ELECTROPHYSIOLOGY
Case Report
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Torsades de Pointes induced by propofol in a patient with Wolff Parkinson White Syndrome: Case report

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ABSTRACT

We describe a clinical case of a 57-year-old female patient with Wolf Parkinson White Syndrome, who, during anesthetic induction with propofol for ablation of the accessory pathway, presented polymorphic ventricular arrhythmia, specifically Torsades de Pointes.

KEYWORDS: Torsades de Pointes; Propofol; Wolff-Parkinson-White Syndrome.

INTRODUCTION

Torsades de Pointes (TdP) is a polymorphic ventricular tachycardia associated with an increased QT interval. The electrocardiographic manifestation occurs due to the prolongation of the action potential and repolarization of the cardiomyocyte, which increases the susceptibility to early depolarizations, the trigger of the arrhythmia. Corrected QT (QTc) intervals >500 ms are associated with an increased risk of cardiovascular events[1-3].

Propofol is an intravenous anesthetic widely used in medical practice for general anesthesia and sedation in diagnostic or therapeutic tests, such as electrophysiological studies (EPS) and ablations. Its pharmacokinetic characteristics allow for rapid awakening and easy titration without cumulative effects. However, propofol is not free from adverse effects, and its ability to act on the cardiac conduction system has been reported in the literature[4].

This report describes the clinical case of a female patient with Wolff Parkinson White (WPW) syndrome who, during anesthetic induction with propofol for ablation of the accessory pathway, presented polymorphic ventricular arrhythmia, specifically TdP.

This study was approved by the Ethics Committee of CEP/HUGO under protocol number 85497418.2.0000.0033.

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CASE REPORT

The present case is of a female patient, 57 years old, teacher, with a personal pathological history of obesity grade I, hyperuricemia, heart failure with preserved left ventricular ejection fraction (LVEF 53% Simpson) in NYHA functional class (FC) I and pulmonary hypertension with pulmonary artery systolic pressure of 43 mmHg. Social drinker, sedentary, denied smoking or use of illicit drugs. She denied the use of continuous medications or allergies. There was no history of sudden death in the family. She sought a cardiologist for investigation after multiple admissions to the emergency unit due to palpitations and pre-syncope with evidence of supraventricular paroxysmal tachycardia. The electrocardiogram showed sinus rhythm, short PR interval with pre-excitation, and prolonged corrected QT interval of 478 ms (Fig. 1). An electrophysiological study (EPS) with conventional mapping was then requested.

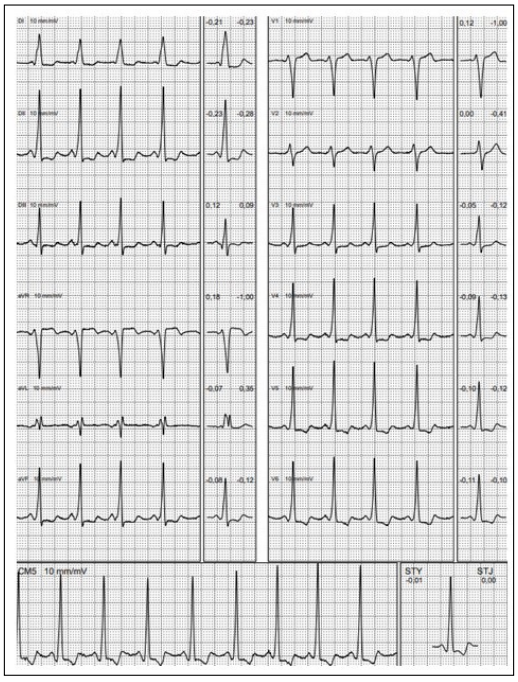

Figure 1. Pre-ablation resting electrocardiogram: Sinus rhythm, short PR interval, delta wave, presence of ventricular extrasystoles and corrected QT interval 478 ms.

The EPS revealed alterations that, together with the clinical history, confirmed the diagnosis of WPW syndrome, with right anterior accessory pathway and induction of sustained orthodromic tachycardia, cycle rate of 328 ms, with reversal by atrial overdrive. The accessory pathway had a refractory period of 288 ms after administration of isoproterenol. In addition, it showed a prolongation of the QT interval of 480 ms and QTc of 544 ms (Table 1).

Table 1. Baseline ranges of the Electrophysiological Study performed by the patient.

Basal Intervals of Electrophysiological Study
PR: 74 ms (average from 110 to 220 ms)
QRS: 182 ms (average from 80 to 110 ms)
QT: 480 ms (average from 300 to 430 ms)
QTc: 544 ms (average from300 to 430 ms)
HV: -12 ms (average from 35 to 55 ms)
PRVAC: 288 ms

PR: PR interval; QRS: QRS interval; QT: QT interval; QTc: corrected QT interval; HV: HV interval; PRVAC: refractory period. Source: Elaborated by the authors.

After three months, radiofrequency ablation of the accessory pathway was scheduled. Punctures were performed in the right and left femoral veins and the right internal jugular vein, with the passage of 8F, 11F, and 6F introducers, respectively. Multipolar diagnostic catheters were positioned in the region of the bundle of HIS, right atrium, right ventricle, and coronary sinus. During the procedure, with the aid of three-dimensional electroanatomical cardiac mapping, when performing programmed ventricular stimulation at the apex of the right ventricle, it was possible to detect ventriculoatrial (VA) conduction through the right lateral accessory pathway, in addition to the right anterior accessory pathway evidenced in the previous EPS. Before starting the ablation, the patient presented nonsustained polymorphic ventricular tachycardia, TdP (Fig. 2), lasting a few seconds, with spontaneous reversal and without hemodynamic repercussions. This event was, therefore, attributed to the propofol administered during sedation.

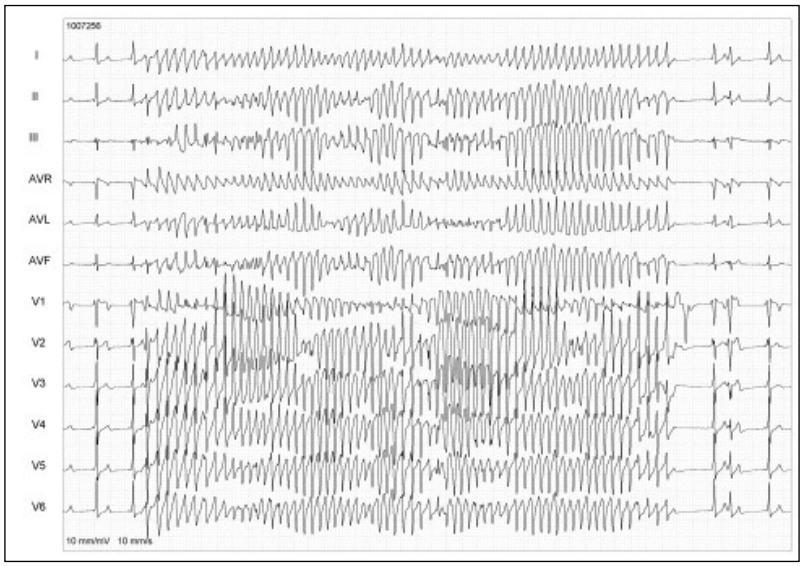

Figure 2. Electrocardiogram showing polymorphic ventricular tachycardia - Torsades de Pointes (TdP) with spontaneous reversal before ablation

Next, radiofrequency ablation of the right lateral and right anterior accessory pathways was performed with eight radiofrequency applications (Fig. 3). Adenosine was administered, which revealed no conduction through the accessory pathways, and the ablation was effective. At the end of the procedure, the ECG showed sinus rhythm without pre-excitation findings (Fig. 4).

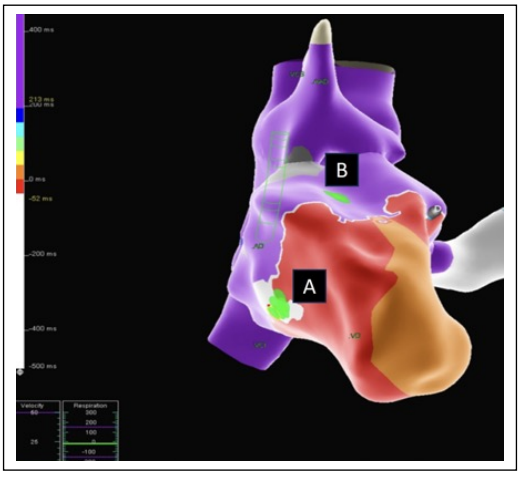

Figure 3. Ablation with radiofrequency electroanatomical mapping of right lateral (A) and right anterior (B) accessory pathways - indicated in green

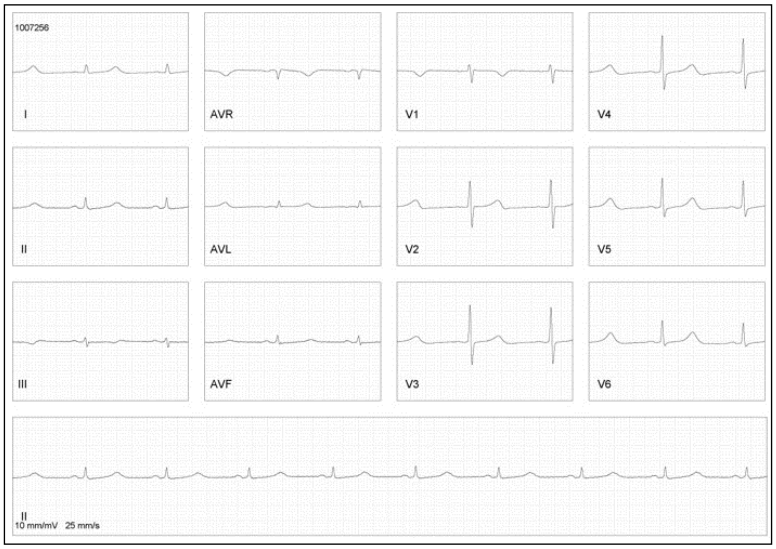

Figure 4. Immediate post-ablation electrocardiogram: Sinus rhythm, no pre-excitation, corrected QT interval 574 ms

The patient was discharged from hospital after 24 hours. At the consultation, 30 days after the procedure, she remained asymptomatic and the control ECG showed a QT interval within normal limits (Fig. 5).

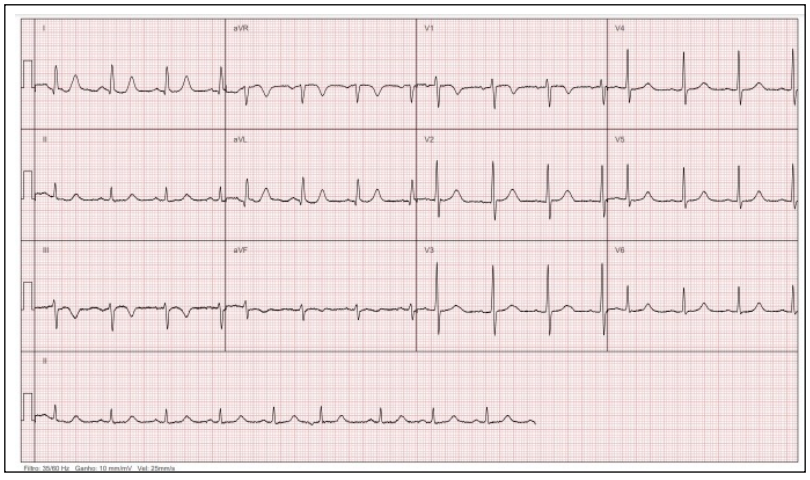

Figure 5. Control electrocardiogram after ablation of accessory pathways

DISCUSSION

Propofol, an anesthetic and sedative drug widely used in perioperative medicine, is cited in the literature for having both proarrhythmic and antiarrhythmic effects. In cardiomyocytes, this drug can block sodium, potassium, and calcium channels, reduce myocardial contractility, and lead to bradycardia, acting on the sinoatrial node. The effect of propofol on the QT interval is controversial. Abrich et al. concluded that its use should be avoided in patients with baseline ECG with prolonged QT. In contrast, Higashijima et al. described a significant reduction in the QTc interval during anesthetic induction with propofol[1,2].

Ellermann et al. showed in their meta-analysis that propofol may be beneficial in drug-induced prolonged QT interval, reducing the risk for TdP[3]. Seki et al. reported a case of normalization of electrocardiographic parameters in a patient with WPW during anesthetic induction with propofol. However, no direct effect on the accessory pathway was shown.[5].

In the present report, no QTc increase had been diagnosed before the electrophysiological procedure. At the time, when performing anesthetic induction with propofol, we observed ventricular arrhythmia, TdP. A similar case was described by Irie et al., a 70-year-old man who presented TdP during propofol infusion in the presence of severe hypoalbuminemia. The study suggested that hypoalbuminemia increased the free fraction of propofol, causing marked prolongation of the QTc interval and TdP[6].

In the study by Wutzler et al., less than 1% of the patients evaluated developed unexplained ventricular tachycardia. This study demonstrated that propofol infusion increased the QTc interval using the Fridericia formula, which was not observed when the calculation was made using the Bazett formula[7]. In our case report, the QTc interval was calculated using both formulas, which concluded that the QTc interval was increased. Furthermore, acquired causes of prolonged QT were excluded from the clinical investigation.

In 2010, Antzelevitch et al. reported changes in the amplitude of the J wave after propofol administration. However, the effects of this drug on ventricular repolarization are not conclusive. It is established that prolonging the QT interval favors the occurrence of ventricular arrhythmias[8].

The accessory pathways could have triggered the ventricular arrhythmia in the case presented. The study by Sharpe et al.[9] included patients with WPW syndrome, and Warpechowski et al. studied patients with nodal reentrant tachycardia, both with reentry mechanisms. Based on these studies, propofol does not interfere with the conduction system in patients with reentry[4].

CONCLUSION

In this report, the hypothesis was raised that propofol, commonly used in anesthetic induction, was responsible for inducing TdP since it may predispose to prolongation of the QTc interval and consequently contribute to ventricular cardiac arrhythmias in the absence of secondary causes.

CONFLICT OF INTEREST

Nothing to declare.

AUTHOR CONTRIBUTIONS

Conception and design: Souza AL, Oliveira LXA, Alessi SRB, Prudente ML, Frota ES, Guimarães EG, Silva RCO, Gardenghi G; **Data analysis and interpretation:** Souza AL, Alessi SRB, Guimarães EG, Gardenghi G; **Article writing:** Souza AL, Alessi SRB, Prudente ML, Guimarães EG, Gardenghi G; **Critical revision:** Souza AL, Alessi SRB, Guimarães EG, Gardenghi G; **Final approval:** Souza AL, Oliveira LXA, Alessi SRB, Prudente ML, Frota ES, Guimarães EG, Silva RCO, Gardenghi G.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable.

FUNDING

Not applicable.

ACKNOWLEDGMENTS

Not applicable.

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