

Mianserin induced ventricular tachycardia

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Introduction

The potential of antidepressant drugs to have cardiovascular toxicity manifested by arrhythmias and impairment of left ventricular systolic function is well known; this is found especially for tricyclic compounds and in the elderly patients with preexisting cardiovascular diseases. However, most of the experimental and clinical studies lack to prove the arrhythmogenic effect of mianserin, a tetracyclic antidepressant even it can induce QT interval prolongation and intracardiac conduction abnormalities. We want to report a ventricular tachycardia in a young patient taking mianserin.

Case report

A 24 years old woman was referred to our clinic with diagnosis of ventricular tachycardia (VT). She had taken mianserin 30 mg/day for depression for the past 6 weeks, and after 1 month of treatment, she started to complain of palpitations and presyncopal episodes. She consulted a physician who recommended beta-blocker treatment (metoprolol 25 mg bid), due to suspicion of thyroid hyperfunction. The thyroid function tests were normal, but she continued to take metoprolol without resolution of symptoms. Noteworthy, two weeks before admission in our department an ambulatory Holter electrocardiogram (ECG) recording revealed nonsustained polymorphic VT (Fig. 1).

At admission, the clinical examination was normal, heart rate was 80 beats/min, blood pressure 120/60 mmHg, and a physiological S3 sound on cardiac auscultation was present. Chest roentgenogram and laboratory analysis were normal, including thyroid function tests and serum electrolytes. The ECG showed sinus rhythm, QRS axis at 60°, without any abnormality. A structurally heart disease was excluded by a normal echocardiography examination.

Late potentials were not present at signal averaged ECG recording. The Holter ECG performed 2 weeks ago, except presence of nonsustained VT with duration of 10 seconds and rate of 165/min, was without other abnormalities (heart rate variability parameters were normal, ventricular premature contractions and supraventricular arrhythmias were not presented).

Regarding the absence of any detectable abnormality mianserin induced VT was presumed and this treatment stopped. After 10 days since mianserin treatment was interrupted, VT was

not induced at programmed ventricular stimulation; we concluded that VT could be provoked by mianserin administration and the patient was discharged without treatment.

Discussion

Mianserin was thought to be a safe drug with respect to induction of cardiac arrhythmias. Experimental studies showed that in vivo mianserin could induce lengthening of the PR interval, widening of the QRS complex, and reduction in heart rate and blood pressure in rabbit (1). Also it can determine development of right bundle branch block, ventricular and supraventricular premature contraction or tachycardias (2). These experimental studies found mianserin to be one of the least cardiotoxic drugs compared with other antidepressants. These effects were produced in animals at plasma concentration several fold higher than therapeutics levels. At lower doses, mianserin seems to have no cardiotoxic effects proved by negative programmed ventricular stimulation in dog hearts after myocardial infarction (3). These effects are not related to blockade of noradrenaline uptake (2), but to the inhibition of HERG channels, similar with QT prolonging drugs.

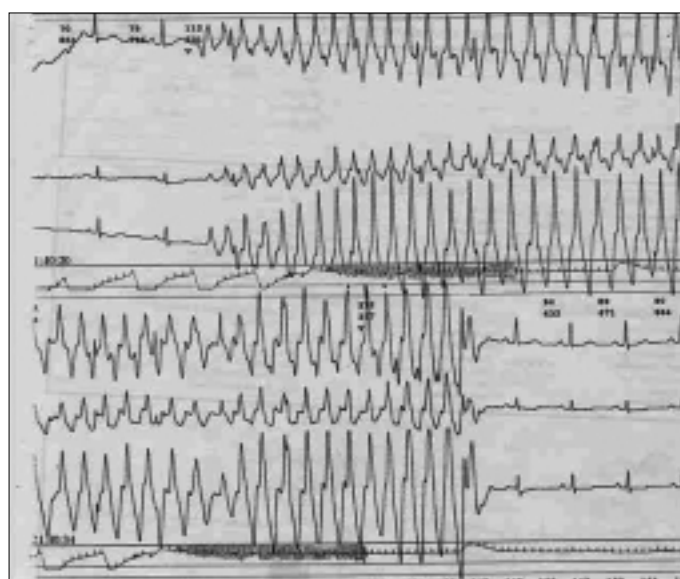


Figure 1. Ambulatory Holter electrocardiogram recording of nonsustained polymorphic ventricular tachycardia

However, clinical studies apparently showed that mianserin has no cardiotoxic effects (4, 5). The ECG parameters like RR and PR intervals, QRS duration and T wave amplitude are not significantly changed with administration of mianserin (5-8). Some authors find that it could induce reversible QT interval prolongation (9), but other study shows no significant changes in duration of QT interval (6). However, 24 hours ECG Holter recordings did not find arrhythmias in a patient taking mianserin (10). In elderly patients with preexisting cardiovascular disease, it can have deleterious effects on ventricular systolic function and peripheral circulation (10, 11).

The studies supported the idea that mianserin can have cardiovascular toxicity like other drugs in the class, but only at high doses, which can be administered only in experimental studies in animals, not in clinical practice. However, our patient took usual low-level doses and we have no reasons to suspect voluntary self-administration of higher doses. In addition, she had no complaint of palpitations before starting mianserin treatment.

This case raises the possibility of VT induced by mianserin in a young patient without detectable abnormalities of the cardiovascular system.

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