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DETECTION AND CONTROL OF BRUGADA SYNDROME DEPENDING ON OCCUPATIONAL RISKS

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Abstract:

The ECG showed an electrocardiographic pattern in which Brugada type 2 syndrome was reported and the patient was sent to a cardiac arrhythmia treatment center, where flecainide confirmed the diagnosis. Electrophysiological examination showed a low arrhythmia profile, and the patient was advised to avoid the consumption of certain drugs and drug abuse in patients with Brugada syndrome. Brugada syndrome is distinguished by fainting or sudden death based on the electro-cardio graphic outline of ST segment advancement in the normal heart, but on the precordial paths from V1 to V3 and the right bundle division block. Polymorphic ventricular tachycardia is caused by syncope and sudden death. These electrocardiographic symbols may appear during a routine professional examination of an asymptomatic patient.

Keywords: *Detection, Control, Brugada Syndrome, Occupational Risks.*

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CASE REPORT:
A 37-year-old man with a history of syncope or syncope has joined a professional medical service for routine monitoring. The ECG showed an electrocardiographic pattern in which Brugada type 2 syndrome was reported and the patient was sent to a cardiac arrhythmia treatment center, where flecainide confirmed the diagnosis. Electrophysiological examination showed a low arrhythmia profile, and the patient was advised to avoid the consumption of certain drugs and drug abuse in patients with Brugada syndrome. The patient remains asymptomatic 6 months after diagnosis. Conclusions. Experts conducting scheduled tests can detect Brugada syndrome and prevent sudden death. It is important to understand the occupational risk of an employee who is diagnosed with Brugada syndrome to prevent or reduce the occurrence of ventricular arrhythmias.

INTRODUCTION:
The band Brugada (BS) was first described in 1992. It is distinguished by a typical electrocardiographic pattern and susceptibility to ventricular arrhythmias and sudden death in normal precordial leads[1,2,3,4].

1. BS is caused by a large abnormality of the myocardium ion channels and is determined by genetic mutations without being associated with any structural heart disease.

2. In recent years, many contributing genetic mutations have been identified, some mechanisms associated with the appearance of the characteristic phenotype have been clarified, and progress has been made in identifying clinical prognostic markers. Three BS associated electrocardiographic patterns have been identified[5]. Type 1 pattern (Fig. 1) is distinguished by a convex ST height of 2 mm ST segment, followed by negative T waves in many exact precordial cables (V1-V3)[6]. The type 2 pattern is explained by an elevated concave elevation of the ST segment by about 2 mm in the right precordial lead, followed by positive T waves or isophase's showing an assisting ECG pattern.

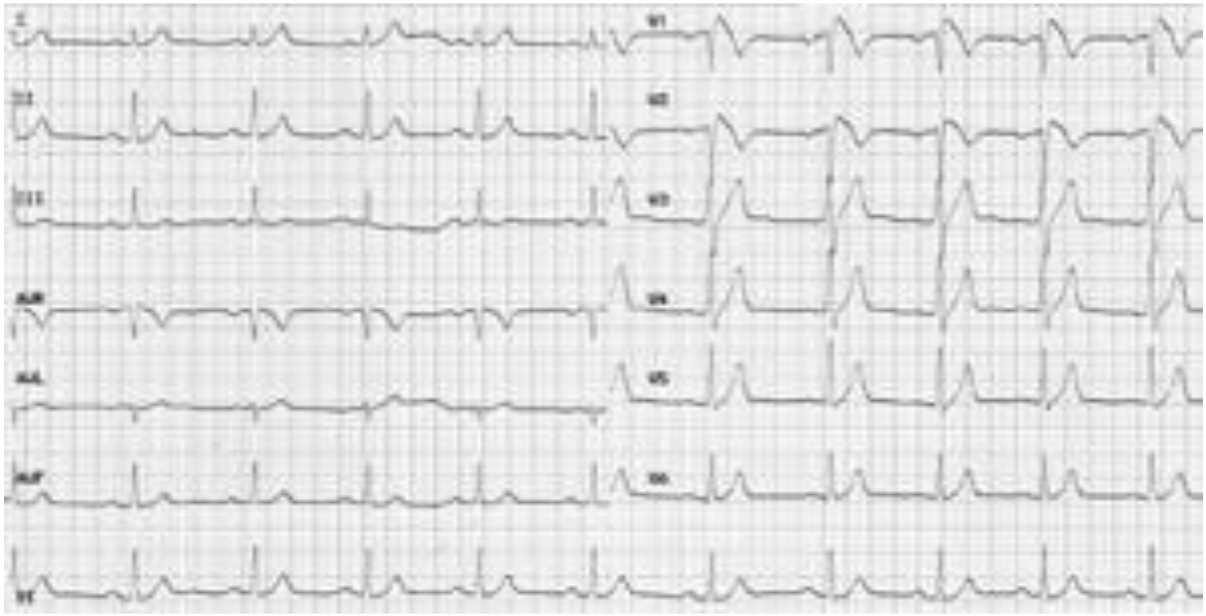

Fig 1. – 12-lead ECG showing a pattern of spontaneous type 1 Brugada syndrome in leads V1 and V2 (diagnostic).
Type 3 model (Figure 2) is defined as any of the above, with the segment height ST <1 mm. Type 1 formula is the only model accepted as a diagnosis. Type 2 and 3 patterns are significant, but BS are not analytical and require a positive result for the presence of ajmaline or flecainide, i.e. BS diagnostic confirmation requires the development of type 1 pattern.

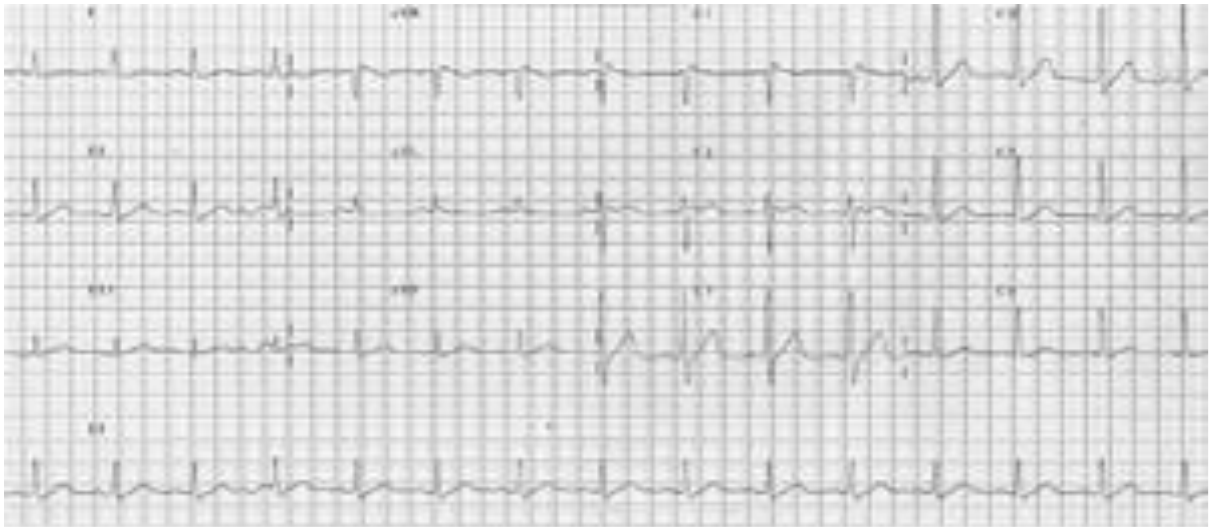

Fig 2. – 12-lead ECG showing a type 3 pattern Brugada syndrome in the exact precordial leads (suggestive, not diagnostic).

Patients with BS remain largely asymptomatic, but fainting or sudden death occurs at any time due to up to 25% of ventricular arrhythmias (torsade de pointes and / or ventricular fibrillation). This requires that all patients with a characteristic ECG pattern are isolated at risk even when they are isolated. We present the case of BS detected during the vocational exam.

A CASE REPORT:

The patient was a 37-year-old man who smoked nine cigarettes a day and 70 cigarettes for 22 years ago, but is currently undergoing smoking cessation therapy and there is no history of alcohol or other substance abuse. bodybuilding and diving. There were no fainting or sudden death in the family. For eight years he worked at a company manufacturing wood cutting machines, the main risk of industrial hygiene was exposed to noise, cutting oils and chlorinated solvents (methylene chloride and trichloroethylene) used for cleaning. He also worked as a security guard, where from time to time he was exposed to very stressful situations. In a routine professional examination, the physical examination was satisfactory, blood pressure was 120/70 mmHg, and total cholesterol 318 mg / dl (LDL: 150 mg / dl) and triglycerides 254 mg / dl in blood counts. The ECG detected a high increase in the segment of the saddle ST 2 in the left pre-moon hemisphere and the right precordial lead (Fig. 3) (type 3).

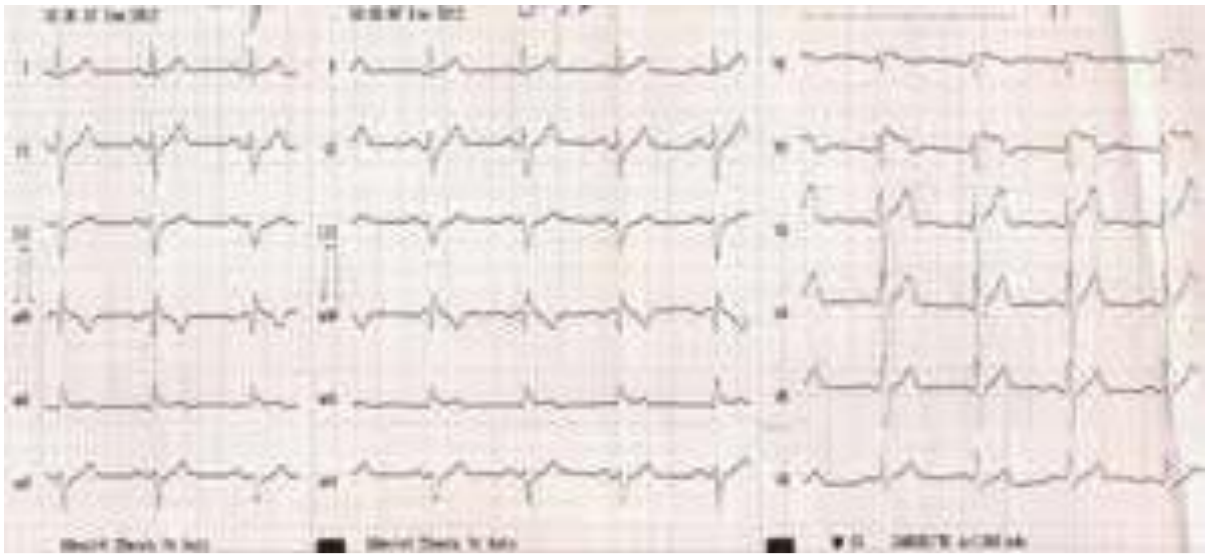

Fig 3. – ECG of the patient reported here, showing a type 2 pattern Brugada syndrome (in V2: concave ST segment elevation> 2 mm followed by isodiphasic saddle back pattern T wave).

The employee was sent to a referral center for cardiac arrhythmias and underwent a flecainide test to detect high-quality clinical tolerance confirming the diagnosis of BS and the transition from model 2 to type 1.

To complete the risk classification, electrophysiological studies were first performed showing normal conduction intervals, and secondly, continuous ventricular arrhythmias were not induced during the ventricular pacing protocol. Therefore, the patient was assessed to have a low arrhythmia risk profile (asymptomatic, non-diagnostic ECG without induction in an electrophysiological study). Since reports show that fever can cause ventricular arrhythmias in patients with BS, it is recommended to avoid energy treatment with drugs and addictive substances and any feverish diseases and drugs listed in the table with the necessary necessary consultation in case of syncope. An appointment was made for genetic tests and check-ups at a cardiology outpatient clinic. Six months later the patient remained asymptomatic.

DISCUSSION:

BS is in a set of heart diseases called channelopathies, diseases caused by abnormalities associated with the formation of cellular action potential in transmembrane ion channels, the most serious consequence is a predisposition to malignant ventricular arrhythmias[7,8]. Channelopathy is simply an electrical disorder and is usually not associated with any underlying structural heart disease 4. BS is expected to increase from 4 to 12% of all sudden expiries and 20% of sudden expiries[9]. They happen in a seemingly normal heart. The estimated incidence of BS is 5/10,000 people, but this number probably underestimates the true incidence because many patients may have silent forms of the disease[10]. Implantable cardioverter-defibrillator (ICD) is the only treatment that prevents sudden death in BS. Current indications for ICD implantation are recommended by the Second International Consensus BS[13]. Patients with symptomatic BS (sudden fainting, sudden death stopped) and asymptomatic patients in whom electrophysiological examination causes continuous ventricular arrhythmias, especially spontaneous[11,12]. ECG patterns are considered candidates for ICD implantation. Regardless of the level of risk of arrhythmia, it is recommended for all patients diagnosed with BS to avoid certain drugs and various drugs (Table I; full list is available at www.brugadadrugs.org. We also recommend aggressive fever management, emergency consultations in the event of sudden loss of consciousness, and regular monitoring by a cardiologist[14,15].

Main medicaments and substances of abuse that should be evaded in people with Brugada disease*

Medicament	Antiarrhythmics	Ajmaline Pilsicainide Flecainide Propafenone Procainamide
	Psychotropic agents	Amitriptyline Desipramine Clomipramine Loxapine Lithium Nortriptyline Trifluoperazine
	Anaesthetics	Bupivacaine Oxcarbazepine Propofol Procaine
	Others	Acetylcholine Ergonovine
Substances of abuse	Cocaine Alcohol	

*** Changed by: Wolpert C, Postema PG, Amin AS et al. patients and Drugs with Brugada syndrome: literature review, approvals and updated website (www.brugadadrugs.org). Heart rate in 2009; 6: 1335–41.**

Biological, physical and chemical hazards in the workplace can lead to cardiac conduction disorders. Table II first describes the main chemicals that can cause arrhythmias, as well as those that can cause toxic hyperthermia. This means that workers exposed to these substances may have an increased risk of unexpected expiry if they have BS. In addition, physical factors such as electricity or extremely stressful professions, such as elite athletics or football, may support the occurrence of arrhythmias and or conduction disorders and may be discouraging in this patient group.

Table II. Main occupational risk associated with the development of cardiac arrhythmias

Main occupational risk associated with the development of cardiac arrhythmias		
Toxic agents	Chlorinated solvents	Trichloroethylene Tetrachloroethylene Methyl chloroform Methylene chloride
	Freons	Chlorodifluoromethane
	Organophosphate insecticides	Chlorpyrifos
	Heavy metals	Antimony
Physical agents	Electricity	Exposure above 30 mA
	High temperatures	Metal fume fever (Zn, Cu, Cd, Mg). Polymer fume fever. Heat stress.
Others	Highly-stressful situations.	

ECG is useful in detecting electrocardiographic abnormalities associated with sudden death, as in the case of BS. In occupations with risk profiles, as described in Table II, the inclusion of ECGs in medical controls may help in the early detection of BS, but additional sensitivity, specificity and predictive value testing will be required.

CONCLUSION:

Brugada syndrome is distinguished by fainting or sudden death based on the electro-cardio graphic outline of ST segment advancement in the normal heart, but on the precordial paths from V1 to V3 and the right bundle division block. Polymorphic ventricular tachycardia is caused by syncope and sudden death. These electrocardiographic symbols may appear during a routine professional examination of an asymptomatic patient.

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