Polymorphic ventricular tachycardia induced by Valsalva manoeuvre in a patient with paroxysmal supraventricular tachycardia

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A 54-year-old patient with episodes of paroxysmal supraventricular tachycardia developed non-sustained polymorphic ventricular tachycardia during Valsalva manoeuvre.

Case Report
A 54-year-old man was admitted for an episode of palpitations. He was previously in good health, took no regular medications, and had no major risk factors for ischaemic heart disease. He reported several episodes of self-terminating palpitations in the past years. Haemoglobin, thyroid-stimulating hormone, and electrolytes serum levels were within the normal range. A 12-lead electrocardiogram showed a narrow QRS-complex tachycardia at a rate of 210 b.p.m.; P-waves inscribed at the end of every QRS complex were detectable in D2 and aVF limb leads.

The patient was then instructed to perform Valsalva manoeuvre, during which a short burst of non-sustained, self-terminating polymorphic ventricular tachycardia was recorded (Figure 1) before cardioversion to stable sinus rhythm occurred. The ventricular arrhythmia onset was not preceded by significant bradycardia or pause. A repeated 12-lead electrocardiogram showed sinus rhythm without conduction system disease and with a normal QT interval.

A transthoracic echocardiography was normal. Cardiac troponin I levels measured on two occasions were within the normal range (<0.02 ng/mL). The patient refused to undergo further investigations (namely coronary angiography and electrophysiological study) and was discharged. He remained asymptomatic for the next 6 months.

Vagal manoeuvre is an effective therapy for patients with paroxysmal supraventricular tachycardia (SVT), having a classification of recommendation I with level of evidence B according to current guidelines. Despite being a safe therapeutic and diagnostic manoeuvre in most of the cases, previous publications reported the onset of ventricular arrhythmias during carotid sinus massage performed in patients both in sinus rhythm and during SVT.

In patients with an overt or concealed accessory atrio-ventricular (AV) connection (Wolff–Parkinson–White syndrome) vagal stimulation during re-entrant SVT can degenerate the arrhythmia into atrial fibrillation with rapid conduction over the accessory pathway.

Figure 1 12-lead ECG showing a burst of non-sustained polymorphic tachycardia elicited by Valsalva manoeuver during a narrow QRS-complex tachycardia (210 b.p.m.).
Given that intracardiac recordings during tachycardia or an electrophysiological study were not obtained, the presence of an accessory AV pathway in this patient cannot be excluded; however, several observations suggest its absence. First, the absence of pre-excitation signs (e.g. delta wave) during sinus rhythm. Secondly, the short RP interval (50 msec) observed during SVT. Thirdly, the first beat of the wide-complex tachycardia is shortly coupled to the previous complex of the SVT. Fourthly, the polymorphic aspect of the wide-complex tachycardia is not typical of conduction through an AV accessory pathway. Fifthly, the first narrow-QRS beat occurring after the burst of wide-complex tachycardia is followed by long-coupled, polymorphic wide-QRS complexes consistent with polymorphic ectopic ventricular beats.

Valsalva manoeuvre elicits a physiological response with alternating prevalence of sympathetic and parasympathetic activity: one might postulate that this complex interplay of the autonomic nervous system activity could have triggered the ventricular arrhythmia.

To the best of our knowledge, this is the first case reported of polymorphic ventricular tachycardia induced by Valsalva manoeuvre in a patient with SVT and without evidence of conduction through an accessory pathway.

This case underscores the need for vigilance and the ready availability of resuscitation equipment while performing vagal manoeuvres for therapeutic or diagnostic purposes.

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**References**


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**CASE REPORT**

**‘False-positive’ intrathoracic impedance monitor alarm caused by amiodarone-induced hypothyroidism in a patient with cardiac resynchronization therapy-defibrillator**

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A 78-year-old female received a cardiac resynchronization therapy-defibrillator equipped with an intrathoracic impedance (ITI) monitor and amiodarone therapy was initiated. A massive and long-lasting decrease in ITI occurred without heart failure (HF) deterioration. Pericardial effusion secondary to amiodarone-induced hypothyroidism may have caused the impedance reduction.

**Introduction**

Intrathoracic impedance (ITI), measured between the right ventricular coil and the device can, reflects intrathoracic fluid status. Intrathoracic impedance monitor (OptiVol) has been developed for early detection of heart failure (HF) deterioration. However, a decrease in ITI may not always mean a worsening of HF.

**Clinical case**

A 78-year-old female who survived cardiopulmonary arrest received a cardiac resynchronization therapy-defibrillator (Medtronic, Concerto) equipped with OptiVol in November 2008, and amiodarone therapy was initiated. The ITI began to decrease in late September 2009 when chest X-ray and echocardiographic findings were unremarkable (Figures 1A, C and G). The OptiVol fluid index crossed the OptiVol alert threshold in early October and reached >200 in late October (Figure 1A). However, she had no symptoms of HF and had been well until December 2009 when she became aware of swelling of the face and lower legs. Although diuretic dose was increased after a diagnosis of ‘HF deterioration’, the oedema did not improve. She noticed increasing easy fatigability and lethargy in February 2010 and was hospitalized. The cardiac silhouette became progressively larger in the past 5 months on the chest X-ray films (Figures 1C, D and G).

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