Intentional Ingestion of Ethanol-Based Hand Sanitizer by a Hospitalized Patient With Alcoholism

To the Editor: Alcohol-based hand sanitizers containing 60% to 95% ethanol or isopropanol are ubiquitous in the health care setting. These preparations prevent pathogen transmission more effectively than hand washing. As such, the Centers for Disease Control and Prevention and the Joint Commission on Accreditation of Healthcare Organizations endorse use of these agents to decontaminate hands that are not visibly soiled.[1,2] Nevertheless, the presence of these agents in hospitals may create some hazards. Herein, we report a case of ingestion of ethanol-based hand sanitizer in a patient with alcoholism who was hospitalized for isopropanol intoxication.

Report of a Case. A 53-year-old man with alcoholism was found unresponsive outside our hospital and brought to our emergency department. His vital signs were normal, but he had signs of intoxication including slurred speech, somnolence, confusion, and unsteady gait. He was treated with intravenous fluids, thiamine, folic acid, magnesium, and glucose. A serum volatile alcohol screen revealed isopropanol (1002 μg/mL) and its metabolite acetone (2071 μg/mL), but was negative for ethanol and methanol (Table 1). A urine drug screen was negative. The patient's mental status gradually improved, and he was admitted to a general medical unit. There, he was alert but oriented only to person, not to place and time. His vital signs and physical examination were otherwise unremarkable. The isopropanol intoxication was managed with intravenous fluids, and he was monitored using continuous one-to-one nursing. After his mental status improved, he eventually disclosed that he had consumed a bottle of rubbing alcohol (isopropanol) before admission.

During the morning of the second day of hospitalization, the patient was examined in his room by the medical team. He was found to be alert and oriented to person, place, and time. The patient then ate breakfast, used the bathroom once, and fell asleep. Approximately 45 minutes later, a nurse examined him and found that he could not be awakened. The primary team was emergently called to the bedside. The patient's vital signs were normal, but he was unresponsive to sternal rub. Naltrexone and flumazenil were administered intravenously with no effect. The patient was transferred to the intensive care unit for further treatment.

In the intensive care unit, a second serum volatile alcohol screen was immediately performed, revealing a high level of ethanol (3759 μg/mL). Notably, isopropanol and acetone levels were lower than those at the time of admission. A urine drug screen was positive for ethanol (Table 1). The patient was treated conservatively and improved. On the third day of hospitalization, he admitted to ingesting the contents of a 500-mL pump bottle of ethanol-based hand sanitizer that was attached to the wall of his hospital room (based on his weight [80 kg] and his blood ethanol level, the patient consumed approximately 450 mL of the product[3]). He remarked, "It had a horrible taste, but I was drunk pretty quick."

Discussion. Cases of intoxication caused by ingestion of an ethanol-based hand sanitizer by a prisoner[4] and an isopropanol-based hand sanitizer by a hospitalized patient[5] have been reported previously. Here we report a novel case of a patient with alcoholism who was initially admitted with isopropanol intoxication due to the consumption of rubbing alcohol outside the hospital and who then consumed ethanol-based hand sanitizer while in the hospital. Notably, accidental and intentional ingestion of alcohol-based hand sanitizer by children and teenagers has been reported in the media, suggesting that public awareness of the high alcohol content of these products and the potential for their misuse are growing.[6]

The hand sanitizer used at our institution is Avagard D Instant Hand Antiseptic with Moisturizers (3M, St Paul, MN), a gel that is 61% ethanol by weight. This product is located inside the doorway of every patient room in our hospital. To prevent transmission of pathogens, health care staff are encouraged to use the sanitizer when entering or leaving patient

TABLE 1. **Serial Toxicology in a 53-Year-Old Man With Alcoholism Who Was Hospitalized for Isopropanol Intoxication and Later Ingested Ethanol-Based Hand Sanitizer**

	Serum volatile alcohol screen					
Day	Methanol	Ethanol*	Isopropanol† (μg/mL)	Acetone (μg/mL)	Ethylene glycol	Urine drug screen‡
Admission	Negative	Negative	1002	2071	Negative	Negative
Hospital day 2	Negative	3759 μg/mL	455	753	Negative	Positive for ethanol

*In nondrinkers or sporadic drinkers, a serum ethanol level greater than 3000 μg/mL results in coma, whereas chronic drinkers may tolerate an ethanol level greater than 5000 μg/mL before experiencing coma. Notably, in most jurisdictions, the legal threshold for "driving under the influence" of ethanol is greater than or equal to 800 μg/mL.

†Serum isopropanol levels do not necessarily correlate with a patient's clinical status; deaths due to intoxication with isopropanol (levels ranging from 100 μg/mLß to 2500 μg/mL) have been reported.

‡The urine drug screen assesses for ethanol, amphetamines, barbiturates, benzodiazepines, cocaine, opiates, phencyclidine, and tetrahydrocannabinol.

rooms. Indeed, we endorse this practice. However, health care institutions should consider removing these agents from rooms occupied by high-risk patients such as those with alcoholism, those who are admitted with acute intoxications, and psychiatrically unstable patients. Indeed, despite one-to-one monitoring, our patient managed to acquire and ingest the sanitizer, perhaps because the health care staff perceived the product to be harmless or nonthreatening as a potential toxin. Our case also affirms the need to repeat toxicology testing in hospitalized patients with abrupt changes in mental status, particularly given that alcohol-based hand sanitizers and other potentially ingestible toxins are ubiquitous in the hospital setting.

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Association Between Priapism and Concurrent Use of Risperidone and *Ginkgo biloba*

To the Editor: Priapism, defined as prolonged and persistent erection of the penis that is not due to sexual stimulation and that is not alleviated by sexual intercourse or masturbation, is a urological emergency that needs timely diagnosis and treatment. Rarely, priapism can be a serious adverse effect of antipsychotic medications; virtually all antipsychotic medications have been reported to cause priapism because of their α-adrenergic antagonism.[1] *Ginkgo biloba* is used worldwide as a complementary medicine for its antioxidant and neuroprotective effects. However, its drug interaction with other medications, including antipsychotic agents, should be considered before it is prescribed for any purpose.

Report of a Case. A 26-year-old man presented to the emergency department of our hospital with a persistent and painful erection of the penis that had lasted for 4 hours. He had a history of paranoid schizophrenia, for which he was treated with 3 mg/d of risperidone for 3 years. He reported no other chronic illness, recent trauma, or use of drugs. He denied any adverse effects due to antipsychotic therapies. Two weeks before admission, he began taking 160 mg/d of generic *Ginkgo biloba* for occasional tinnitus.

On arrival at the emergency department, his blood pressure was 150/88 mm Hg; his pulse rate, 94 beats/min; and his temperature, 35.9°C. Findings on physical examination were normal except for a persistent erection of the penis. His hematocrit was 37%, and no dysmorphic red blood cells were noted on microscopic examination. Color flow Doppler sonography of the penis showed a decreased blood flow in the cavernosal arteries. His priapism was refractory to analgesics and ice packing. Blood gas analysis of the aspired cavernous venous blood revealed a pH value of 6.79, a P_{CO_2} of 114.9 torr, and undetectable P_{O_2}, findings consistent with veno-occlusion (low-flow) priapism. Two hours after corporal irrigation with diluted epinephrine, priapism subsided. The patient was discharged uneventfully and was instructed to discontinue the use of risperidone and *Ginkgo biloba*. On direct questioning, he reported having no prolonged erection or priapism when he was being treated with risperidone. The 3 mg/d dose of risperidone was resumed without adverse effect. On follow-up at 6 months, he reported normal erections with no further episodes of priapism.

Discussion. Priapism has multiple etiologies, including hematologic, neurological, iatrogenic, and pharmacological causes. Drug-induced priapism is usually of the ischemic (low-flow, veno-occlusion) type; arterial (high-flow) priapism is relatively rare and predominantly caused by trauma. An estimated 50% of those who experience drug-induced priapism are taking antipsychotic agents; α-adrenergic antagonism is thought to be the most important underlying mechanism.[2]

Risperidone, an atypical antipsychotic agent used primarily to treat psychosis and schizophrenia, has serotonergic, dopaminergic, and α-adrenergic antagonist properties. Its propensity to cause priapism can be linked to its high affinity for α_1-adrenergic blockade, an affinity higher than that of other atypical antipsychotics such as clozapine and olanzapine.[3]

Ginkgo biloba is extracted from leaves of *Ginkgo biloba* (the maidenhair tree); its most active ingredients are flavonoids (ginkgo-flavone glycosides) and terpenoids (ginkgolides and bilobalide). Because of its vasoregulatory activity and free-radical scavenging properties, *Ginkgo biloba* has been used to treat early-stage Alzheimer disease, vascular dementia, peripheral claudication, and tinnitus of vascular origin. Its use is generally well tolerated, but adverse effects such as gastrointestinal upset, headache, and dizziness have been reported.[4]

Extracts of *Ginkgo biloba* have many ingredients, the pharmacological and pharmacokinetic effects of which can be difficult to ascertain. Different active compounds may exert their pharmacological effects on different sites or organs. In humans, risperidone is mainly metabolized by cytochrome P450 isoform 2D6 (CYP2D6) and cytochrome P450 isoform 3A4 (CYP3A4),[5] both of which enzymes are inhibited by *Ginkgo biloba*.[6] Hence, concurrent use of *Ginkgo biloba* may