Aborted Sudden Death Secondary to Recurrent Transient Apical Dyskinesia

To the Editor,

We describe the case of a 77-year-old woman, ex-smoker, with mild emphysema, and bronchial hyperreactivity, admitted 2 years earlier at another hospital for bronchospasm with severe concomitant heart failure, who required treatment with vasoactive amines and ventilation support. The clinical condition was associated with generalized T-wave negativity, inversion mild enzyme elevation and alterations in cardiac contractility assessed as “consistent with myopericarditis”; the symptoms had all resolved at the time of discharge.

The patient was asymptomatic when she suddenly experienced sudden severe exertional dyspnea followed shortly afterwards by cardiopulmonary arrest. Basic cardiopulmonary resuscitation maneuvers were started immediately, followed by advanced maneuvers at 3 min. After monitoring the patient, sustained monomorphic ventricular tachycardia at 150 bpm was observed (Figure 1A); electrical cardioversion was applied and sinus rhythm recovered (Figure 1B). The patient was hemodynamically stable upon arrival to the emergency room. Echocardiography showed akinesia of the apical and middle segments with a left ventricular ejection fraction of 25%. Creatine kinase and troponin I concentrations were initially normal, but increased at 12 h to 374 U/L and 3.6 µg/L, respectively. NT-pro-BNP, a marker of acute heart failure, was raised to 15 944 pg/mL.

Clinical progress was favorable with diuretic and inotropic treatment. An electrocardiogram performed at 48 h shown deep, generalized T-wave negativity with QT interval prolongation (Figure 1C). The patient underwent catheterization, with no significant coronary lesions observed. Left ventriculography showed severe systolic dysfunction in the apical and middle segments (Figure 2). The endomyocardial biopsy was nonspecific.

On day 6 post-admission, the patient underwent an electrophysiological study. Sustained ventricular tachycardia

Figure 1. A: sustained monomorphic ventricular tachycardia with atrioventricular dissociation (arrows). B: ECG in sinus rhythm at the time of admission. C: ECG on day 2, showing deep, extensive T-wave negativity, and QT interval prolongation.
with 2 extrastimuli was induced, and on the basis of these findings, an automatic defibrillator was implanted. At the time of discharge, she presented mild apical hypokinesia with left ventricular ejection fraction of 55% and electrocardiographic persistence of negative T-wave, which had normalized in the 4-month follow-up.

Transient apical ballooning can simulate an acute coronary syndrome due to the onset of chest pain or sudden dyspnea and ECG alterations. It is characterized by transient akinesia of the apical and middle segments of the left ventricle with absence of significant coronary lesions.2,3 The condition mainly affects elderly women (82%-100%) and is usually associated with emotional or physical stress. A relationship with strenuous physical exertion and episodes of asthmatic bronchitis has been reported.2,4 Between 3% and 46% of patients develop severe acute heart failure. The majority recover ventricular function within 2-3 weeks. Associated tachycardia and/or ventricular fibrillation1 is described in 9% of patients, with the origin possibly related to the etiology (transient ischemia or catecholaminergic release) or the consequences of transient apical ballooning (QT interval prolongation, ventricular dysfunction).

Over the long-term, the condition can recur in up to 8% of patients.5 In our case, the patient presented a clinical picture consistent with transient apical ballooning two years before, which had been attributed to myopericarditis.

Therefore, transient apical ballooning should be considered in the differential diagnosis of ventricular arrhythmia and sudden death, particularly after physical or emotional stress.

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REFERENCES


Sildenafil Treatment of Unilateral Pulmonary Edema and Pulmonary Hypertension in Pulmonary Artery Agenesis

To the Editor,

Congenital pulmonary artery agenesis is a rare cardiovascular malformation, frequently oligosymptomatic,1,2 that can be complicated with unilateral pulmonary edema3 or pulmonary hypertension.1,2,4 We report the case of a patient with congenital pulmonary artery agenesis and pulmonary hypertension who developed unilateral pulmonary edema and discuss the role of sildenafil in pulmonary hypertension.

A 47-year-old woman, smoker of 20 cigarettes/day, with recurrent pulmonary infections, consulted for a hypertensive crisis. The physical examination was normal. The hemogram, biochemistry, coagulation tests, and electrocardiogram showed no alterations; there was mild hypoxemia in the arterial blood gas study. The chest x-ray revealed right pulmonary hyperlucency and a prominent left pulmonary artery. The echocardiogram showed concentric left ventricular hypertrophy.