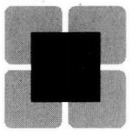

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Postoperative confusion in a 42-year-old man

A 42-YEAR-OLD MAN was admitted through the emergency department with a hip fracture sustained in a motor vehicle accident in which he was the driver. He underwent open reduction and internal fixation of the hip the evening of his admission. Two days later, you are consulted because he has become confused.

During the initial examination in the emergency department, which was unremarkable except for the hip fracture, his mental status had been clear. The medical history was notable for hypertension, dyspepsia, and a fractured clavicle sustained in a motor vehicle accident 3 years previously. Medications on admission included atenolol 50 mg daily and cimetidine as needed. Medications at the time of consultation include morphine 2 mg intramuscularly every 4 hours as needed and cimetidine 300 mg twice a day. Pneumatic compression stockings were ordered the evening of admission and continued after surgery.

Physical examination reveals an agitated, disoriented man, mumbling and unaware of his surroundings. The blood pressure is 140/100 mm Hg, pulse 110 beats per minute, and respiratory rate 20 per minute. The lungs and heart are normal. There is mild right upper quadrant tenderness but no hepatomegaly. The stools are negative for occult blood. The neurologic examination reveals a symmetric tremor in the upper extremities and diffusely brisk reflexes. The remainder of the examination is normal. Results of laboratory studies are shown in the *Table*; other results not shown were normal.

1 What is the most likely cause of this patient's confusion?

- ❑ Beta-blocker withdrawal
- ❑ Alcohol withdrawal
- ❑ Morphine intoxication
- ❑ Fat emboli
- ❑ Hepatic encephalopathy

TABLE
RESULTS OF LABORATORY STUDIES

Study	Day of admission	Day of consult
Hemoglobin, g/dL	14.2	11.4
Hematocrit, %	43	35
Mean corpuscular volume, fL	100	97
White blood cell count, $\times 10^9$/L	9.6	11.2
Platelet count, $\times 10^9$/L	310	400
Gamma glutamyltransferase, U/L	74	—
Coagulation profile	Normal	—
Sodium, mEq/L	136	134
Potassium, mEq/L	3.5	3.3
Glucose, mg/dL	69	72
Blood urea nitrogen, mg/dL	21	16
Creatinine, mg/dL	1.4	1.2
Urinalysis	Normal	Normal
PaO_2 (room air), mm Hg	—	80
$PaCO_2$ (room air), mm Hg	—	42
pH	—	7.36

The development of confusion on the second day in the hospital is compatible with alcohol withdrawal. Several studies have shown that alcoholism is present in 20% to 40% of patients in general medical-surgical units. Several clues point to a chronic alcohol problem in this man. Hypertension and dyspepsia are common early medical complications of alcohol use. An elevated gamma glutamyltransferase concentration and mean corpuscular volume are also suggestive of heavy alcohol use.

Beta-blocker withdrawal might cause sympathetic hyperactivity, but not confusion. Morphine intoxication would likely cause sedation rather than agitation and confusion. Fat emboli typically occur in long-bone fractures and would be associated with respiratory distress and fat globules in the urine. Hepatic encephalopathy is a late manifestation of liver failure, which is not seen in this patient.

2 Which of the following is *not* a treatment option?

- ❑ Parenteral metoprolol
- ❑ Transfer to an intensive care unit
- ❑ A parenteral benzodiazepine
- ❑ Parenteral haloperidol

Alcohol withdrawal can progress through mild, moderate, and severe stages. Mild withdrawal can begin within hours of the last drink and manifests as anxiety, insomnia, and, often, a mild tremor. Because these symptoms also occur for a variety of other reasons in hospitalized patients, mild alcohol withdrawal is often not recognized.

Hospitalized patients, without access to alcohol, often progress to moderate withdrawal, showing increasing anxiety, agitation, tremor, and signs of autonomic instability such as increased heart rate and blood pressure. Typically, this stage appears 18 to 24 hours after the last drink and resolves in 3 to 5 days. Appropriate treatment with benzodiazepines at this point may prevent severe withdrawal, ie, delirium tremens (DTs).

Severe withdrawal is potentially life-threatening, but fortunately occurs in only a small percentage of patients with alcohol problems. The patient is at greatest risk 2 to 3 days after the last drink and typically has all of the symptoms of moderate withdrawal with the addition of delirium. Hallucinations often occur, but are not always present. Seizures, if they occur in the course of withdrawal, usually precede DTs.

Treatment of alcohol withdrawal is stage-specific. All patients should receive thiamine 50 mg intramuscularly. Benzodiazepines are the sedatives of choice and decrease the likelihood of severe withdrawal. Patients with moderate withdrawal symptoms should receive oral therapy; those with severe withdrawal need parenteral (IV) therapy.

This patient is in severe alcohol withdrawal and should be transferred to a hospital unit with more intensive nursing care. His treatment should include parenteral administration of benzodiazepines. Haloperidol is relatively contraindicated because of the possibility of lowering the seizure threshold. Beta-blockers are not primary therapy for alcohol withdrawal, but may be useful as secondary treatment of sympathetic hyperactivity. In this case, parenteral metoprolol would be reasonable, especially if the patient had not received a beta-blocker since admission.

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SUGGESTED READING

Barnes HN, Aronson MD, Delbanco TL, editors. Alcoholism: a guide for the primary care physician. New York, Springer-Verlag, 1987.

Clark WD. Alcoholism. In: Noble J, editor. Textbook of general medicine and primary care. New York, Little, Brown and Company, 1987:1619–1644.

Kraus ML, Gottlieb LD, Horwitz RI, Anscher M. Randomized clinical trial of atenolol in patients with alcohol withdrawal. N Engl J Med 1985; 313:905–909.

Moore RD, Bone LR, Geller G, Mamon JA, Stokes EJ. Prevalence, detection and treatment of alcoholism in hospitalized patients. JAMA 1989; 261:403–407.