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Diagnosis and Treatment of Brugada Syndrome: Review of the Literature

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ABSTRACT

Brugada syndrome is a genetic disorder that causes abnormalities in the correct functioning of the electrical system of the heart, which can end in health complications such as arrythmias, syncope or, in the most severe cases, sudden death. Nowadays, stratification of the risks derived from this disease is still a great challenge due to the disorder's complex nature influenced by several factors. Such investigations have been able to obtain evidence of the role that autonomic imbalance plays in the pathology of the disorder.

Subsequently, a Matlab code inspired by pre-existing algorithms was written, carrying out research on the methods currently used to detect this syndrome, in order to study the different biomarkers obtained through a simple and non-invasive procedure such as the electrocardiogram(EKG). After the study, the length of the QRS complex and the T wave, the absolute area , the RMS amplitude, as well as the length of the QT and QTc segments of each of the patients´ EKGs were measured.

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INTRODUCTION

Brugada syndrome is a genetic (inherited) disease that causes sudden death mainly in young people, between 35 and 40 years old. It was first described in 1992 by Pedro and Josep Brugada in eight patients (Arbelo and Brugada, 2014). What was initially treated as a medical curiosity quickly became a recognized clinical entity due to the great affectation observed, especially in certain Asian areas, such as the Philippines. It is currently associated with sudden cardiac death, mainly in men, in 1 in 10,000 people in the Western world, and 20% of sudden deaths in people with a healthy heart are attributed to it. Brugada syndrome involves an alteration in the ion channels that control the electrical activity of the heart. These alterations, converted into total failure of the channels, cause cardiac arrhythmias and even sudden cardiac death. Since its identification in 1992 to date, hundreds of patients with Brugada syndrome have been diagnosed, a genetic basis has been established and up to 20 genes associated with this disease have been identified.[1,2]

DIAGNOSIS OF BRUGADA SYNDROME

The diagnosis of Brugada syndrome can be obtained through various mechanisms. When the patient has a family history of sudden cardiac deaths or arrhythmias, a genetic test can be performed that will identify a carrier of this syndrome or a person who is at risk of suffering from it. An electrophysiological study can also be performed, which will cause an arrhythmia in the patient suffering from Brugada syndrome. However the diagnosis of Brugada syndrome is mainly based on the 12-lead electrocardiogram where a pattern known as "shark fin" is identified. A patient affected by this disease will show one or several of the following types of electrocardiographic patterns: [3]

Type 1: It is the only type of diagnostic ECG pattern. It is characterized by a concave elevation in the ST segment, or J point, greater than 2mm, followed by a negative T wave in at least one of the right precordial leads. [4]

Type 2: Characterized by a J-point elevation of more than 2mm followed by a descent of approximately 1mm and a positive T wave. This pattern is known as "saddle".[4]

Type 3: It is similar to the type 2 pattern with the exception that, in this case, the elevation of the J point is less than 1mm. [4]

Both type 2 and type 3 patterns are considered non-diagnostic and require a flecainide test. Flecainide is a drug that produces arrhythmias in patients with Brugada syndrome and causes type 2 and 3 patterns on an ECG to become type 1 during an arrhythmia provocation test. [5]

RISK STRATIFICATION IN ASYMPTOMATIC PATIENTS

Based on what is described in the 2015 clinical practice guideline of the European Society of Cardiology on sudden death and the 2013 Consensus on primary arrhythmic syndromes; asymptomatic patients with an induced Brugada I pattern should continue with clinical follow-up until the appearance of the spontaneous pattern is determined. An electrophysiological study should continue in those with a Brugada type I basal pattern. The induction of ventricular tachycardia or fibrillation is a criterion used for the selection of candidates for cardio-defibrillator implantation; however, there are divergences regarding its value due to the high variability in the degree of induction, stimulation protocol and its reproducibility. Thus, supported by smaller trials, the ventricular refractory period <200 ms and QRS fragmentation are outlined as new predictors of arrhythmic events. [6,7]

The consensus for "J wave syndromes" published in 2017, outlines in the protocol for the spontaneous asymptomatic Brugada type I pattern the possibility of electrophysiological study, and for that induced by sodium channel blockers, clinical follow-up. In case of inducing ventricular arrhythmia, cardio-defibrillator implantation should be considered. [8]

CLINICAL-PROCEDURAL FOLLOW-UP

The decision to implant a cardioverter defibrillator in asymptomatic patients continues to be a controversial challenge, due to the cost of the device, the risk inherent to the implant and the possibility of inappropriate shocks, which increase the rate of complications compared to the potential benefit of aborting sudden death. For those patients in whom long-term clinical follow-up is decided, the time and frequency of follow-up, control procedures and medical discharge have not been defined. [9]

TREATMENT OF BRUGADA SYNDROME

Once a patient has been diagnosed with Brugada syndrome, the most effective treatment currently available is the insertion of an ICD or implantable cardioverter-defibrillator, since there are few drugs that are effective against sudden death. ICDs are medical devices that detect arrhythmia in a patient and automatically deliver an electrical shock that will restore normal rhythm. In cases where a patient presents with symptoms such as sudden recovered death or the occurrence of syncope, the solution will be to implant an automatic defibrillator. [10]

There are cases in which patients are intolerant to these devices, are too young to wear them or are simply against implantation. In these cases, there is a drug known as quinidine that can be administered and can reduce some of the symptoms of Brugada syndrome; but it is not considered 100% effective, so it is a measure reserved for the previously mentioned exceptions. In recent years, work has been done on the development of a technique known as cardiac ablation, which consists of burning part of the heart tissue using an epicardial catheter. [11,12]

For patients with Brugada syndrome, ablation is used to burn off tissue in the right ventricular outflow tract, which is believed to be the site responsible for this disease. In patients who have undergone surgery, it has been observed that the characteristic pattern of Brugada syndrome disappears from the ECG and that neither flecainide nor the electrophysiological study causes arrhythmias. The combination of these events is very important, since it suggests that the risk of sudden death has practically disappeared. However, due to the novelty of this technique, it is still too early to confirm that it is a long-term cure and an evaluation must be carried out over a prolonged period. [13,4]

CONCLUSION

It should be taken into account that multiple factors such as gender, age, family history, type and penetrance of the mutation can influence the development of Brugada Syndrome and determine its symptoms.

Some potential inducers of Brugada Syndrome electrocardiographic patterns have been described, for example, febrile episodes caused by infections, cocaine intake, or drugs that block sodium channels, which in turn make it possible to unmask the disease.

The hidden or intermittent form of the syndrome makes timely diagnosis difficult and increases the risk of sudden death. It is known that at rest or during sleep a decrease in sympathetic activity can occur, which leads to an increase in parasympathetic tone, increasing the possibility of suffering an arrhythmia that could lead to the death of the patient.

In clinical practice, an in-depth study must be carried out on the risk that the patient has of developing LRAMC in order to be able to take preventive measures and thus avoid complications in the patient. There is enough information to be able to make evidence-based decisions when a patient with chronic kidney disease who requires a study with CM presents and to guarantee the quality of care.

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