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CASE REPORT

NEUROLEPTIC-INDUCED BRUGADA SYNDROME A CASE REPORT

Dr Fouad Laboudi[1], MD PhD ; Dr Ghizlane Slimani[1], MD ; Dr Mohamed Essaid Gourani[2], MD ; Pr Abderrazzak Ouanass[1] MD.

[1] Psychiatric Emergency Department, Ar-razi Psychiatric University Hospital, Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, Morocco.

[2] Psychiatry Department, Ouarzazate, Morocco.

ABSTRACT

Brugada syndrome is a rare genetic disease of autosomal dominant inheritance with low penetrance, manifested by ST segment elevation at right precordial V1, V2 and V3 shunts, and a right bundle branch block aspect at the electrocardiogram. It exposes to a high risk of ventricular arrhythmia that can cause syncope (fainting) and even sudden death on a structurally normal heart.

We report here the case of a 25-year-old patient with neuroleptic-induced Brugada syndrome.

To our knowledge, this is the first reported case of neuroleptics-induced Brugada syndrome, Morocco. Therapeutic management is based on Amiodarone and beta-blockers. Regular monitoring of the ECG should be performed, however, on patients taking psychotropic drugs and also during associations.

KEY WORDS: Brugada syndrome, Neuroleptic, Electrocardiogram, Sudden death, Morocco.

Corresponding author:

Dr Laboudi Fouad, MD, PhD: Assistant Professor at Psychiatric Emergency Department,
Ar-razi Psychiatric University Hospital, Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, Morocco.
Email: docteurfouadlaboudi@gmail.com ; Tel: +212661439780

INTRODUCTION

Brugada syndrome is a rare genetic disease of autosomal dominant inheritance with low penetrance, manifested by ST segment elevation at right precordial V1, V2 and V3 shunts, and a right bundle branch block aspect at the electrocardiogram. It was first described in 1992. It exposes to a high risk of ventricular arrhythmia that can cause syncope (fainting) and even sudden death, on a structurally normal heart. It is a disease that few carriers of its mutation show the clinical signs of the disease. Its prevalence is about 0.15% in adults and 0.005% in children in Asia and less than 0.02% in the West. For the higher prevalence in Asia, it is likely that specific ethnic polymorphisms alter the activity of the main pathogenic mutation. In Japan, the incidence of Brugada electrocardiogram is 14.2 per 100,000 people / years. Data from multicenter reports indicate that its frequency in men with Brugada syndrome is higher in Japanese patients (94-96%) than in Caucasian patients (72-80%). It seems more common in men who may have more severe forms. Healthy people with a Brugada-type ECG have a favorable prognosis with an annual mortality rate of less than 0.5% [1]. The prognosis is severe in patients with clinical symptoms. Current researches on this disease mechanism are in favor of mutations of mainly sodium channel receptors in myocardial cells. Sometimes it is possible to look for family mutations when there is evidence of several family cases; however, despite this research, the presence of mutation is not correlated with an increase in the risk of occurrence of serious events, so this research remains questionable [2].

The objective of this work is to report a neuroleptic-induced Brugada syndrome. To our knowledge, this is the first reported case of neuroleptic-induced Brugada syndrome Morocco. Hence the need to highlight the peculiarity of this disorder.

CASE REPORT

This is a 25-year-old patient, with no notion of sudden death in the family or similar case, he was hospitalized at the psychiatric university hospital Arrazi of Salé for a symptomatic reactivation of schizophrenia and was put under neuroleptics (Risperidone 4mg / d). After two days of hospitalization, there was the installation at the walking force of syncope without notion of palpitations or chest pains. The patient reports the notion of dizziness that preceded syncope when changing position (from the lying to the standing position). The clinical examination finds temperature at 37 ° C, respiratory rate at 18 / min, blood pressure at: 100/60 mmHg in a lying position and 97/60 mmHg when standing. The biological exam was normal. The electrocardiogram found heart rate at 78 / min, without extrasystoles, QTC 40 / m and Aspect of Brugada syndrome type 1 (Fig. 1), that showed a right bundle branch block aspect associated with a domed ST segment elevation and T wave abnormalities in right precordial derivations (V1 to V3). A cardiology opinion was requested with stopping the Risperidone and putting the patient under Diazepam alone. We redid the biological exam which was strictly normal. The etiological exam was without anomalies. The patient was putted under Amisulpride at a dose of 400 mg / day and referred to the cardiology clinic for further follow-up.

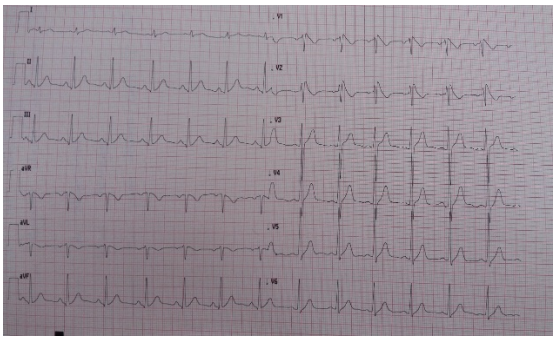

Figure 1: Patient Electrocardiogram

DISCUSSION

Brugada syndrome is a rare genetic disease of autosomal dominant inheritance with low penetrance, its diagnosis is based on the association of electrocardiogram abnormalities (ST segment elevation), and syncope or sudden death events. In many cases, practitioners are led to comment on the reality of this diagnosis in asymptomatic patients with only an abnormal appearance on the electrocardiogram. In practice, it is necessary to distinguish the Brugada syndrome diagnosed from a simple appearance of Brugada on the electrocardiogram, often discovered fortuitously, and whose risks are much lower. Brugada syndrome was described for the first time in 1992 by Pedro and Josep Brugada in the form of a right bundle branch block and persistent ST-segment elevation in the right precordial shunts (V1 to V3) [3]. Since then, it has been diagnosed in more than 300 patients in the literature [4 ,5] and is thought to be responsible for 40-60% of idiopathic ventricular fibrillations and in some families it is associated with a mutation in the sodium channel gene SCN5A [6] causing intramyocardial sodium disturbances [4,7], leading to characteristic electrocardiographic (ECG) changes. There is growing evidence that some drugs or medical conditions can unmask Brugada's syndrome in a patient with previously normal electrocardiograms and no cardiac history. it is reported in the literature that the electrocardiogram is often hidden, but may be unmasked or modulated by sodium channel blockers, febrile states, vagotonic agents, alpha-adrenergic agonists, beta-blockers, tricyclic or tetracyclic antidepressants, first-generation antihistamines (Dimenhydrinate), a combination of glucose and insulin, hyperkalemia, hypokalemia, hypercalcemia, and alcohol and cocaine toxicity [8-10]. The syndrome is thought to be responsible for 4 to 12% of all sudden deaths and at least 20% of deaths in patients with structurally normal hearts. The clinical phenotype is 8 to 10 times more prevalent in men than in women.3-5 BrS typically manifests with syncope and cardiac arrest, occurring in the third and fourth decades of life, and usually at rest or during sleep [11]. We report here the first case of the Brugada syndrome in psychiatry in Morocco precipitated by the use of Risperidone, in a patient without a cardiac history.

There are three ECG types of syndrome. Only the type 1 electrocardiographic appearance or typical appearance should be retained, and which corresponds to a downward shift of the ST segment superior to 0.2 mV on more than one right precordial shunts (V1-V3), with a domed appearance or "coved type" and this is the case of our patient who was put under Risperidone type of antipsychotic. It should be noted that all cases of Brugada syndrome induced by antipsychotics and antidepressants were of the type 1 phenotype. Rouleau et al. described three cases of electrocardiogram induced by a psychotropic drug; during concomitant administration of Amitryptyline and a phenothiazine (case 1), overdose of Fluoxetine (case 2) and co-administration of Trifluoperazine and Loxapine (case 3) [12]. A study of 98 patients with an overdose of tricyclic antidepressants indicated that 15 of them had an electrocardiogram consistent with Brugada syndrome . Overall mortality was 3% in all patients, but 6.7% in patients with a Brugada phenotype. For the management of Brugada syndrome, several recommendations have been published in 2005 [13]. Others, Americans and Europeans, concerning the disorders of the rhythm of genetic origin were published in 2013 [14]. Currently there is no drug treatment that can prevent ventricular arrhythmias in Brugada syndrome. Quinidine is used by some, empirically for the moment [15]. Therapeutic management is based on Amiodarone and betablockers. If they prove to be ineffective, the implantable automatic defibrillator (ICD) helps prevent sudden death in this syndrome . There is currently consensus to suggest ICD implantation in most patients who have had symptoms [13]. However, in these cases it must be ensured that the episode of sudden death and / or syncope do not have a cause other than Brugada syndrome, this is not always easy as the discomfort and syncope "vagal appearance" are common in these patients .

Note that regular ECG monitoring should be performed on patients taking psychotropic drugs and also during associations.

CONCLUSION

We report here the first case of Brugada syndrome in psychiatry in Morocco precipitated by the use of Risperidone, in a patient without cardiac history. There is currently no drug treatment that can prevent ventricular arrhythmias in Brugada syndrome. The prognosis is severe in patients with clinical symptoms, so regular ECG monitoring should be performed on patients taking psychotropic drugs and also during associations. The case is reported here to highlight the obscure prognosis of the disease. Even among asymptomatic patients.

AUTHORS' CONTRIBUTIONS

The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly work in Medical Journals of the International Committee of Medical Journal Editors. Indeed, all the authors have actively participated in the redaction, the revision of the manuscript and provided approval for this final revised version.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report.

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Declared none.

COMPETING INTERESTS

The authors declare no competing interests.

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