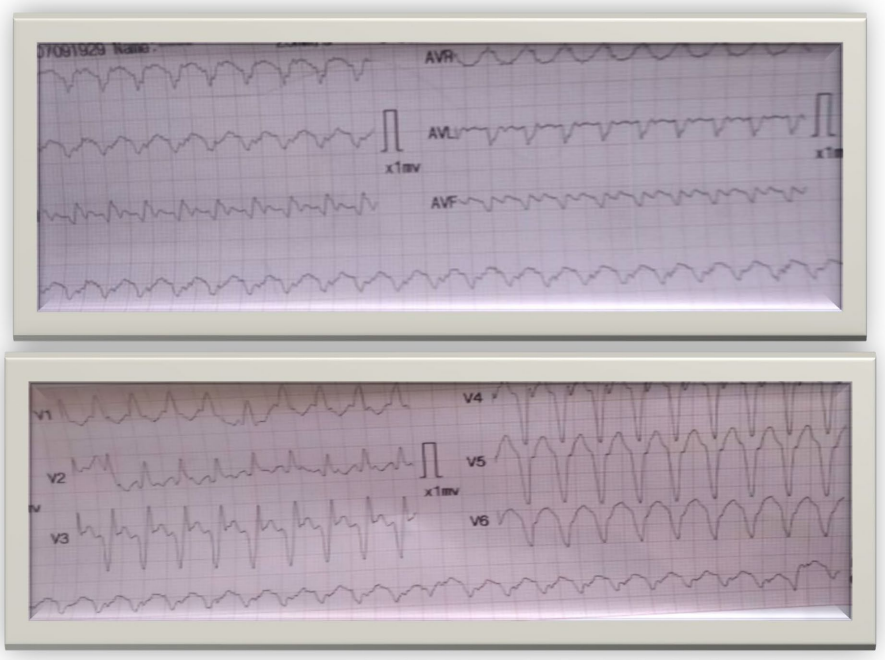

Figure 1. EKG showing monomorphic ventricular tachycardia.

Figure 2. Echocardiographic image showing an apical thrombus.

2.2. Emergency Management

- Continuous cardiac monitoring (**Figure 3**).

Figure 3. Cardiac monitor showing ventricular tachycardia.

- Amiodarone: 300 mg IV bolus over 30 min, then 600 mg via syringe pump over 24 h, followed by oral 200 mg/day.
- Enoxaparin 0.9 mL twice daily.
- Acenocoumarol 4 mg daily.
- Insulin (Actrapid) 4 IU/h via syringe pump for hyperglycemia (18 mmol/L).
- Atorvastatin 80 mg loading dose, then 40 mg daily.
- Pantoprazole and magnesium sulfate.

2.3. Laboratory Results

- WBC 7.98 × 10^9/L, hemoglobin 11.8 g/dL, platelets 285 × 10^9/L.
- Urea 3.88 mmol/L.
- Creatinine 82.53 µmol/L (eGFR 101.21 mL/min, MDRD formula).
- Sodium 139.9 mmol/L, potassium 5.57 mmol/L.
- Random glucose 18.57 mmol/L.
- CRP 48 mg/L; PT 78%; INR 1.15.

2.4. Immediate Outcome

VT persisted for 24 hours despite loading and maintenance amiodarone. Lidocaine was also tried without success. Because of persistent VT despite optimal medical therapy, a 100-joule electrical shock was delivered in collaboration with the intensive care team despite the risk of stroke from the apical thrombus (**Figure 4**).

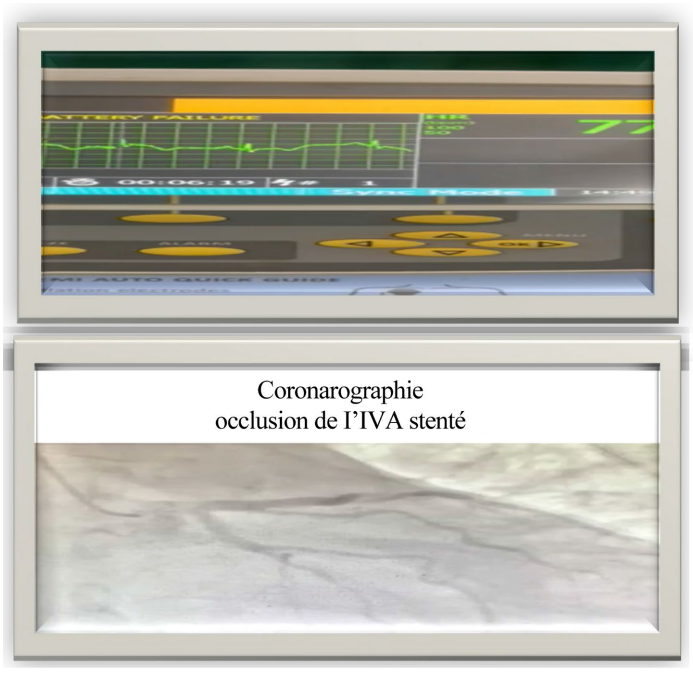

Figure 4. Return to sinus rhythm after a 100-joule shock.

Sinus rhythm was restored, but sudden hemodynamic and respiratory instabil-

ity ensued.

The patient was intubated, mechanically ventilated, and placed on vasopressors with satisfactory stabilization by day 3.

Coronary angiography performed in Morocco revealed LAD occlusion, which was successfully stented.

3. Discussion

Ventricular is a frequent arrhythmia following ACS and is a marker of severity requiring urgent management. It originates below the His bundle bifurcation and is defined as upper three consecutive wide-QRS complexes at >100 bpm lasting > 30 seconds (sustained). Ischemic heart disease and MI account for 75% of cases of VT. VT occurring within the first 48 hours of MI generally has no adverse long-term prognostic impact, particularly in limited infarcts, whereas VT occurring after 48 hours in extensive MI with LVEF < 35% carries a high risk of recurrent fatal VT [2]. Our patient had typical anginal pain for one week without seeking care until arrhythmic and thromboembolic complications developed. Ventricular tachycardia is a major contributor to sudden cardiac death in patients with ischemic and non-ischemic cardiomyopathy. Ventricular tachycardia in cardiomyopathy is usually monomorphic due to scar-related reentry, and its degeneration into ventricular fibrillation may result in cardiac arrest or even sudden cardiac death [13]-[15]. The clinical presentation of ventricular tachycardia varies from palpitation to sudden cardiac death. For appropriate management of VT and prevention of sudden cardiac death, it is essential to understand the pathophysiology of ventricular tachycardia and underlying structural heart disease.

Early monomorphic VT increases mortality. In the series by Demidova *et al.* [3] of 2277 patients with STEMI, early monomorphic VT (1.5%) was associated with higher mortality than non-monomorphic VT/VF (37% vs. 19%; $p = 0.011$). Our patient had monomorphic VT persisting ≈ 24 hours and resistant to chemical cardioversion, leading to hemodynamic and respiratory deterioration. Our patient first symptom is palpitation and secondly cheats pain.

Several publications report similar findings. Yameogo *et al.* [4] found ventricular arrhythmias complicating MI in 9.3% of cases. Liliane *et al.* [5] in Douala reported one case of sustained VT among 11 MI patients that progressed to unrecovered cardiac arrest.

MI in our patient was complicated by an apical thrombus, delaying electrical cardioversion because of embolic risk. Thrombus formation follows Virchow's triad (endothelial injury, stasis, hypercoagulability) [6]. Extensive apical akinesia and reduced LVEF contributed to thrombus formation in our patient. Elevated CRP is significantly associated with apical thrombus [7]; our patient had CRP 48 mg/L.

Guidelines recommend electrical cardioversion as the treatment of choice for poorly tolerated VT [8]. Despite cardiovascular collapse, the presence of apical thrombus initially led us to attempt chemical cardioversion with amiodarone (300

mg IV over 30 min followed by continuous infusion). A cardioselective beta-blocker (bisoprolol) was added because of severely reduced LVEF. Lidocaine (1.5 mg/kg IV bolus followed by 20 mg/kg/24 h infusion) was also unsuccessful. Ultimately, a 100-joule shock restored sinus rhythm, followed by hemodynamic/respiratory instability requiring intubation. Coronary angiography in Morocco showed LAD stenosis, which was successfully stented.

ICD implantation is recommended (Class I, Level A) [13] to reduce sudden death and all-cause mortality after hemodynamically unstable sustained VT in patients with life expectancy upper one year and good functional status. Secondary-prevention ICD and cardiac rehabilitation were not performed owing to lack of patient consent. All patients with structural heart disease and left ventricular systolic dysfunction should be offered guidelines-directed medical therapy for heart failure [17]. Patients with ischemic cardiomyopathy who survive sudden cardiac arrest due to ventricular tachycardia, or expérience hemodynamically unstable or stable sustained ventricular tachycardia, should have an implantable cardiac defibrillator (ICD) placed if their estimated meaningful survival is greater than one year [18]-[20]. Patients with unexplained syncope who have ischemic cardiomyopathy, non-ischemic cardiomyopathy, or adult congenital heart disease who do not meet the criteria for an ICD can undergo an electrophysiological study to assess the risk of sustained ventricular tachycardia, however, performing the study solely for risk stratification is not indicated [16] [21] [22]. If a sustained VT is induced during an electrophysiology study, implantation of an ICD should be recommended for the prevention of sudden cardiac death.

The patient currently has stable chronic heart failure with LVEF 45% and resolution of the thrombus. Current treatment includes antiarrhythmic agents, ACE inhibitors, beta-blockers, statins, aldosterone antagonists, metformin, and aspirin.

4. Conclusions

Ventricular tachycardia is one of the most common arrhythmias in myocardial infarction and worsens prognosis. Electrical cardioversion is the most effective means of termination. Cardiac rhythm monitoring by scope or telemetry is essential in the acute phase of MI given the risk of fatal rhythm disturbance.

In the chronic phase, the occurrence of a ventricular arrhythmia and/or the persistence of LV dysfunction despite optimal medical treatment should prompt discussion of ICD implantation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Abbreviations

MI	Myocardial infarction
VT	Ventricular tachycardia
VF	Ventricular fibrillation
ICD	Implantable cardioverter defibrillator
LV	Left ventricle
LAD	Anterior descending artery
ECG	Electrocardiogram
EF	Ejection fraction
HNABD	Amirou Boubacar Diallo National Hospital