metabolic clearance and distribution of the parent drug. They also suggest that if pill bezoar formation occurred, that WBI (administered here as MDAC + sorbitol) could have further decreased drug absorption. Unfortunately, this conclusion is entirely speculative. There is not enough experience with bupropion overdose to fully understand the toxicokinetics of the drug let alone the efficacy of treatment.

We do have some data regarding the pharmacokinetics of bupropion. Therapeutic bupropion is eliminated in a biphasic manner.[9] After 6 hours, the bupropion plasma level is 30% of the peak plasma level. In the terminal phase, the half-life changes to an average of 14 hours (range, 8-24 hours). In one case of fatal bupropion overdose, there was a plasma level obtained at 18 hours postpresentation of 446 ng/mL.[10] The level 13 hours later was 135 ng/mL, making the elimination half-life 7.5 hours. Unfortunately, it is difficult to compare this with the current case. In the case by White, there are also only two levels, one at 3 hours and one at 24 hours postingestion. In therapeutic dosing, the peak level would be obtained at 3 hours.[9] It is unclear when the peak would occur in overdose. Additionally, to attempt to assess the elimination half-life without multiple levels, without knowledge of time to distribution and the onset of the elimination phase is not valid.

We suggest that when future cases of bupropion overdose arise, that multiple samples be sent for analysis so that true toxicokinetic data can become available. Once we have this information, than we can begin a discussion of efficacy of therapy.

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RUPTURED RENAL ARTERY ANEURYSM PRESENTING AS HEMATURIA

To the Editor:—The differential diagnosis for hematuria is quite extensive and could be clinically challenging. Diagnostic entities include infection, renal colic, glomerulonephritis, fulminating renal papillary necrosis, tumors, benign prosthetic hypertrophy, erosion of an aortic aneurysm, and trauma. Another disease associated with hematuria, ruptured renal artery aneurysm, is extremely rare, but carries a significant risk of morbidity and mortality, if misdiagnosed. Patients with these aneurysms are usually asymptomatic until rupture. More frequently, they are observed in postsurgical, hypertensive, or gravid patients. We describe an unusual case involving a ruptured renal artery aneurysm that presents with hematuria.

A 52-year-old man presented to the ED with urinary retention, suprapubic discomfort, and hematuria of several hours' duration. He also reported intermittent chills beginning approximately 6 hours before arrival. The patient was in his "normal state of health" before this event. His medical history included untreated hypertension. There was no history of bleeding disorders, recent surgeries, trauma, or episodes of urinary problems.

The patient was in moderate distress with a normal mental status. His vital signs were temperature, 37.6°C, heart rate 75 beats/min, respiratory rate 20 breath/min, and blood pressure 190/108 mm Hg. He had a normal cardiopulmonary examination. There were no purpura or petechiae on mucosa or skin. On abdominal examination, there was suprapubic tenderness and a palpable bladder. There was no flank tenderness and the remainder of his examination was normal.

A Foley catheter was inserted and 1 L of bloody urine with small clots drained immediately with resolution of symptoms. His gross hematuria cleared after lavage with normal saline irrigation (500 cc). The patient's complete blood count, electrolytes, and prothrombin time/partial thromboplastin time were all normal. Urine microscopy revealed: white blood cell count 5-10 per high-power field, red blood cells 10-20 per high-power field, and no squamous epithelial cells.

The patient was discharged home with the Foley catheter to a leg bag and oral ciprofloxacin.

Six hours after ED discharge, the patient experienced severe right upper quadrant and right flank pain. This was accompanied by a syncopal episode. The patient's blood pressure on ED return was 70/32 mm Hg. On physical examination, a large ecchymotic area was noted over his right flank. The patient was administered 2.5 L of normal saline and transfused with 2 units of packed red blood cells, resulting in stabilization of his blood pressure. A bedside ultrasound demonstrated a large fluid collection around the right kidney, no hydronephrosis, and no free fluid in the abdomen. A spiral computed tomography (CT) scan of the abdomen revealed a massive right perinephric hematoma, which had extended into the anterior retroperitoneum and continued caudally into the pelvis (Fig 1). These findings were consistent with a mass in the anteromedial portion of the midpole of the right kidney.

The patient subsequently underwent an abdominal aortogram, which revealed a right renal artery aneurysm with associated atrioventricular fistula. Successful coil embolization of the proximal right renal artery was performed (Fig 2).

The patient's hospitalization was complicated by a myocardial infarction, presumed secondary to his blood loss. The patient

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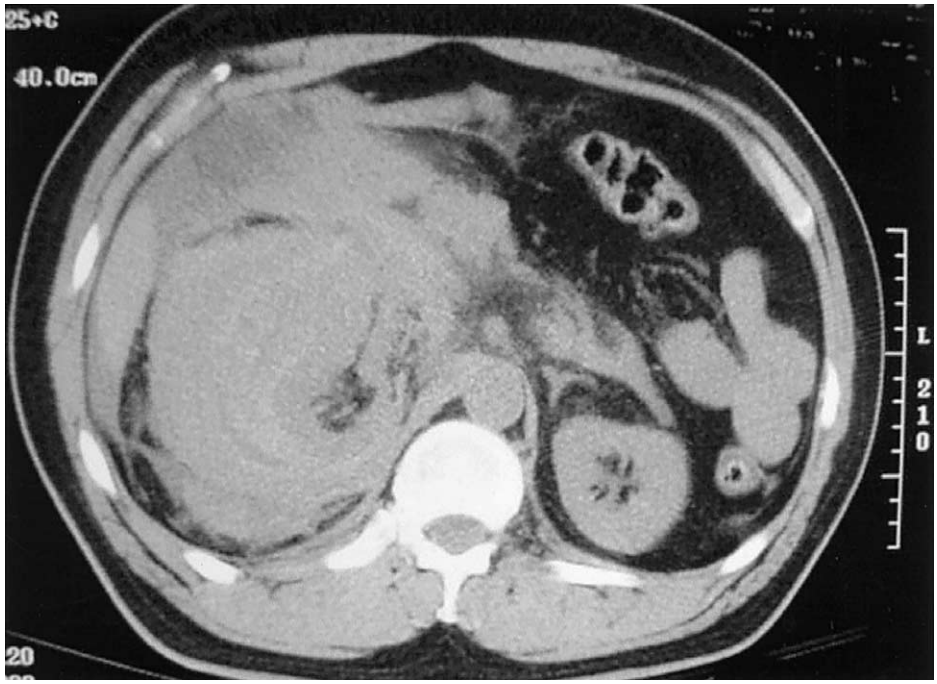

FIGURE 1. Ruptured right renal artery aneurysm with surrounding hematoma.

underwent cardiac catheterization that showed no obvious coronary artery vascular disease. He was discharged home 5 days after admission.

Previously published information regarding ruptured renal artery aneurysms is limited. One of the first cases described was reported in 1928 by Varela.[1] The incidence of this type of aneurysm varies from 0.3% to 0.7% on angiography.[2] According to Gunner's study of 83 people with a nonsymptomatic renal artery aneurysm, it was found that the majority could be treated conservatively. Observations demonstrate that these aneurysms occur more frequently on the right, have a predilection for males, and occur in patients with a mean age of 60 years.[2] In a report of 36,656 autopsies of patients with sudden, unexplained deaths in Sweden between 1970 and 1979, 19 ruptured arterial aneurysms were found as the cause of death. Of these, 12 were within the iliac arteries, five originated in the splenic artery, and one was within the hepatic artery. None were found to have originated in the renal arteries.[2]

Like with all aneurysms, there is a potential for rupture and mortality. Twenty-four of 169 patients with noncalcified aneu-

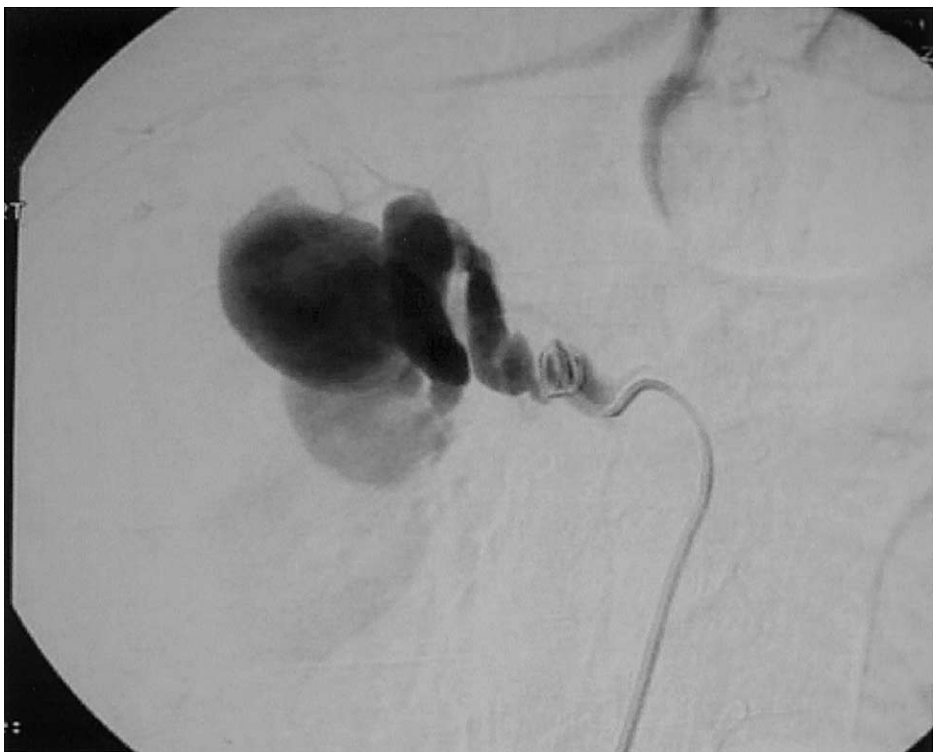

FIGURE 2. Angiographic insertion of right renal artery coil.

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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