(mean corpuscular volume: 67.3 μm^3). Renal and liver functions were within the normal range and the inflammatory index level (C-reactive protein) was not elevated. The admission course was uneventful and the abdominal pain resolved after conservative treatment.

The differential diagnosis between life-threatening diseases and benign lesions in the ED remains a challenge to both radiologists and EPs. The retroperitonium is relatively inaccessible to conventional roentgenologic imaging techniques. Many radiographic signs of retroperitoneal lesions depend on indirect evidence such as displacement of the ureter or alternation of the psoas margin.[1] The appearance of the psoas margin varies according to local pathologic conditions such as hematoma, abscess, and retroperitoneal tumors.[2,3] However, obliteration of the psoas shadow is not a reliable indicator of a pathogenic process; some benign variants could mimic acute abdominal conditions.[4] This patient's tumor-like lesion was brought to our attention only because of a delineated shadow on his KUB radiograph. The massive psoas muscles were not large enough to indent the patient's bowel or be accompanied by acute urinary retention,[5] and some of the patient's gastrointestinal problems could have been coincidental. The patient's history was remarkable only for lifting dumbbells in a body-building program designed to build up his back and trunk muscles immediately before hospitalization.

The psoas muscle is a major flexor of the hip. It stabilizes the lumbar spine while standing and walking, and it contributes to compressive and shear forces at the lumbar intervertebral discs during specific exercises. It reaches its maximum cross-sectional areas (CSAs) at the L_4/L_5 level. This is why patients with bowel displacement or hydroureter by a hypertrophic psoas is always at this level. Our patient demonstrates a similar clinical presentation, except for symmetric CSAs over the L_3/L_4 level, and is the youngest subject reported in the literature.

Hypertrophy of the psoas muscles is a benign anatomic variant that could be recognized easily on a KUB image and might need an abdominal CT to confirm the normal entity. A CT scan is an effective screening modality because it provides not only an etiologic explanation of retroperitoneal lesion, but also because it directs appropriate clinical management. However, the cost and effectiveness of a CT scan for diagnosing an acute abdomen in a pediatric ED seems to be another challenging issue.[6] More evidence is needed to establish a clinical algorithm that allows ED physicians to avoid a costly and time-consuming workup for a benign condition. With physical fitness becoming a national trend, it is important to recognize the more common entity we described. A detailed history and a careful examination continue to play important roles in diagnosing this normal variant.

CHIA-CHANG CHUANG, MD
Department of Emergency Medicine
Institute of Clinical Medicine
National Cheng Kung University Hospital
and College of Medicine
Taiwan, R.O.C.
MING-CHE TSAI, MD
WEN-CHAU CHEN, MD
Department of Emergency Medicine
National Cheng Kung University Hospital and
College of Medicine
Taiwan, R.O.C.
SHIH-MIN WANG, MD
Departments of Emergency Medicine and Pediatrics
National Cheng Kung University Hospital and
College of Medicine
Taiwan, R.O.C.
CHIN-YIN YU, MD
Department of Radiology
National Cheng Kung University Hospital and
College of Medicine
Taiwan, R.O.C.

References

1. Ralls PW, Boswell W, Henderson R, et al: CT of inflammatory disease of the psoas muscle. Am J Radiol 1980;31:767-770
2. Feldberg MAM, Koehler PR, Van Waes PFGM: Psoas compartment disease studied by computed tomography. Radiology 1983;148:505-512
3. Stephens DH, Sheedy PF, Robert RH, et al: Diagnosis and evaluation of retroperitoneal tumors by computed tomography. Am J Radiol 1977;129:395-402
4. Zeiss J, Smith RR, Taha AM: Iliopsoas hypertrophy mimicking acute abdomen in a bodybuilder. Gastrointest Radiol 1987;12:340-342
5. Frank RG: Retroperitoneal mass and hydronephrosis. Urology 2000;55:141-142
6. Garcia Pena BM, Taylor GA, Fishman SJ, et al: Costs and effectiveness of ultrasonography and limited computed tomography for diagnosing appendicitis in children. Pediatrics 2000;106:672-676

GASTRIC DECONTAMINATION, ENHANCED ELIMINATION, AND TOXICOKINETICS IN A SUSTAINED-RELEASE BUPROPION OVERDOSE

To the Editor:—We appreciated the letter describing the overdose of sustained-release bupropion by White et al.[1] This is an uncommonly seen overdose and all experience with this ingestion is valuable. We want to comment on the interpretation of treatment, recommendations, and laboratory analysis.

Multiple dosing of activated charcoal (MDAC) has been described to be an effective treatment for five medications (theophylline, phenobarbital, carbamazepine, dapsone, and quinine). This is according to the consensus statement by the American Academy of Clinical Toxicologists and European Association of Association of Poison Centers.[2] This is typically recommended to be done as an initial dose of activated charcoal (AC) at 1 g/kg with a cathartic (eg, sorbitol), followed by ½ g AC without a cathartic approximately every 4 hours. Most authors agree that only one dose of cathartic should be given within a 24-hour period. In the case report by White, four additional doses of 30 g of AC with sorbitol were given at 6-hour intervals. The risk of multiple dosing of a cathartic is developing electrolyte abnormalities (hypernatremia, hypermagnesemia, dehydration) and potentially causing seizures.[3,4] According to the position statement of the American Academy of Clinical Toxicologists and European Association of Association of Poison Centers, the addition of a cathartic to AC has not been demonstrated to improve outcome in a poisoned patient.[5]

There is a problem with the use of the phrase "whole bowel irrigation" (WBI) by the authors. This procedure is generally carried out with the use of a polyethylene glycol electrolyte lavage solution (PEG-ELS). The goal is to speed the transit of a toxin through the gut before it is absorbed. It is typically administered at a rate of 1 to 2 L per hour in an adult. MDAC with sorbitol should not be confused with WBI with PEG-ELS. The latter treatment has been demonstrated to be safe and does not cause electrolyte imbalance as could the multiple dosing of sorbitol. WBI has been shown to decrease the area under the curve in volunteers ingesting a delayed release preparation of lithium.[6] It has also been speculated to be effective in the setting of sustained-release preparation overdose.[7] Another author has also suggested its use in sustained-release bupropion overdose, but its efficacy remains unproven.[8]

The current case also does not prove efficacy. In this case, two levels were obtained. The 3-hour post-ingestion level was 2200 ng/mL and a level obtained at 24 hours postingestion was 57 ng/mL. The authors speculate that the difference between plasma levels seems greater than one would expect based on a normal

0735-6757/04/2203-0023$30.00/0
doi:10.1016/j.ajem.2004.02.038

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.

KIRK L. CUMPSTON, DO
Department of Emergency Medicine
School of Medicine
School of Pharmacy
University of New Mexico
New Mexico Poison Center
Albuquerque, NM
SEAN M. BRYANT, MD
STEVE E. AKS, DO
Toxikon Consortium
Department of Emergency Medicine
Cook County Hospital
Department of Emergency Medicine
University of Illinois at Chicago
Chicago, IL

References

1. White RS, Langford JR: Sustained release bupropion: overdose and treatment. Am J Emerg Med 2002;20:388-389
2. Position statement and practice guidelines on the use of multidose activated charcoal in the treatment of acute poisoning. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. J Toxicol Clin Toxicol 1999; 37:731-751
3. Allerton JP, Strom JA: Hypernatremia due to repeated doses of charcoal-sorbitol. Am J Kidney Dis 1991;17:581-584
4. Farley TA: Severe hypernatremic dehydration after use of an activated charcoal- sorbitol suspension. J Pediatr 1986;109:719-722
5. Barceloux D, McGuigan M, Hartigan-Go K: Position statement: cathartics. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. J Toxicol Clin Toxicol 1997;35:743-752
6. Smith SW, Ling LJ, Halstenson CE: Whole-bowel irrigation as a treatment for acute lithium overdose. Ann Emerg Med 1991;20: 536-539
7. Tenebein M: Position statement: whole bowel irrigation. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. J Toxicol Clin Toxicol 1997; 35:753-762
8. Sigg T: Recurrent seizures form sustained release bupropion. J Toxicol Clin Toxicol 1999;37:634
9. McEnvoy GK, ed: AHFS Drug Information. 2001:2152-2161
10. Harris CR, Gaultieri J, Stark G: Fatal bupropion overdose. J Toxicol Clin Toxicol 1997;35:321-324

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

0735-6757/04/2203-0024$30.00/0
doi:10.1016/j.ajem.2004.02.037