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Challenge of Managing the Cocktail of Cardiac Arrhythmias Induced by Cocaine and Cannabis Poly-Drug Addiction: A Case Report.

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ABSTRACT: Consumers of illicit toxicants combine toxicants in order to potentiate and prolong the duration of the effects. This results in an increase in acute or chronic toxicity, in particular cardiac toxicity, associating a variety of arrhythmias ranging from benign sinus tachycardia to the most complex ventricular arrhythmias. Heart risks are higher when people are young. We report the case of a 48 year old male chronic user of illicit substances, including cocaine, cannabis, amphetamines as well as alcohol; and who presents a wide variety of ventricular arrhythmia and conduction disorders associated with dilated cardiomyopathy (DCM). We describe through a review of the literature the physiopathological mechanisms involved in the genesis of rhythmic disorders and myocardial damage secondary to toxicity; before considering the management challenge of arrhythmias induced by multiple drug addiction

KEYWORDS: Poly-drug addiction, cocaine, cannabis, heart Arrhythmia, dilated cardiomyopathy, cardiotoxicity.

INTRODUCTION

Poly-drug addiction calls for more and more varied products, presenting themselves in many ways, and whose mode of administration knows no limits. Two illicit drugs constantly increasing dominates: cannabis and cocaine. Associated with alcohol and new drugs, they have high cardiovascular toxicity. Their associations generate clinical pictures complex, which physicians face. We report the case of a 48-year-old man, poly-drug addict, presenting rhythmic ventricular and myocardial complications, including complexity and polymorphism has led to cardiac resynchronization therapy (CRT).

Patient(S) /Observation(S)

M.H, 48 years old, is admitted for palpitations with a sensation of death and atypical chest pain, evolving for 1 month. Interrogation finds active smoking massive chronic and multiple drug addiction for 10 years, including cocaine, Cannabis, amphetamines, diazepam, and regular alcohol consumption. Moreover, the patient reports a history of non-ischemic dilated cardiomyopathy known for 4 years, of which he would have on his own, stopped the treatment 1 year ago. The clinical examination found normal blood pressure = 110 /80mmhg, a heart rate baseline at 45 / min, with a slight sign of right heart failure, and a patient overall sober.

12-lead ECG on admission revealed (figure 1A/1B): ventricular tachycardia (VT) with right-delayed types, originating from the upper ventricular wall; interspersed with high degree conductive disorders such as high degree atrioventricular block (AV) associated with polymorphic ventricular premature beat (VPB) with wide QRS.

The assessment after stabilization of the patient including:

A 24-hour Holter ECG (figure 2A/2B/): highlights a cocktail of conductive and rhythmic disorders associating complete AV block, Mobitz I degree, polymorphic VPB with QRS duration at 135ms, several episodes of polymorphic VT, as well as a long QT interval to 554ms on average.

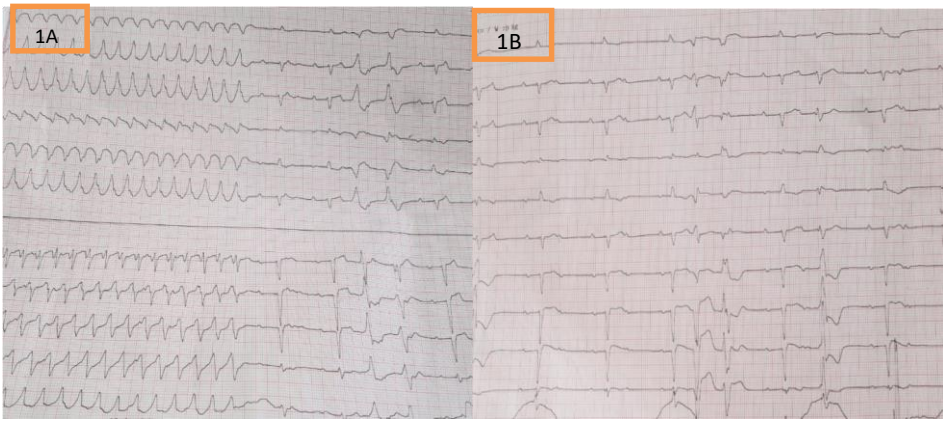

Figure 1A-1B: 12-lead ECG recording ventricular tachycardia (VT) withhigh degree conductive disorders

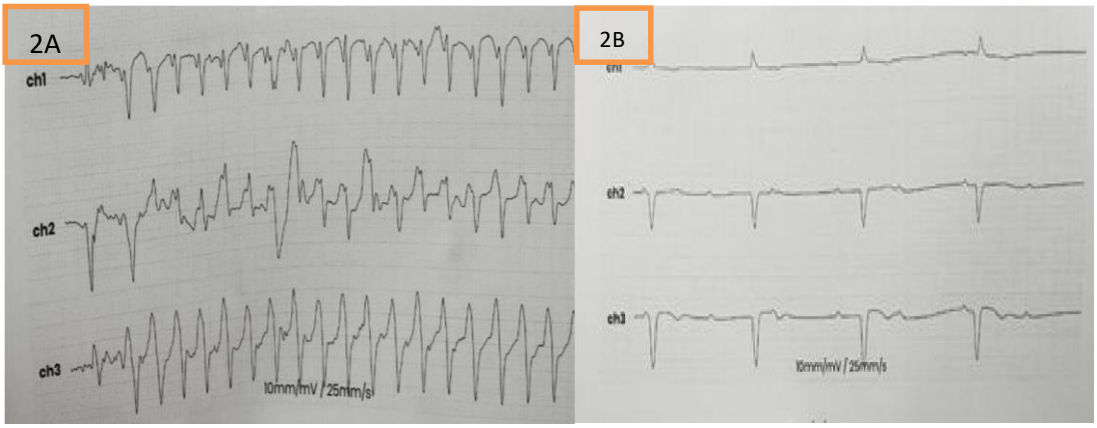

Figure 2A-2B:A 24-hour Holter ECG recording: conductive and rhythmic disorders

Transthoracic ultrasound shows dilated cardiomyopathy (DCM) with biventricular dysfunction (LV end-systolic diameter: 35mm/m^2; LV end-diastolic diameter: 23mm/m^2) with normal ventricular thicknesses, global hypokinesia, for LVEF at 20%; bi-atrial dilation (area left atrium: 36cm^2; area right atrium: 24cm^2) with mild tricuspid and mitral regurgitation, and PAPS estimated at 46mmHg.

Cardiac MRI finds the presence of diffuse extensive fibrosis in the septum, anterior and lower walls (Figures 3A and 3B).

The biological assessment found no metabolic, endocrine, or serological abnormalities that could explain the clinical condition.

The therapeutic approach to this dilated cardiomyopathy of toxic origin complicated by high-risk arrhythmic and conductive disorders, including medical treatment of heart failure excluding β-blockers, antiarrhythmic. We gave magnesium and potassium supplements to prevent hypokalemia and hypomagnesemia. A CRT-D system was performed after stabilization (figure 4).

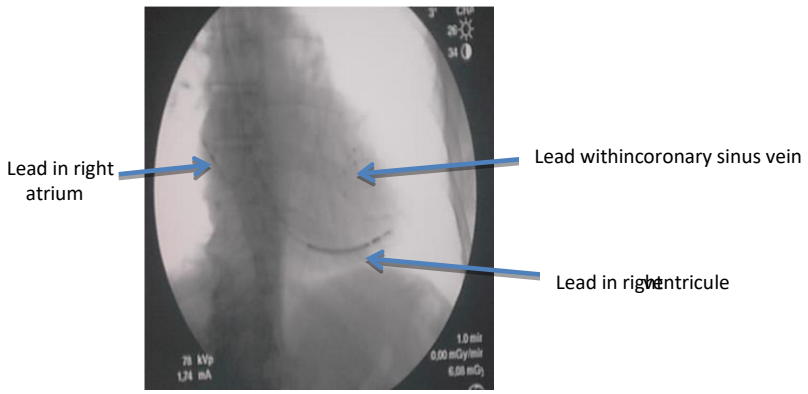

Figure 4: An implanted CRT-D system.

DISCUSSION

The use of illicit drugs, including cocaine and cannabis, is common in many parts of the world, especially the United States and Western Europe. Toxicity from the use of these drugs is associated with a wide range of clinical consequences, especially since illicit drug users are poly-drug addicts. The alcohol dependence of drug addicts potentiates the euphoric effect and craving, worsening cardiovascular effects.

Cardiotoxicity of illicit drug use includes acute coronary syndrome (ACS), myocardial infarction (MI), cardiomyopathy, hypertension, events cerebrovascular (ischemic and or hemorrhagic) and arrhythmias. The effects cardiotoxic drugs from cocaine are indirectly mediated by an increase in sympathetic cardiac stimulation and coronary vasoconstriction and by a direct effect on ion channels responsible for rhythm disturbances, even in the absence of any myocardium ischemia [1]. Indeed, cocaine inhibits the currents of calcium (Ca), the currents of potassium (K) of delayed recovery and sodium (Na) currents of cardiomyocytes [1]. These changes electrophysiological effects are generally attributed to the direct effects of cocaine on the cardiac ions and are responsible for arrhythmias. The powerful inhibition of Na currents cardiac, prolongs the refractory period, which induces a slowing down of conduction myocardial characterized by prolongation of PR, QRS and QT [1-2].

Moreover Coca- ethylene, a metabolite of cocaine and ethanol, slow down cardiac conduction, delay repolarization and are a potent inhibitor of potassium channels [1]. This alchemy of rhythmic and conductive disorders increases the risk of complex cardiac events.

Cannabis and, in particular, its active principle D9-tetrahydrocannabiol (THC) causes a variety of tachy- and bradyarrhythmias on the cardiovascular system, causing sudden death [3]. Low to moderate doses of cannabinoids induce sympathetic stimulation with a decrease in the duration of the action potential, the atrial refractory period and a modification of the myocardial electrophysiological properties, promoting automaticity and micro-reentry. Higher doses stimulate the parasympathetic, with an increase in the duration of the action potential and the refractory period [3]. The association between sympathetic hyperactivity and increased vagal stimulation leads to catecholergic atrial and ventricular hyperexcitability responsible for acute atrial fibrillation or ventricular fibrillation, but also bradyarrhythmias, including sinus arrest, atrioventricular block [34].

The existence of myocarditis and dilated cardiomyopathy is widely reported in the literature among cocaine users. These mechanisms would involve a disorder of cytokine production as well as a modification of myocardial collagen and induction of myocyte apoptosis responsible for the destruction of myofibrils and interstitial fibrosis's [5-6]. Dilation of the left ventricle is the second anatomical finding in an autopsy series of sudden deaths [6]. Myocardial dysfunction usually occurs with long-term cocaine poisoning.

As for cannabis, it induces myocardial dysfunction especially through an infarction, favored by vasospasm associated with the thrombotic phenomenon [3-4].

Regarding amphetamines and ecstasy, these substances have indirect sympathetic activity related to the release of norepinephrine, dopamine and serotonin in the central nervous system and the autonomic nervous system système [7]. Chronic consumption of amphetamines leads to myocardial inflammation which can progress to genuine cardiomyopathy with cell infiltration, myocardial hypertrophy and fibrosis [7].

Due to activation of the sympathetic system, the use of these substances always results in a certain degree of tachycardia and vasoconstriction of the coronary arteries which reduces the supply of oxygenated blood to the myocardium, thus promoting heart attacks and cardiac arrhythmias. Similarly, oxidative stress characterized by an accelerated state of atherosclerosis, and platelet hyperaggregability, promotes coronary artery disease [2-6].

The treatment of cardiovascular manifestations secondary to multiple drug addiction is poorly codified. Not being able to incriminate a single toxicant in the mechanisms of complex arrhythmias, a measured and prudent approach which includes sedation, volume resuscitation and the correction of electrolyte abnormalities, is fundamental and is recommended in cases of acute and chronic poisoning.

While sodium bicarbonate is used for the treatment of cardiac sodium channel blockade induced by several agents including cocaine and heroin, it is questionable in cases of poisoning with amphetamines and ecstasy since it appears to decrease their urinary excretion [8].

Complex tachyarrhythmias usually respond to hypertonic sodium bicarbonate and QT prolongation is usually treated with magnesium [8-9]. Lidocaine appears safe for reentrant ventricular rhythms, especially in patients pretreated with benzodiazepines; but is controversial due to the potential for proconvulsant effects of lidocaine and cocaine [10]. Calcium channel blockers are not indicated as the first-line treatment for arrhythmias, unless indicated, for resistant coronary vasospasm and associated myocardial ischemia. In all cases, the use of β-adrenergic antagonists and IA and IC class antiarrhythmics are contraindicated, as is the use of mixed α and β-adrenergic antagonists [8-10].

In patients at very high risk of sudden death from arrhythmia, biventricular pacing has provided significant clinical benefit, compared to right ventricular pacing, in severe left ventricular dysfunction and atrioventricular block requiring cardiac pacing [10-11]. In any case, referring patients to drug treatment programs is essential to prevent recurring events.

CONCLUSION

Complex and synergistic mechanisms underlie the generation of arrhythmic cocktail in multiple drug addicts. These electrolyte disturbances result from the direct effects of these substances and their metabolites on the Na, K and Ca channels, as well as on the impairment of the coronary circulation (accelerated atherosclerosis, vasospasm, endothelial dysfunction and platelet hyper aggregation) predisposing individuals healthy with ventricular arrhythmias and sudden death. Likewise, the direct toxic effects are responsible for myocardial inflammation, destruction of myofibrils, interstitial fibrosis, which may progress to genuine hypertrophic or dilated cardiomyopathy with severe ventricular dysfunction. The simultaneous use of cannabis, cocaine, alcohol and other toxic substances potentiates the effects of cardiac toxicity. The therapeutic approach to the cardiovascular effects of poly-drug addiction must be careful and measured; βadrenergic antagonists, IA and IC class antiarrhythmics, as well as mixed α and βadrenergic antagonists, are contraindicated. Patients at very high risk of sudden death from ventricular arrhythmia with high degree ventricular dysfunction and conductive block, ventricular pacing may be considere.

Declaration of Competing Interest

The authors declare that they have no competing interests.

Authors' Contributions

Karimou Bondabou Abdoul wahab: principal investigator, Contributions to the acquisition, analysis, clinical data and imaging; the critical review of intellectual content; Final approval of the version to be published

Amadou Daouda: principal investigator; acquisition, analysis, clinical data and imaging; the critical review of intellectual content; Final approval of the version to be published

Sayarh Salma: associate investigator, Contributions to the acquisition, analysis, clinical data and imaging; the critical review of intellectual content; Final approval of the version to be published

Bouzouba Otman: Contributions to the acquisition, analysis, data imaging; the critical review of intellectual content; Final approval of the version to be published

Nghomo Yves-Noel: Contributions to the acquisition, analysis, data imaging; the critical review of intellectual content; Final approval of the version to be published

Doghmi Nawal: Contributions to the acquisition, analysis, clinical data and imaging;

Final approval of the version to be published

Fellat Ibtissam: Contributions to the acquisition, analysis, clinical data and imaging; the critical review of intellectual content; Final approval of the version to be published; responsible for all aspects relating to the accuracy or integrity of work.

Cherti Mohamed: Contributions to the acquisition, analysis, clinical data and imaging; the critical review of intellectual content; Final approval of the version to be published; responsible for all aspects relating to the accuracy or integrity of work.

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