Research Paper Cardiology

Brugada Syndrome: Detection and Control Depending on Occupational Risks

Marisa Ruiz	Prevengraf. Barcelona, Spain
Eva Marín	Prevengraf. Barcelona, Spain
Santiago Nogué	Clinical Toxicology Unit. Hospital Clínic. Barcelona, Spain
Paola Berne	Arrhythmia Section, Cardiology Department, Thorax Institute. Hospital Clinic. Barcelona, Spain
Josep Brugada	Arrhythmia Section, Cardiology Department, Thorax Institute. Hospital Clinic. Barcelona, Spain
George Delclos	The University of Texas School of Public Health, Houston, Texas, USA
Pere Sanz-Gallen	Clinical Toxicology Unit. Hospital Clínic. Barcelona, Spain

ABSTRACT

Introduction
Brugada syndrome is characterized by episodes of syncope or sudden death in patient with a normal heart but with an electrocardiographic pattern of ST segment elevation in precordial leads V1 to V3 and morphology resembling right bundle branch block. Syncope and sudden death are due to polymorphic ventricular tachycardia. These electrocardiographic signs may be discovered by chance during a routine occupational check-up of an asymptomatic patient.
Case report
A 37-year-old man with no history of syncope or fainting attended an occupational medicine service for a routine check-up. ECG showed an electrocardiographic pattern suggestive of Brugada syndrome type 2. The patient was referred to a reference centre for cardiac arrhythmias, where a flecainide test confirmed the diagnosis. Electrophysiological study showed a low profile of arrhythmic risk, and the patient was advised to avoid consumption of some medicaments and drugs of abuse that have been associated with sudden death in patients with Brugada syndrome. The patient remains asymptomatic 6 months after the diagnosis.
Conclusions
Occupational physicians making routine check-ups may diagnose Brugada syndrome and help prevent sudden death.
It is important to know the occupational risks of a worker diagnosed Brugada syndrome to prevent or minimize the occurrence of ventricular arrhythmias.

KEYWORDS Brugada Syndrome. Ventricular Arrhythmia. Occupational Health. Occupational Medicine.

INTRODUCTION

Brugada syndrome (BS) was first described in 1992 and is characterized by a typical electrocardiographic pattern in right precordial leads and predisposition to ventricular arrhythmias and sudden death [1].

BS is due to a primary abnormality of the ion channels of the myocardium and is determined by genetic mutations, without necessarily being associated with any structural heart disease [2]. In recent years, many causative gene mutations have been identified, some of the mechanisms involved in the appearance of the characteristic phenotype have been elucidated, and advances have been made in identifying the clinical prognostic determinants.

Three electrocardiographic patterns associated with BS have been described. The type 1 pattern (Figure 1) is characterized by convex elevation of the ST segment of ≥ 2 mm in more than one right precordial lead (V1-V3), followed by negative T waves. The type 2 pattern is characterised by a concave elevation of the ST segment of ≥ 2 mm in right precordial leads followed by positive or isodiphasic T waves, showing a saddle back pattern on ECG.

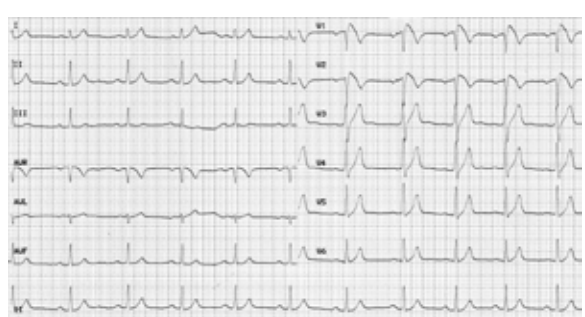

Fig 1. – 12-lead ECG showing a pattern of spontaneous type 1 Brugada syndrome in leads V1 and V2 (diagnostic).

The type 3 pattern (Figure 2) is defined as either of the above with an ST segment elevation of <1 mm. The type 1 pattern is the only one considered diagnostic. Type 2 and 3 patterns are suggestive but not diagnostic of BS and require a positive ajmaline or flecainide test, i.e., diagnostic confirmation of BS requires the development of a type 1 pattern.

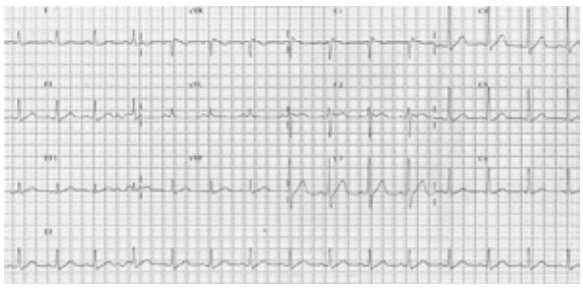

Fig 2. – 12-lead ECG showing a type 3 pattern Brugada syndrome in the right precordial leads (suggestive, not diagnostic).

BS patients remain largely asymptomatic, but up to 25% present with syncope or sudden death due to ventricular arrhythmia (torsade de pointes and/or ventricular fibrillation) at some time. This requires that all patients with a characteristic electrocardiographic pattern, even when it is isolated, should be considered as at risk.

We report a case of BS detected during an occupational check-up.

CASE REPORT

The patient was a 37-year-old man who smoked four cigarettes/day and previously had smoked 50 cigarettes/day for 18 years, but was currently receiving smoking cessation therapy, and had no history of alcohol or other substance abuse, and engaged in bodybuilding and diving. He had no history of syncope or of sudden death in the family. For eight has worked at a company that made machines for sawing wood, the main industrial hygiene risk was exposed were noise, cutting oils and chlorinated solvents used to clean (methylene chloride and trichlorethylene). For four works as a security guard where occasionally exposed to highly- stressful situations

In a routine occupational check-up, the physical examination was normal, blood pressure was 110/60 mmHg and, in the blood count, total cholesterol was 328 mg/dl (LDL: 160 mg/dl) and triglycerides were 244 mg/dl. The ECG detected a left anterior hemiblock and elevated ST saddle back pattern segment elevation in the right precordial leads (Figure 3) suggestive of a type 2 BS.

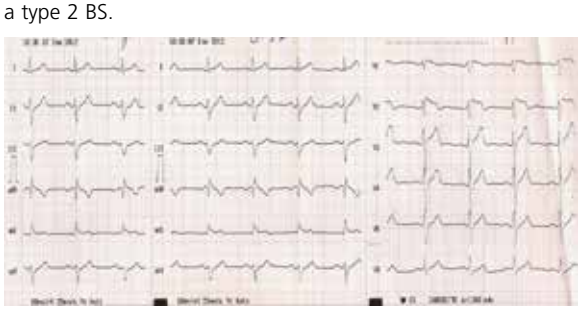

Fig 3. – ECG of the patient reported here, showing a type 2 pattern Brugada syndrome (in V2: concave ST segment elevation> 2 mm followed by isodiphasic saddle back pattern T wave).

The worker was referred to a reference centre for cardiac arrhythmias, and underwent a flecainide test with good clinical tolerance and the detection of transition from a type 2 to type 1 pattern, confirming the diagnosis of BS.

To complete risk stratification, electrophysiology studies were carried out, which showed, first, normal conduction intervals and, second, the non-induction of sustained ventricular arrhythmias during the ventricular stimulation protocol. Therefore, the patient was judged to have a low profile of arrhythmic risk (asymptomatic, non-diagnostic baseline ECG, no inducibility in the electrophysiological study). Avoidance of the medicaments and substances of abuse listed in table I[3] and vigorous treatment of any febrile illness was advised, as reports show that fever can trigger ventricular arrhythmias in patients with BS, with urgent consultation advised in the case of syncope. An appointment was made for genetic studies and follow-up in the cardiology outpatient clinic. Six months later, the patient remained asymptomatic.

DISCUSSION

BS is included in the group of heart diseases known as channelopathies, diseases caused by abnormalities in the transmembrane ion channels involved in the genesis of the cellular action potential, whose most serious consequence is the predisposition to malignant ventricular arrhythmias. Channelopathies are purely electrical disorders and typically are not associated with any underlying structural heart disease [4].

BS is estimated to cause 4-12% of all sudden deaths and up to 20% of sudden deaths that occur in an apparently normal heart. The estimated prevalence of BS is around 5/10,000 persons, although this figure probably underestimates the true prevalence, since many patients may have silent forms of the disease.

An implantable cardioverter defibrillator (ICD) is the only proven treatment for the prevention of sudden deaths in BS. Current indications for ICD implantation are those recommended by the Second International Consensus on BS [5]. Patients with BS with symptoms (sudden syncope, aborted sudden death) and asymptomatic patients in whom the electrophysiology study induces sustained ventricular arrhythmias, especially those who present a spontaneous type pattern on ECG are considered candidates for ICD implantation. All patients diagnosed with BS, regardless of the level of arrhythmic risk, are advised to avoid certain medicaments and a number of drugs (Table I; for the complete list see www.brugadadrugs.org). We also recommend aggressive treatment of fever, urgent consultation if sudden loss of consciousness occurs and regular monitoring by a cardiologist.

Table I. Main medicaments and substances of abuse that should be avoided in people with Brugada syndrome.

Main medicaments and substances of abuse that should be avoided in people with Brugada syndrome*		
Medicament	Antiarrhythmics	Ajmaline Flecainide Pilsicainide Procainamide Propafenone
	Psychotropic agents	Amitriptyline Clomipramine Desipramine Lithium Loxapine Nortriptyline Oxcarbazepine Trifluoperazine
	Anaesthetics	Bupivacaine Procaine Propofol
	Others	Acetylcholine Ergonovine
Substances of abuse	Alcohol Cocaine	

*** Modified from: Postema PG, Wolpert C, Amin AS et al. Drugs and Brugada syndrome patients: review of the literature, recommendations, and an up-to-date website (**www.brugadadrugs.org**). Heart Rhythm 2009;6:1335-41.**

Biological, physical and chemical risks in the workplace may lead to heart conduction disorders [6].

Table II describes, first, the main chemicals which can cause cardiac arrhythmias and also those that may cause toxic hyperthermia. This shows that workers exposed to these sub-

stances may have an increased risk of sudden death if they have BS. In addition, physical agents such as electricity or highly-stressful occupations, such as elite athletics or football, could favour the presentation of arrhythmias and/or conduction disorders and should perhaps be discouraged in this group of patients.

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 for the detection of electrocardiographic abnormalities associated with sudden death, as in the case of BS. In occupations with a risk profile such as that described in table II, the inclusion of ECGs in medical check-ups could aid the early detection of BS, although further studies of the sensitivity, specificity and predictive value would be required.

REFERENCES

| 1. Brugada P, Brugada J. Right bundle branch block, Persistente ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome. J Am Coll Cardiol. 1992;20:391-6. | 2. Lippi G, Montagnana M, Meschi T, Comelli I, Cervellin G. Genetic and clinical aspects of Brugrada syndrome: an update. Adv Clin Chem. 2012;56:197-208. | 3.- Postema PG, Wolpert C, Amin AS et al. Drugs and Brugada syndrome patients: review of the literature, recommendations, and an up-to-date website (www.brugadadrugs.org). Heart Rhythm 2009;6:1335-1341. | | 4. Capulzinia L, Brugada P, Brugada J, Brugada R. Arrhythmia and right heart disease: From genetic basis to clinical practice. Rev Esp Cardiol.2010;63:963-83. | | 5.Sacher F, Probst V, Iesaka Y, Jacon P, Laborderie J, Mizon-Gerard F, et al. Outcome after implantation of a cardioverterdefibrillator in patients with Brugada syndrome. Circulation. 2006;114:2317-22. | | 6. Chamoux A, Malaville P-Y. Pathologie cardiovasculaires professionnelles. EMC (Elsevier Masson SAS, Paris), Pathologie professionnelle et de l'environnement 16-531-A-10, 2010. |