rysms in Ippolito's study ruptured, yielding an 80% mortality rate.[3] Another study of eight gravid females who ruptured a renal artery aneurysm resulted in seven deaths.[4]

Initial examination of a patient with severe hematuria should start with a complete history, physical examination, and laboratory evaluation. History of recent operative procedure, trauma, coagulopathy, cancer, aneurysm, irradiation, or cyclophosphamide use are all concerning and should heighten your suspicion of a renal artery aneurysm.

The presentation of a patient with ruptured renal artery aneurysm is usually that of flank or abdominal pain associated with hematuria and a longstanding history of hypertension. Case reports have also demonstrated clot retention as a common presentation.[5,6] Physical examination could reveal an abdominal mass, abdominal bruit, and/or flank pain with palpation. Hypertension or hypotension could be seen on presentation.

Diagnosis of a renal artery aneurysm is becoming increasingly common. This growth is most likely the result of the increased availability and utilization of angiography in the diagnostic workup for hypertension. Delayed uptake and other abnormalities can be seen on an intravenous pyelogram in 60% of patients.[5-7] Emergent uroradiology is warranted in nontrauma patients if they have severe hematuria with shock or impending shock. Renal artery angiogram is the diagnostic modality of choice.[8] Bedside ultrasound can be a useful adjunct in the initial evaluation of these unstable patients. Plain radiographs are of limited use, because only approximately 12% of these aneurysms are calcified.[7]

Morphology and locality of this aneurysm could also differ. In a study by Bulbul of 56 patients with renal artery aneurysms, 62 were extrarenal and five were intrarenal. Seventy percent of the aneurysms were saccular, 22% were fusiform, and 8% were dissecting. Only one of these aneurysms ruptured, and this occurred in a pregnant patient.[7]

Treatment for renal artery aneurysm includes nephrectomy, partial nephrectomy, or ligation of the renal artery.[9] Rupture, expanding aneurysm, intractable hypertension, hematuria, and renal infarction represent the most common indications for surgical repair.[7] Confirmed renovascular hypertension and renal artery stenosis in the presence of an aneurysm are also indications for surgical intervention. Many experts also recommend surgery for renal artery aneurysms greater than 1 cm in diameter.[7] However, surgical indications for this entity are rapidly diminishing as transarterial embolization or ablation techniques are developed. Gelatin foam, coils, or alcohol are the first choices for ablation of the artery.[9]

Most cases of hematuria are not associated with acute life-threatening sequela. Patients with moderate-to-severe hematuria can be life-threatening like in this case of ruptured renal artery aneurysm. Aggressive diagnostic testing needs to be used when this pathologic entity is considered.

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SIMVASTATIN-INDUCED RHABDOMYOLYSIS

To the Editor:—The National Health and Nutrition Examination Survey III (NHANES III) completed in 1994 estimated that 102.3 million American adults had total blood cholesterol values of 200 mg/dL and higher. Elevated cholesterol levels have been shown to be a major cause of coronary heart disease. The National Cholesterol Education Program (NCEP) strongly recommends that cholesterol-lowering medications along with environmental changes be used in an effort to prevent coronary heart events in patients who have elevated cholesterol levels.[1] Lemaitre et al. recently confirmed the beneficial effect of the hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, also know as the statins, in decreasing cardiovascular events.[2] Increased prescribing of the statins have required that physicians be more aware of their side effects and drug interactions. Our case reports on a patient with a statin-induced rhabdomyolysis.

An 82-year-old man presented to our emergency department with bilateral leg weakness and muscle cramping for approximately 2 weeks. He stated that he had trouble walking short distances and also standing up for prolonged periods of time. The patient also reported increased dyspnea on exertion, more so than his usual baseline. He denied any chest pain, nausea, vomiting, fevers, chills, headaches, or upper respiratory infection symptoms. He denied any focal weakness or slurred speech. Also, he denied any history of falls or trauma. The patient had been started on simvastatin approximately 4 weeks previously and stated that he had been seen 1 week before at an urgent care facility with muscle spasms.

The patient's medical history included coronary artery disease, early Alzheimer's dementia, osteoarthritis, cerebrovascular accident (CVA), and benign prostate hypertrophy. He had no neurologic deficits from his prior CVA. Ten years previously, he had coronary artery bypass graft surgery. His medications were donepezil, fluoxetine, alprazolam, tamsulosin, aspirin, atenolol, and simvastatin. He had no known drug allergies. He lived with his wife and denied any alcohol or tobacco use.

On physical examination, his vitals were an oral temperature of 37.2°F, heart rate of 73 beats/min, respiratory rate 18 breaths/min, and blood pressure of 136/69 mm Hg. There were no orthostatic changes. Neurologic examination revealed that he had mild memory loss secondary to his Alzheimer's. He also had diffuse weakness going from a lying to a sitting position and needed assistance

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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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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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