in standing. There were no noted focal neurologic deficits. The rest of his physical examination was unremarkable. Laboratory data revealed sodium 140 meq/L, potassium 4.0 meq/L, chloride 109 mmol/L, bicarbonate 27 mmol/L, blood urea nitrogen 25 mg/dL, creatinine 1.0 mg/dL, and glucose 108 mg/dL. The white blood cell count was 8.6 thou/mm^3, hemoglobin 14.8 g/dL, platelets 205 thou/mci. Troponin I was less than 0.4 ng/mL. Electrocardiogram revealed a normal sinus rhythm with old Q waves inferiorly and without peaked T waves. Creatine phosphokinase (CPK) revealed a level of 20,135 IU/L. CPK-MB fraction was 242 ng/mL, which was not elevated in relation to the total CPK. Urine dip was 4+ for blood with urine microscopic negative for red blood cells.

The patient was admitted and started on aggressive intravenous hydration. His simvastatin was stopped. Alkalinizing his urine was not performed because his renal function was adequate and hyperkalemia was not present. The patient was admitted for 5 days for intravenous hydration. At discharge, his CPK was 4421 with no evidence of renal failure. Follow-up outpatient visits revealed a normal CPK, normal kidney function, and a return to his baseline strength with absence of muscle spasms or pain.

Simvastatin is classed as a HMG-CoA reductase inhibitor. HMG-CoA acts as a catalyst in the early stages of cholesterol biosynthesis. The effect of decreasing cholesterol by the statins has a proven effect of decreasing future risks of coronary heart events and stroke. Some studies have shown a reduction in short-term recurrent ischemic events for patients presenting with acute coronary syndrome (ACS) who are started on a statin soon after admission.[3,4] Recent studies, however, caution its use in the acute setting of ACS unless the level of low-density lipids is known, citing that short- and long-term morbidity rates were not statistically significant.[5]

With today's statins, the most serious side effects are rare but do include rhabdomyolysis, as seen in our patient, as well as liver toxicity. Cerivastatin, a commonly prescribed statin in the past, was withdrawn from the market as a result of 31 reports of fatal rhabdomyolysis. Twelve of the 31 cases involved concomitant use of gemfibrozil.[6] Concomitant use of statins with systemic antifungals, macrolides, HIV protease inhibitors, nefazodone, and grapefruit juice is not recommended because it has been reported to increase the risk of myopathy. Reduced doses of simvastatin is suggested for patients taking cyclosporine, fibrates, or niacin.[7,8]

Although reports of statin-induced rhabdomyolysis have been frequently reported in the literature, our OVID and PUBMED search could not find any related reports or discussions in any EM journals. EM physicians frequently see patients whose medications include a statin. We feel that this case emphasizes that EM physicians are on the front line in diagnosing and treating conditions that are potentially iatrogenically drug-induced. EPs must maintain current knowledge of the many side effects and drug interactions of commonly prescribed medications, especially in the ever-increasing elderly population.

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References

1. 3rd Report of the National Cholesterol Education Program, National Institutes of Health. Publication no 01-3670, May 2001
2. Lemaitre RN, Psaty BM, Heckbert SR, et al: Therapy with hydroxymethylglutaryl coenzyme A reductase inhibitors (statins) and associated risk of incident cardiovascular events in older adults: evidence from the Cardiovascular Health Study. Arch Intern Med 2002;162:1395-1400
3. Schwartz GG, Olsson AG, Ezekowitz MD, et al: Effects of atorvastatin on early recurrent ischemic events in acute coronary syndromes: the MIRACL study. A randomized controlled trial. JAMA 2001;285:1711-1718
4. Acevedo M, Sprecher DL, Lauer MS, et al: Routine statin treatment after acute coronary syndromes? Am Heart J 2002;143: 940-942
5. Newby LK, Kristinsson A, Bhapkar MV, et al: Early statin initiation and outcomes in patients with acute coronary syndromes. JAMA 2002;287:3087-3095
6. Center for Drug Evaluation and Research FDA Talk Paper. Bayer Voluntarily withdraws Baycol. August 2001. Available at: www.fda.gov//bbs/topics/ANSWERS/2001/ANS01095.html. Accessed August 29, 2002
7. Product Information for Zocor Tablets. Whithouse Station, NJ: Merck & Co
8. Pasternak RC, Smith SC, Bairey-Merz CN, et al: ACC/AHA/NHLBI Clinical Advisory on the Use and Safety of Statins. Circulation 2002;106:1024-1028

BIDIRECTIONAL VENTRICULAR TACHYCARDIA RESULTING FROM DIGOXIN AND AMIODARONE TREATMENT OF RAPID ATRIAL FIBRILLATION

To the Editor:—In the current guidelines for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC),[1] amiodarone is highly recommended for treatment of supraventricular tachycardia (SVT) and ventricular tachycardia (VT), especially in patients with impaired left ventricular (LV) function. However, not all patients are suitable. We report a case with congestive heart failure (CHF) and new onset of atrial fibrillation (AF) with rapid ventricular response (RVR). Bidirectional VT attacked after digoxin and amiodarone were administered.

A 69-year-old man had shortness of breath and edema of bilateral legs for 1 month. Decompensated heart failure with pulmonary edema and respiratory failure happened on February 2, 2001. He was transferred to the ED of our tertiary medical center for further intensive care. On admission to our intensive-care unit, the patient was acutely ill-looking. The blood pressure was kept around 144/94 mm Hg under high doses of dopamine and norepinephrine intravenous infusion; pulse rate was 114 beats/min and respiratory rate was 26 breaths/min. Physically, jugular veins were engorged. Auscultation of the lungs revealed diffuse crackles. Heartbeats were regular without significant murmur. Extremities were cyanotic with pitting edema. The remainder of the physical examination was unremarkable. Serum potassium was abnormally high (6.1 mmol/L); blood urea nitrogen (BUN; 38.2 mg/dL) and creatinine (2.3 mg/dL) were both above the upper limits. The serum cardiac enzymes were all within normal limits.

Given the symptoms and signs of CHF with cardiogenic shock, he received continuous infusion of furosemide and dobutamine. As his condition improved gradually, inotropic agents were tapered and diuretics were discontinued. However, new onset of AF with a ventricular rate of approximately 140 beats/min was noted on the second day. Digoxin was used intravenously: 0.5 mg for loading, 0.25 mg twice with a 6-hour interval. Amiodarone was then infused intravenously for persistent AF with RVR with the dose of 1 mg/kg per hour for 6 hours then 0.5 mg/kg per hour. Sustained monomorphic VT was noted intermittently after 450-mg amiodarone infusion. At that time, amiodarone was not held for its potential to treat VT. Bidirectional VT without hypotension attacked after 17 hours and 690 mg amiodarone administration (Fig 1). Serum potassium was in the lower normal limit (3.5 mmol/L); the renal function was deteriorating with BUN 73.8 mg/dL and creatinine 3.04 mg/dL. The serum cardiac enzymes were all within normal limits. Infusion of amiodarone was stopped under the

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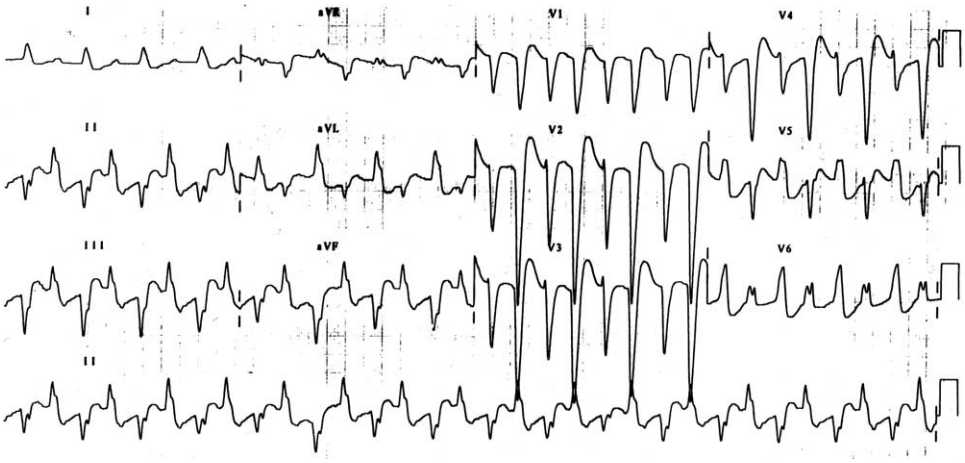

FIGURE 1. Bidirectional ventricular tachycardia after 1 mg digoxin and 690 mg amiodarone.

impression of digoxin intoxication and lidocaine was used. Besides, potassium was supplemented. Serum digoxin level was markedly elevated (4.33 ng/mL). After the treatments, normal sinus rhythm resumed 5 days later and the serum digoxin level was gradually down to 1.47 ng/mL. He was successfully weaned from the ventilator and transferred to the general ward.

Amiodarone is a complex drug with effects on sodium, potassium, and calcium channels as well as α- and β-adrenergic blocking properties. In the International Guidelines 2000 for CPR and ECC, amiodarone became the first choice for SVT and VT in patients with LV dysfunction and was addressed for AF unresponsive to digoxin.[1] In our patient with CHF, approximately 690 mg amiodarone was administered after 1 mg digoxin for treating new onset of AF. Sustained monomorphic VT occurred intermittently then and bidirectional VT associated with digoxin toxicity attacked 17 hours after addition of amiodarone. It returned to normal sinus rhythm after discontinuing digoxin and amiodarone and the addition of lidocaine.

Bidirectional VT is rare but classic arrhythmia of digitalis toxicity.[2] It was defined as VT with a regular rate and 2 QRS morphologies with opposite polarities.[3] The mechanism remains uncertain, and it could be infra-atrioventricular junctional in origin.[4] It is well known that amiodarone increases serum digoxin concentrations by inhibiting tubular secretion or displacing bound digoxin from tissue. The combination will increase the serum level of digoxin. As a result of increased cardiac automaticity, premature ventricular beats are often the earliest dysrhythmia,[5] and bidirectional VT are considered pathognomonic for digitalis toxicity.[6] In our patient, mild deterioration of renal function, relative hypokalemia and mainly, the addition of amiodarone, contributed to digoxin intoxication.

Amiodarone has been a star treatment for SVT and VT. However, it could lead to digitalis toxicity with previous or concomitant use of digoxin in patients with CHF. Amiodarone should be prescribed carefully in those patients.

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References

1. Cummins RO: Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2000;102: I112-I129
2. Nogrady S, Nicolic G: Bidirectional tachycardia. Heart Lung 1998;27:149-150
3. Wagner G: Morriott's Practical Electrocardiography, 9th ed. Baltimore: Williams and Wilkins, 1994
4. Cohen SI, Deisseroth A, Hecht HS: Infra-His bundle origin of bi-directional tachycardia. Circulation 1973;47:1260-1266
5. Ma G, Brady WJ, Pollack M, Chan TC: Electrocardiographic manifestations: digitalis toxicity. J Emerg Med 2001;20:145-152
6. Hauptman PJ, Kelly RA: Digitalis. Circulation 1999;99:1265-1270

RETROPERITONEAL HEMATOMA AS A COMPLICATION OF CORONARY ANGIOGRAPHY AND STENTING

To the Editor:—Retroperitoneal hematomas (RPH) are usually the result of trauma of the retroperitoneal organs such as the pancreas, duodenum, and kidneys. In elderly people and those with long-standing hypertension and atherosclerotic disease, they could also be a sign of a leaking abdominal aortic aneurysm (AAA).[1] Although most cases are associated with trauma or leakage of an AAA, they could also be caused by vascular procedures such as percutaneous transluminal coronary angioplasty (PTCA), cardiac catheterization, peripheral angioplasty, valvuloplasty, and peripheral arterial access catheterization.[1]

These hematomas are often difficult to diagnose and could masquerade as other retroperitoneal and intestinal pathology. The diagnosis is thus a challenge. We report a case of RPH in a middle-aged man, suffered after cardiac catheterization, to discuss possible misdiagnoses and to review this uncommon entity.

A 43 year-old man presented to the ED with vomiting for 8 hours. He awoke 9 hours prior with sudden onset of left groin/testicular pain. He took a standard over-the-counter dose of ibuprofen, which brought about minor relief. One hour after, the patient had several episodes of vomiting and was unable to tolerate anything by mouth. He denied any chest pain or shortness of breath, hematemesis, diarrhea, melena, or bright red blood per rectum. All other review of systems was negative. His medical history was significant for coronary artery disease, stent placement 7 years previously, and two coronary angiograms with stent placement 1 week ago. His regular medications were metoprolol, clopidrogel, atorvastatin, lisinopril, and aspirin. He had no known drug allergies and denied alcohol, tobacco, or illicit drug use.

On physical examination, the patient was pale, diaphoretic, and writhing in pain. Vital signs were all normal. The physical examination was normal except for the abdominal examination. On

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