was subsequently noted to have no dramatic change in clinical status, either objectively or subjectively, and was admitted to the hospital where intensive therapy for congestive heart failure and chronic obstructive pulmonary disease was initiated. The patient gradually improved and was discharged one week later.

On a follow-up visit one month later, the patient had no discernable adverse sequelae from nebulized tuberculin: 0.5 ml of tuberculin was administered intradermally at this time without any induration appearing at the site at 48 hrs. There was no history of tuberculous exposure or reaction to tuberculin.

Administration of tuberculin should be by the intradermal route. Inadvertent intravenous and intramuscular injection have been reported to cause no adverse effects (personal communication, E. W. Pearson). Tuberculin administered via the pulmonary route has not previously been described, to our knowledge. In a patient previously sensitized to tuberculin, placement of a PPD can result in a large local reaction. In our case, there was no known prior history of tuberculin reactivity. No adverse effect occurred, although one could speculate that a severe reaction may have occurred if the patient had been sensitized to tuberculin.

The substitution of tuberculin for terbutaline was done because of similar sounding names. As tuberculin doesn’t have known therapeutic effects in bronchospasm, not only did the patient not receive a therapeutic drug, but the potential for adverse effects from tuberculin existed. To prevent further mishaps such as this one, we recommend that “tuberculin PPD” be spelled out when reactivity to tuberculin needs to be assessed.

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REFERENCE

Flecainide-Induced Cardiogenic Shock

To the Editor:

The recent availability of several new orally active antiarrhythmic agents allows greater flexibility in managing patients with cardiac arrhythmias. One of these agents is flecainide (Tambocor, Riker), a class IC agent with relatively few serious noncardiac toxicities. Its potential, however, to adversely affect the cardiovascular system must not be overlooked. We would like to report our experience with a patient on flecainide therapy who developed cardiogenic shock. Risk factors for the development of cardiogenic shock other than the initiation of flecainide were not demonstrated in this patient.

An 82-year-old man was admitted to the CCU through the emergency room with cardiogenic shock. This patient had a history of prior myocardial infarction complicated by congestive heart failure and ventricular arrhythmias. One month prior to admission the patient developed sustained ventricular tachycardia requiring electrical cardioversion. The patient was placed on therapy with oral tocainide, 400 mg every 8 hrs. Three days prior to admission the patient presented with a maculopapular rash. Tocainide treatment was discontinued and flecainide (100 mg three times daily) was started. After initiation of flecainide therapy, the patient complained of nausea, dyspnea and fatigue. On the day of admission, the patient was found at home to be difficult to arouse, confused, cold and clammy. The patient was taken to his local emergency room and then was transferred by helicopter to our institution.

Examination on admission revealed an elderly man who was obtunded but easily arousable in acute respiratory distress. Systolic blood pressure was 70 mm Hg, diastolic was inaudible. The patient was intubated and a triple-lumen catheter placed in the right internal jugular vein. Initial hemodynamics were: cardiac index 1.3 L/min/m², pulmonary capillary wedge pressure 28 mm Hg; systemic vascular resistance 2,138 dynes·sec·cm⁻⁴; pulmonary vascular resistance 1,008 dynes·sec·cm⁻⁴; and mean arterial pressure 55 mm Hg. Bedside echocardiogram revealed an ejection fraction of less than 10 percent, four-chamber enlargement and global left ventricular dyskinesia. Dopamine and dobutamine were titrated to 10 and 40 μg/kg/min, respectively, in an attempt to improve hemodynamics, with little response. Nitroprusside therapy was initiated and titrated to 5 μg/kg/min. Serial electrocardiographic examinations and cardiac enzyme test results were negative for myocardial infarction. A flecainide blood level drawn on admission was 1,820 ng/ml (therapeutic range 300 to 700 ng/ml). The patient continued to deteriorate over the next 24 hrs and expired secondary to cardiogenic shock.

Flecainide is a new antiarrhythmic agent with few noncardiac toxicities. However, its potential to induce cardiac toxicity—as with any antiarrhythmic agent—is potentially great. Cardiac toxicity with flecainide includes a proarrhythmic effect, a negative inotropic action and conduction disturbances. The risk of cardiac toxicity is greatest in patients with hemodynamically unstable ventricular arrhythmias, left ventricular dysfunction, or with suprathapeutic dosages. In reviewing the safety data base collected by Riker Laboratories from February, 1980 to July, 1985, Morganoth et al observed an overall incidence of new or worsened congestive heart failure causing discontinuance of flecainide in 1.4 percent of patients. Overall, new or worsened congestive heart failure was observed in 5 percent of patients. Of the 1,330 patients evaluated, six (0.5 percent) died of congestive heart failure that may have been attributed to flecainide.

Our reported case of fatal cardiogenic shock in a patient receiving short-term oral flecainide is important as it underscores the importance of avoiding flecainide in patients with risk factors for toxicity. Our patient had a history of congestive heart failure and malignant ventricular arrhythmias. In addition, the initial flecainide dose of 100 mg three times daily in this case exceeds current recommendations of 100 mg twice daily. Actually, patients with congestive heart failure or liver or kidney dysfunction should be started on 50 mg every 12 hours. This is supported by our patient’s plasma flecainide concentration of 1,820 ng/ml (therapeutic range 300 to 700 ng/ml). Although flecainide’s negative inotropic effect occurs infrequently, our case highlights the potential severity and irreversibility of it. With appropriate patient selection and monitoring, flecainide can be used safely.

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