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Idiopathic Refractory Ventricular Fibrillation as a Cause of Cardiorespiratory Arrest (PCR) That Required Cardiopulmonary Resuscitation (RCP) Through Percutaneous Veno-Arterial Extracorporeal Membrane Oxygenation (ECMO). Could Caffeine be the Cause?

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INTRODUCTION

The incidence of refractory Ventricular Fibrillation (VF) in our environment is almost 25% of all Cardiopulmonary Arrests (CPR) and presents a high mortality rate [1]. It has been described that VF and pulseless Ventricular Tachycardia (VT) as the initial rhythm cause greater instability in patients with CPR, and special attention should be paid to the transient recovery of spontaneous circulation as a potential sign of reversibility [2]. A wide variety of structural abnormalities is associated with CPR. However, there is no evidence of structural heart disease in approximately 5% of sudden death victims, indicating that cardiac arrest in the absence of organic heart disease is more common than previously thought. The risk of recurrence and the long-term response to treatment are important questions, but unanswered. The data from the small series published so far have limited value due to the lack of uniform criteria to define and diagnose Idiopathic Ventricular Fibrillation (IVF) [3].

In recent years, the implementation of percutaneous veno-arterial Extracorporeal Membrane Oxygenation (ECMO) in patients with out-of-hospital cardiac arrest has been developed in our center. This has allowed the stabilization and diagnostic study of this type of patients in the initial moments of the CPR. This has involved a great effort and coordination with the out-of-hospital emergency services.

CASE PRESENTATION

We present the case of a 36-year-old man, with no drug allergies or relevant medical history except for chronic consumption of four liters of Coca Cola. In addition, he takes tadalafil. The patient, while exercising, experiences

sudden Cardiopulmonary Arrest (CPR). Witnesses initiate basic CPR for 10 minutes. Upon the attention of emergency services, the patient is found pulseless with VF being observed. He requires a total of 10 defibrillations (3 of them consecutive) until the recovery of spontaneous circulation. The patient requires orotracheal intubation and during his transfer to our hospital high doses of noradrenaline due to hemodynamic instability. Upon arrival at our center, he presents electrical instability after CPR with hemodynamic repercussions. Given the need for catheterization in order to rule out ischemic heart disease, it is decided to implant percutaneous VA ECMO femoro-femoral, guided by transthoracic echocardiogram and without incidents. A 15 French (Fr) arterial cannula and 23 Fr venous cannula are introduced. In the initial diagnostic study, hypokalemia (2.8 mmol/L) was observed, an initial electrocardiogram with a QTc of 503 msec as relevant findings without other alterations. The catheterization and echocardiogram did not present alterations. Moreover, absence of toxins in urine. The patient presented a favorable evolution allowing the ECMO to be removed after three days and subsequent extubation. Cardiotropic viruses were negative. Cardiac magnetic resonance imaging showing a moderately dilated left ventricle and negative genetic tests were requested. The stress ergometry ruled out catecholaminergic cardiomyopathy being diagnosed with Idiopathic Ventricular Fibrillation (FVI) (Figures 1,2).

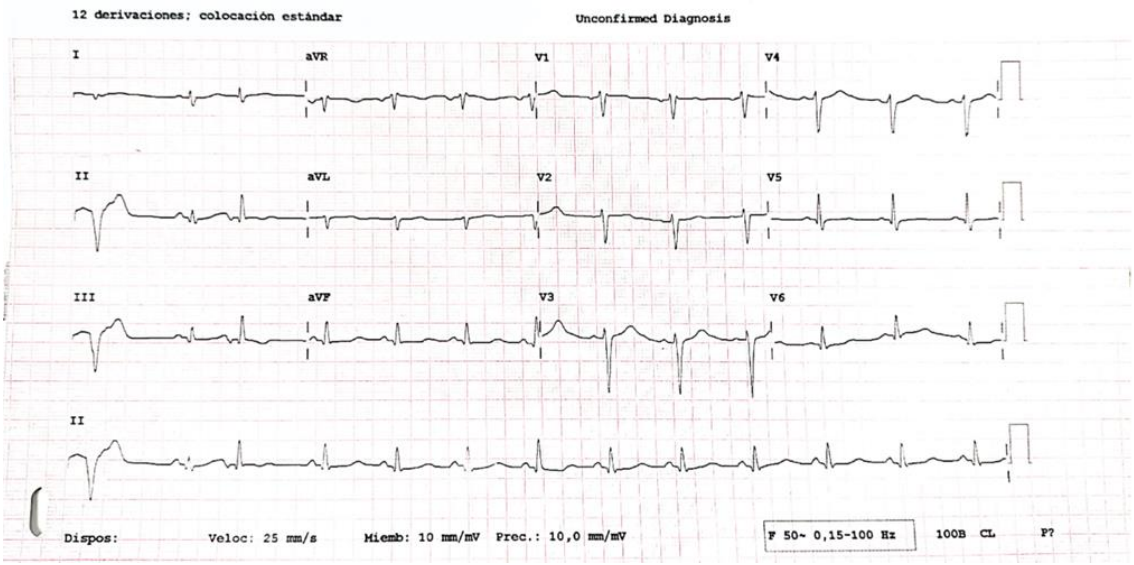

Figure 1: Patient's electrocardiogram once stabilized. The main finding is QT prolongation.

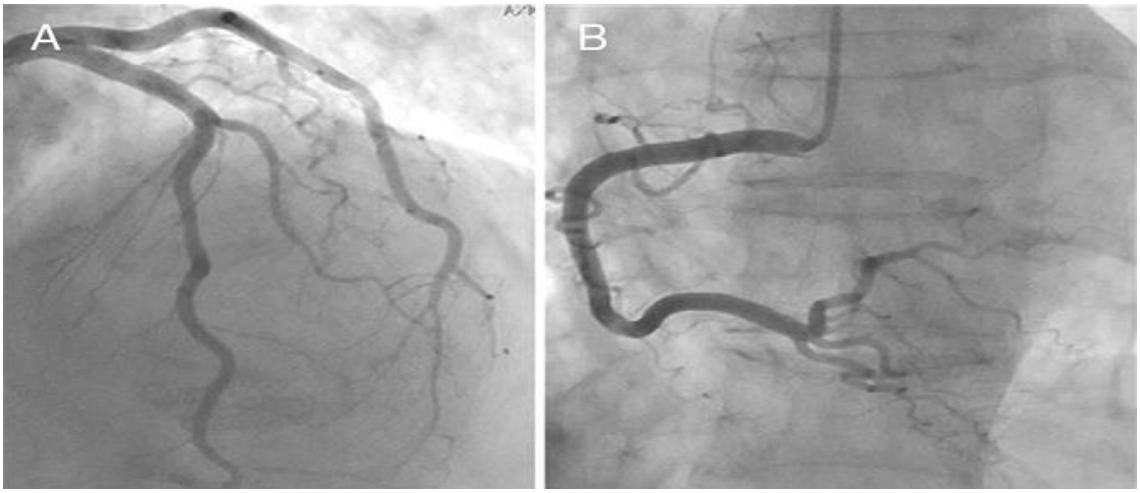

Figure 2: Catheterization of the patient upon admission where no alterations are observed.

DISCUSSION

Given the results of the diagnostic tests, our patient was diagnosed with idiopathic VF. The different causes of ventricular fibrillation were reasonably ruled out; drug abuse was ruled out with initial toxic tests in urine and detailed anamnesis of witnesses and emergency services, ischemic heart disease was ruled out by catheterization, coronary anomalies, structural cardiomyopathy and arrhythmogenic right ventricular dysplasia were ruled out by magnetic resonance imaging, myocarditis by serologies of negative cardiotropic viruses and rest of mentioned imaging tests, preexcitation phenomena and Brugada syndrome by electrocardiogram and finally congenital long QT syndrome by genetic tests.

As a probable causal agent, we observed the abusive consumption of Coca-Cola. Retrospectively, we found that the initial blood analysis showed hyperglycemia (285mg/ml). There are several articles and case descriptions in which it is described as the cause of fatal arrhythmias [4,5]. Coca-Cola generates hypokalemia due to osmotic polyuria, osmotic diarrhea, and caffeine itself. In turn, caffeine is an agonist of ryanodine receptors that stimulates intracellular calcium flows and can generate arrhythmias. In addition, it inhibits the NOS enzyme, which causes tachycardia and hypokalemia by inhibiting the Na/k pump of skeletal muscle. However, the doses needed to produce arrhythmias from caffeine are very high, requiring approximately 150 mg/kg of caffeine. On the day of admission, our patient had consumed 10 liters of Coca-Cola, which constitutes around 1222 mg of caffeine. A dose far from the theoretically necessary to produce fatal arrhythmias that in our patient is around 12000 mg (the patient's weight is 80 kg). However, afterwards, it was observed that tadalafil (a drug that the patient regularly consumes) is a potent inhibitor of CYP1A2 that metabolizes caffeine, so theoretically sufficient levels could have been reached. Unfortunately, we do not have caffeine levels of the patient.

Finally, hypokalemia is found as a probable trigger (our work team concluded it is unlikely to attribute the cause of the PCR to a potassium of 2.8 mmol/L) and the presence of undetected mutations in genetic tests cannot be ruled out. We must consider that congenital long QT syndrome (LQTS) presents great genetic heterogeneity and more

than 500 mutations have been identified so far in 10 genes (*KCNQ1*, *HERG*, *SCN5A*, *KCNE1*, *KCNE2*, *ANKB*, *KCNJ2*, *CACNA1*, *CAV3* and *SCN4B*). Despite advances in the field, 25% to 30% of patients remain without a genetic diagnosis [6,7].

CONCLUSION

Refractory VF is a significant cause of CPR and sometimes a diagnostic challenge. ECMO is a fundamental tool for the hemodynamic stabilization of patients that is being implemented in more and more hospitals every day.

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