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Chad Hood, Joe L. Lezama, Rajani P. Shah, Harold M. Adelman, Edward P. Cutolo, Bryan A. Bognar & Charlotte A. Truitt

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An Unresponsive Patient with a History of Stroke

CHAD HOOD JOE L. LEZAMA RAJANI P. SHAH HAROLD M. ADELMAN

University of South Florida, James A. Haley Veterans Hospital, Tampa

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Dr. Adelman is Professor, and Drs. Cutolo, Bognar, and Shah are Assistant Professors, Department of Internal Medicine, University of South Florida College of Medicine, Tampa. Dr. Adelman is also Assistant Chief, Drs. Cutolo and Shah are Hospitalists, Medical Service, and Ms. Truitt is Clinical Medical Librarian, James A. Haley Veterans Hospital, Tampa. In addition, Dr. Bognar is Codirector of Medical Clerkship at the university and Attending Physician, Tampa General Hospital. This case was presented by Dr. Hood, Medical Resident, and moderated by Dr. Lezama. Chief Medical Resident. Veterans Hospital.

75-year-old woman was brought by her family for evaluation of her mental status, which had begun to deteriorate several weeks earlier. For the last two days, she had been unresponsive and had had a low-grade fever (≤37.8°C).

MEDICAL HISTORY. Seizures and dementia, beginning after a stroke four years ago; parkinsonism; chronic obstructive pulmonary disease; coronary artery disease with angina pectoris; hypertension; allergy to levofloxacin.

MEDICATIONS. Phenytoin, carbidodopa/levodopa, paroxetine, albuterol, atenolol, aspirin. (The family was unaware of any recent change in regimen.)

FAMILY HISTORY. Noncontributory.

SOCIAL HISTORY. Married; lives at home with her husband; smokes and drinks socially.

PHYSICAL EXAMINATION. Well developed, well nourished; minimally responsive. Vital signs: blood pressure, 125/72 mm Hg; pulse, 88 bpm; temperature, 36.5°C; respirations, 22/min; SpO₂ on room air, 92%. Eyes: pupils dilated, unresponsive

to light; extraocular muscles could not be tested. Neck: supple. Breasts: unremarkable. Heart: regular rate and rhythm; no murmur or gallop. Lungs: clear to percussion and auscultation. Abdomen: normal bowel sounds; soft, nontender; no masses or hepatosplenomegaly. Pelvis: unremarkable. Rectum: no masses; hard guaiac-negative stool. Extremities: normal. Neurologic responses: cogwheel rigidity bilaterally; 2+ deep tendon reflexes bilaterally; Babinski sign bilaterally.

LABORATORY FINDINGS. Hemoglobin, 8.4 gm/dL (normal, 14-17); mean corpuscular volume, 109 μ m³ (80-100); white blood cell count, 4,200/mm³; platelet count, 75,000/mm³ (150,000-410,000); Serum: sodium, 131 mEq/L (136-145); potassium, 4.4 mEq/L; chloride, 99 mEq/L; CO₂, 21 mEq/L; blood urea nitrogen, 25 mg/dL; creatinine, 0.9 mg/dL; glucose, 220 mg/dL (65-110); total protein, 6.1 gm/dL (6.3-8.2), albumin, 2.7 gm/dL (3.8-4.8), liver enzymes, within normal limits; calcium, 8.2 mg/dL. Urine: average of 35 white and 24 red blood cells per high powered field; ketones, present; few bacteria.

piagnostic imaging. Chest x-ray: normal. Electrocardiogram: junctional rhythm, nonspecific ST and T wave changes (same as earlier ECG). Computed tomography of the head: cortical atrophy, ventricular prominence, right parietal infarct.

DIFFERENTIAL DIAGNOSIS. Stroke; bilateral subdural hematoma; decompensated parkinsonism, phenytoin toxicity; urinary tract infection or constipation causing changes in mental status.

HOSPITAL COURSE. Additional laboratory tests revealed a plasma phenytoin level of 102 µg/mL (adjusted for albumin binding). Once the drug was withdrawn, the level rapidly returned to the normal therapeutic level of less than 20 µg/mL, and the patient's mental status improved. Her prescription for phenytoin was changed from 125 mg to tid to 125 mg bid with careful monitoring. She was also started on an antibiotic after urine cultures grew enterococci. A problem with constipation was resolved by disimpaction, and she was discharged on day 8.

FINAL DIAGNOSIS. Phenytoin toxicity, urinary tract infection, fecal impaction.

OUTCOME. At the two-month follow-up examination, the patient appeared to be doing well. The reason for the apparent overdose was not determined. She and her family were reshown how to administer the medication properly.

Suspicion of phenytoin intoxication and subsequent confirmation by laboratory testing.

1. The antiepileptic drug phenytoin has a notoriously high risk/benefit ratio. At serum levels of 30 mg/mL, the drug is toxic in up to 50% of patients.

- 2. The first sign of toxicity is usually nystagmus, which manifests at about 20 µg/mL. Cerebellar ataxia, tremor, and hyperreflexia occur at about 30 µg/mL, and confusion, lethargy, and coma at 40 µg/mL and higher.
- 3. Many authorities advise maintaining a serum level of 14 to $18 \mu g/mL$. Measurements must be adjusted for albumin binding. The adjusted value is calculated according to the following formula: phenytoin concentration/ $(0.2 \times albumin concentration) + 0.1$.
- 4. Drugs known to increase the serum level of phenytoin include amioderone, cimetidine, fluconazole, isoniazid, and certain sulfonamides. The effects of alcohol are variable.
- 5. Hyperglycemia occasionally occurs even with careful monitoring of phenytoin levels. The mechanism is believed to be postreceptor inhibition of insulin action.
 - 6. Other effects of phenytoin

include interference with folic acid absorption and stimulation of folate metabolism by enzyme induction, which leads to megaloblastic anemia or pancytopenia in a small percentage of patients (<1%). Either condition can be corrected with 1 mg/day of folic acid, but the serum level of phenytoin will be lowered.

- 7. A least 25% of patients taking phenytoin have mild macrocytosis without anemia.
- 8. Phenytoin overdose is generally treated by drug withdrawal and gastrointestinal decontamination by gastric lavage and repeated administration of an activated charcoal slurry. Hemodialysis and hemoperfusion are ineffective.
- 9. Deaths from phenytoin toxicity are rare. □

SELECTED READING

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