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REFERENCES

Adenosine-induced Torsades de Pointes*

Maj Gerald R. Harrington, MC, USA; and
Capt Edward G. Froelich, MC, USA

Physicians are finding increased applications for adenosine as a diagnostic and therapeutic modality for a variety of cardiac dysrhythmias. Its short half life and lack of reported major complications make it an ideal pharmacologic agent to utilize for diagnosis and treatment. Herein we report a case of polymorphic ventricular tachycardia induced by adenosine.

(Chest 1993; 103:1299-1301)

The use of adenosine as a diagnostic and therapeutic agent for supraventricular dysrhythmia has increased markedly since it was introduced into clinical practice. A potential complication from the use of adenosine may be found in patients with a prolonged QT interval. The atrioventricular block induced by adenosine may allow for the development of bradycardia-induced polymorphic ventricular tachycardia.

CASE REPORT

A 62-year-old man was taken to the operating room for treatment of a diverticular abscess involving the left iliopsoas muscle. Postoperatively, he demonstrated multiple episodes of atrial flutter and paroxysmal supraventricular tachycardia with heart rates up to 170 beats per minute. In order to control the recurrent episodes of supraventricular tachycardia, the patient eventually required a continuous infusion of procainamide, 2 mg/min, and esmolol, 50 µg/kg/min. Digitalis also was utilized both for rate control and positive inotropic effect. On the 60th postoperative day, the patient again manifested a supraventricular tachycardia with ischemic ECG changes. His elevated heart rate was not responsive to increased doses of esmolol. For diagnostic purposes, 6 mg of adenosine was administered by a central venous catheter. Approximately 10 s following the bolus injection, the patient developed the expected atrioventricular block which revealed the underlying mechanism to be atrial flutter (Fig 1). A normally conducted beat returned after a 6-s pause followed by several premature ventricular depolarizations. This initiated sustained polymorphic ventricular tachycardia. Sinus rhythm was restored via defibrillation with 360 J. At the time of the event, the serum potassium level measured 3.6 mEq/L; magnesium, 2.0 mg/dL; digoxin, 1.4 ng/ml; procainamide, 7.4 µg/ml; and N-acetyl procainamide, 19.5 µg/ml. The patient's corrected QT interval both prior to and following the event was prolonged to nearly 500 ms (Fig 2).

DISCUSSION

Adenosine recently was approved by the Food and Drug Administration for intravenous use in patients with paroxysmal supraventricular tachycardia. It is particularly effective in those patients whose supraventricular tachycardia is the result of atrioventricular reciprocating or atrioventricular nodal reentrant tachycardia. In many circumstances, it has supplanted verapamil as the treatment of choice for supraventricular dysrhythmia where the underlying electrophysiologic mechanism is unclear. This is due in part to its extremely short half-life, which is reported to be 10 s. A variety of relative contraindications to the use of verapamil have been reported. These include hypotension, congestive heart failure and prior intravenous beta blocker administration. Precipitation of cardiac arrest has been reported in patients treated with verapamil who subsequently were demonstrated to have preexcitation via an anomalous atrioventricular pathway. In addition, misdiagnosis of wide complex tachycardia as supraventricular in origin with aberrant conduction often leads to therapy with verapamil.

*From the Critical Care Medicine Service, Walter Reed Army Medical Center, Washington, DC.
The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or reflecting the views of the Department of the Army or the Department of Defense.
Reprint requests: Dr. Harrington, Critical Care Medicine, Walter Reed Army Medical Center, Washington, DC 20037

Figure 1. Rhythm strip 10 s after adenosine is administered.
Such treatment almost invariably results in hemodynamic deterioration. For these reasons, in conjunction with the high success rate and lack of reported major complications, many physicians utilize adenosine as their first-line agent for supraventricular tachycardia.

A recent editorial discussed the safety of adenosine and stated that there were no reported cases of hemodynamic collapse or cardiac arrest following the administration of adenosine. The Adenosine for Paroxysmal Supraventricular Tachycardia Study Group reported a 33 percent incidence of ventricular ectopic beats at or after termination of the tachycardia in patients given adenosine. Rankin et al stated that 44 percent incidence of ventricular extrasystoles in their series of patients treated with adenosine for sustained supraventricular tachycardia. In this patient, the ventricular tachycardia and subsequent degeneration into ventricular fibrillation resulting from adenosine is similar to that reported by Cohen in 1972. He reported a patient who developed ventricular tachycardia and ventricular fibrillation following carotid sinus pressure for treatment of paroxysmal supraventricular tachycardia. His patient had been treated with propranolol, quinidine, and digoxin with the propranolol and quinidine discontinued two days prior to the carotid sinus pressure. He hypothesized a number of factors which may have contributed to this event. In our case, the patient was receiving continuous intravenous beta blockers and procainamide therapy along with daily digoxin. Procainamide has been associated with the development of polymorphic ventricular tachycardia, although to a lesser degree than quinidine. It would appear that the premature ventricular depolarization occurred at a vulnerable period during repolarization of the ventricular myocardium. On this basis, patients with a prolonged QT interval from any cause would be at increased risk for this complication of adenosine therapy. Additionally, the issue of administration of adenosine via central venous catheter requires further study. It is likely that a far greater amount of the drug reaches the cardiac conduction system when this route of administration is utilized. In this setting, it may be advisable to halve the recommended dose from the initial 6 to 3 mg in order to avoid the potential for a more profound effect on atrioventricular conduction.

In summary, this case represents the first reported instance of cardiovascular collapse induced by adenosine. A potential electrophysiologic mechanism is proposed to explain this event.

Following submission of this case report, a brief report was published describing a similar occurrence in a patient with a prolonged Q-T interval syndrome who was treated with adenosine.

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Treatment of Pulmonary Aspergilloma by Endoscopic Intracavitary Instillation of Ketoconazole*

Randeep Guleria, M.D., D.M.; Dheeraj Gupta, M.D., D.M.; and Surinder K. Jindal, M.D., F.C.C.P.

The authors report a case of pulmonary aspergilloma in which a fungus ball was visualized and a biopsy specimen was obtained at fiberoptic bronchoscopy. The fungus ball was successfully treated with bronchoscopic instillation of ketoconazole. It appears that this approach can be useful in the treatment of patients who are high-risk candidates for pulmonary resection. (Chest 1993; 103:1301-02)

Pulmonary aspergilloma usually arises from colonization and proliferation of Aspergillus organisms in a preexisting cavity. A standard chest radiograph showing an intracavitary mass with an air-crescent sign is usually sufficient to make the diagnosis of mycetoma. Visualization of the mycetoma during fiberoptic bronchoscopy is extremely rare.

Hemoptysis is the cause of death in 2 to 24 percent of patients with pulmonary aspergilloma. Traditionally, surgical resection has been the treatment of choice in these patients, although morbidity and mortality rates may be as high as 25 and 8 percent, respectively. Antifungal agents have been used in a variety of ways. Several investigators have instilled antifungal agents endobronchially or via the transthoracic route into the cavity. We report a case of pulmonary aspergilloma in which the fungus ball was visualized on fiberoptic bronchoscopy and which was successfully treated by bronchoscopic instillation of ketoconazole into the cavity.

Case Report

A 34-year-old nonsmoking man presented with a history of recurrent, moderate to massive hemoptysis for 5 months. He had been treated at a private nursing home with blood transfusions and antibiotics. There was no history of fever, chest pain, breathlessness, or weight loss. Three years previously, on the basis of a chest radiograph, a diagnosis of pulmonary tuberculosis had been made, and the patient was treated with rifampicin, isoniazid, and ethambutol for 1 year. There was significant improvement with this treatment, and the patient had been asymptomatic until the present illness.

The patient had evidence of a left upper lobe fibrocavitary lesion. A chest radiograph (Fig 1) showed a left upper lobe cavity with fibrosis. A radiodense shadow was seen inside the cavity, and the air-crescent sign was present. The right upper lobe also showed evidence of fibrosis. Fiberoptic bronchoscopy revealed a yellowish mass protruding in the anterior segment of the left upper lobe. A biopsy specimen from the lesion showed clusters of fungal hyphae suggestive of aspergillosis. The sputum examination was negative for acid-fast bacilli, but showed both Aspergillus flavus and Candida albicans organisms. Fungal serology was positive for A flavus. While in the hospital, the patient was noticed to be jaundiced. Liver function tests revealed a serum bilirubin level of 4 mg/100 ml; SGOT and SGPT of 67 and 175 IU, respectively; and normal serum alkaline phosphatase. He was positive for hepatitis B surface antigen (HBsAg).

Six months later, he presented again, with recurrent streaky hemoptysis. His liver function test results were normal, and he was negative for HBsAg. Similar bronchoscopic findings were noted. During bronchoscopy, he was positioned in the left lateral position, and the bronchoscope was advanced to the opening of the cavity.

Ketoconazole, 400 mg mixed in 10 ml of normal saline solution, was slowly injected into the cavity through the bronchoscope. The bronchoscope was then withdrawn, and the patient was kept in the

*From the Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India.

FIGURE 1. Chest radiograph shows left upper lobe fibrocavitary lesion with a fungus ball (arrows).

FIGURE 2. Chest radiograph obtained after bronchoscopic instillation of ketoconazole shows complete disappearance of the fungus ball.