Bidirectional Ventricular Tachycardia Due to Digitalis Poisoning

To the Editor:

Digitalis intoxication is a frequent outcome of chronic treatment with digitalis glycosides but uncommon following accidental intake or self-poisoning. Ventricular arrhythmias are an expression of this complication and can usually be controlled thru conventional means including suspension of the drug. However, in the presence of potentially lethal arrhythmias and kidney failure difficult decisions must be taken as normal antiarrhythmic therapy may be inappropriate and put the patient at risk. In ventricular arrhythmia, bidirectional ventricular tachycardia (BVT) is a rare outcome; it is virtually a diagnosis of digitalis toxicity and indicates a life-threatening situation.

We present the case of a 75-year-old woman with antecedents of chronic atrial fibrillation and arterial hypertension, being treated with acenocoumarol, digoxin (0.25 mg/day) and lisinopril, who attended Emergency Room for dyspnea. In the initial physical examination we found tachypnea, jugular venous distension when reclined at 45 degrees, normotension and a poor general clinical status. In the cardiorespiratory examination, auscultation showed arrhythmic tones at 150 beats/minute and bibasal crepitations. Analysis showed urea 149 mg/dL, creatinine 3.1 mg/dL, and potassium 5.1 mEq/L. The electrocardiogram (ECG) showed atrial fibrillation with a 150 beats/minute mean ventricular frequency (Figure 1). The patient was clinically stable following diuretic treatment and was hospitalized with improved renal function (urea, 130 mg/dL; creatinine, 2.6) and persistent hyperpotassemia (5.8 mEq/L). She maintained a regimen of 0.25 mg/day of oral digoxin until day 5 when she presented symptoms of left heart failure and hypotension (the ECG is in Figure 2). Frequency, regularity, and bidirectionality of the QRS complex in the frontal plane led us to diagnose BVT possibly caused by digitalis. Digoxinemia was 6 ng/mL (normal: 0.8-2 ng/mL). Given the potential life-threatening risk, disturbed ventricular rhythm in the presence of heart failure, kidney failure, hyperpotassemia, the fact that antiarrhythmic agents can entail increased risk and that electric cardioversion is counterindica-
work or ventricular myocardium, when QRS complex duration is $\geq 120$ ms.

Clinical use of antidigoxin Fab fragments was introduced in 1976 by Smith et al. It is generally agreed that they are effective and should be indicated in acute digitalis glycoside intoxication but no such consensus exists as to indications in intoxication during chronic treatment. The antiarrhythmic effect observed was especially relevant in our patient and avoided the risk associated with using antiarrhythmic agents and/or electric cardioversion in the presence of heart and kidney failure.

Administration of antidigoxin antibodies is a useful, safe alternative in the treatment of arrhythmias due to digitalis intoxication and hyperkalemia, which is frequently associated with acute toxicity, especially in life-threatening situations. Our patient presented BVT, an infrequent condition almost exclusively associated with digitalis intoxication. Bidirectional ventricular tachycardia is a polymorphic VT originating below the bundle of His bifurcation in which QRS complex morphology changes beat by beat. Although it has been related to effort- and/or emotion-induced sympathetic stimulation linked to a cardiac ryanodine receptor gene (hRyR2) mutation, it is most frequently found in other pathologies associated with elevated sympathetic tone, as in digitalis intoxication or in hydroelectrolytic disturbances, especially in the presence of heart failure. It has an ominous prognosis due to possible degeneration to ventricular fibrillation and sudden death. In digitalis intoxication, Na/K pump block provokes increased automatism of the subsidiary pacemakers situated in some of the principal specific conduits fascicles. Its involvement in impulse transmission means that in this type of ventricular tachycardia the QRS complex duration can be $<120$ ms; this contrasts with ventricular tachycardia originating in the Purkinje network or ventricular myocardium, when QRS complex duration is $2120$ ms.

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**REFERENCES**


Mycotic Aneurysm of the Aortic Arch

To the Editor:

Mycotic aneurysms are rare and potentially lethal if not diagnosed early: it is the total infection of the arterial wall. 1 Epidemiology changes and although bacterial endocarditis was originally the principal cause, it currently presents in older patients with atherosclerosis. Staphylococcus aureus, 2 Salmonella, and gram-negatives are the germs most frequently involved although in immunodeficient patients and intravenous drug-users any opportunist germ can be found.3,4 Seeding generally occurs on a diseased intima by direct contact with an adjacent focus of infection or by hematogenous or lymphatic dissemination.

Symptoms are highly nonspecific and patients often present a septic condition that is difficult to control and clinical manifestations related with early aneurysm rupture and rapid expansion, acting as local mass compressing adjacent structures.3,4 The diagnosis should be based on clinical suspicion and imaging techniques such as angiography and computerized tomography (CT), the technique of choice.3-5 We present the case of a young immunodeficient patient presented to our clinic with fever and cough, intense expectoration, continuous chest pain extending to the left shoulder with characteristics typical of pleuropericarditis.

In the physical examination he appeared in poor health with a temperature of 39°C, 30 respirations/minute tachypnea, 150 beats/minute tachycardia and blood pressure 120/70 mm Hg with absence of paradoxical pulse. We observed jugular venous distension when reclined at 45 degrees, as well as a pericardial friction rub and hypophony in the right base. The electrocardiogram revealed an atrial flutter at 140 beats/minute. Chest x-rays showed bilateral alveolar infiltration and grade II global cardiomegaly. Transthoracic echocardiography showed grade II pericardial effusion without hemodynamic deterioration in the right cardiac cavities. The valves were structurally and functionally normal without lesions indicating endocarditis. The root of the aorta and the ascending aorta were normal.

Following initial diagnosis of respiratory infection and associated pleuropericarditis we started treatment with wide spectrum antibiotics. At 10 days post-admission, we found no improvement in symptoms and modified the treatment after detecting 2*S aureus* positive blood cultures.

The x-ray showed progressive mediastinal thickening and helicoidal CT (Figure 1) identified a 10×13 cm saccular aneurysm in the aortic arch that compressed and displaced the left carotid artery, right brachiocephalic trunk and aortic arch. On confirming the presence of the aneurysm thru aortic angiography (Figure 2), the patient presented extensive ischemic stroke with abrupt diminution of level of consciousness and right hemiparesis. Initially, surgical intervention was not considered and the patient died 24 hours later, with strong suspicion of aneurysm rupture.

To summarize, we are dealing with an immunodeficient patient with initial symptoms of pneumonia, pleuropericardial infection, atrial arrhythmias and symptoms of sepsis, who did not respond to treatment. Following x-ray evidence of mediastinal thickening, CT confirmed the presence of a large saccular aneurysm in the aortic arch. Aortic angiography was performed to determine the exact location of the aneurysm neck and its anatomic relationship with the aorta branches.4,5