We thank Dr. Bollinger for his comments and interest in our report.

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REFERENCE


Amiodarone-Induced Adverse Effects at the Beginning of Oral Therapy

Clinical Implications

To the Editor:

We read with interest two reports published in a recent issue of CHEST (May 1997), highlighting the risk of rare but serious complications at the beginning of amiodarone therapy: torsade de pointes and acute pulmonary toxicity.1,2

Compared with other antiarrhythmic drugs, amiodarone appears to have fewer proarrhythmic effects. The occurrence of torsade de pointes with the use of amiodarone has been reported to be rare (less than 1%).1,3 It is usually observed during chronic treatment of patients with cardiac dysfunction in association with increases in drug dosage, electrolyte disorders, or concomitant antiarrhythmic therapy with class IA compounds.3

The report by Winters and colleagues1 involved a case of recurrent torsade de pointes occurring 11 days after initiation of oral amiodarone (200 mg tid) for the prevention of recurrences of atrial fibrillation after cardioversion in a patient with idiopathic dilated cardiomyopathy. We previously described a similar case of amiodarone proarrhythmia, which occurred earlier and at lower oral dosage than that observed by Winters and colleagues; our patient had a prosthetic mitral valve and heart failure treated for recurrent episodes of atrial flutter and fibrillation.4 Approximately 36 h after starting the amiodarone loading (total dose, 1,600 mg), a fast torsade de pointes causing palpitations and dizziness was documented. It was effectively treated with magnesium sulfate infusion and amiodarone discontinuation. No predisposing factors to the development of torsade de pointes were present, such as electrolyte disturbances, other antiarrhythmic or non-antiarrhythmic drugs, severe bradycardia, or abnormal QT prolongation. Atrial fibrillation recurred 20 days later and amiodarone was stopped again at a dosage of 200 mg/d. After 72 h of therapy (total dose, 600 mg), a new episode of asymptomatic torsade de pointes was recorded, again in the absence of any predisposing factor. Amiodarone was stopped indefinitely.

The second report, by Goldstein and colleagues,5 also attracted our attention. These authors described a case of amiodarone pulmonary toxicity, which occurred a few days after the drug was administered at a relatively low dose (total dose, 4,400 mg over 15 days). Unlike what occurred in this case, pulmonary toxicity is generally insidious and it develops months after the beginning of therapy (in 5 to 10% of patients) and at higher dosages than those reported in the cited paper.

Amiodarone is widely used and is very effective in the treatment of different supraventricular and ventricular tachyarrhythmias in various heart diseases. The early and unpredictable occurrence of such dangerous complications only few days after the start of amiodarone is in contrast with the expected delayed onset of antiarrhythmic effects, which is the criterion that generally guides clinical management. In our opinion, these cases emphasize the importance of careful clinical monitoring, possibly in the hospital in selected cases, when oral amiodarone is initiated. Furthermore, depending on the clinical picture and on the urgency of antiarrhythmic therapy, low dose loading should be preferable.

Amiodarone is a nonsustained polymorphic ventricular tachycardia during amiodarone therapy for atrial fibrillation complicating cardiomyopathy: management with intravenous magnesium sulfate. Chest 1997; 111:1454-57

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To the Editor:

The cases that Gardini et al mention are significant and raise concerns regarding the recognized, unrecognized, and potential adverse effects of amiodarone. As amiodarone use has accelerated for treatment of patients with cardiac rhythm disturbances other than life-threatening ventricular tachyarrhythmias, the need for all physicians (not just electrophysiologists) to recognize the potential acute and subacute adverse effects must be reinforced. Clearly, the possibility of aggravating underlying cardiac rhythm disturbances, as well as precipitating acute systemic and/or organ toxicities (such as pulmonary toxicity), needs to be considered. Only if physicians are vigilant will these potential problems be avoided or treated when they occur.

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To the Editor:

We thank Gardini et al for their enlightening remarks concerning our recently published report of a case of very early amiodarone pulmonary toxicity manifesting with hemoptysis. Indeed, we