cose tolerance was found in 17 of 182 (9%) patients receiving nelfinavir, 17 of 109 (16%) receiving indinavir, 4 of 45 (9%) receiving saquinavir, 3 of 14 (21%) receiving ritonavir, and 3 of 29 (10%) receiving ritonavir plus saquinavir ($P > 0.05$).

Hyperglycemia, but not diabetes, was more frequent in men than in women (12% compared with 3%; $P < 0.002$). The median age of patients with impaired glucose tolerance (45.5 years) or diabetes (48 years) was higher than the median age of the entire study sample (37 years) ($P < 0.05$).

In conclusion, treatment with protease inhibitors, male sex, and older age are risk factors for impaired glucose tolerance. Only age, not treatment with protease inhibitors, was identified as a risk factor for diabetes.

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Thrombotic Thrombocytopenic Purpura after Ecstasy-Induced Acute Liver Failure

To the Editor: A 20-year-old man was admitted to the hospital with a 6-day history of progressive fatigue and jaundice. Laboratory tests revealed markedly elevated liver enzyme levels and reduced synthesis of clotting factors. Electrolyte levels, creatinine level, leukocyte count, thrombocyte count, hemoglobin level, lactate dehydrogenase level, and pancreatic enzyme levels were in the normal range. Disseminated intravascular coagulation, hemochromatosis, Wilson disease, and infectious and autoimmune hepatitis were excluded.

While recovering from acute liver failure, the patient became delirious and developed acute renal failure, acute pancreatitis, pronounced thrombocytopenia, microangiopathic hemolytic anemia, epistaxis, and petechial hemorrhage. A blood smear revealed fragmentocytes. T2-weighted magnetic resonance imaging showed high-signal bilateral areas in the parieto-occipital subcortical white matter (**Figure**). Skin biopsy and immunofluorescence microscopy excluded generalized vasculitis. Thrombotic thrombocytopenic purpura (TTP) was diagnosed. Neurologic symptoms improved and renal function recovered immediately after prednisolone (1 g daily) and plasma exchange therapies began. After recovery, the patient reported that he had ingested one tablet of 3,4-methylenedioxymethamphetamine (Ecstacy) 6 days before symptoms developed. Four weeks after the initiation of therapy, the platelet count and hemolysis measures returned to normal, and kidney function improved markedly. After 4 months, the patient was in good health and ready to return to work.

Acute adverse reactions to Ecstasy result from the variable stimulation of adrenergic receptors. These reactions are commonly idiosyncratic (1) and include delirious states, convulsions, cerebral hemorrhage, cardiac arrhythmias, hyperpyrexia, rhabdomyolysis, and acute renal and liver failure (2, 3).

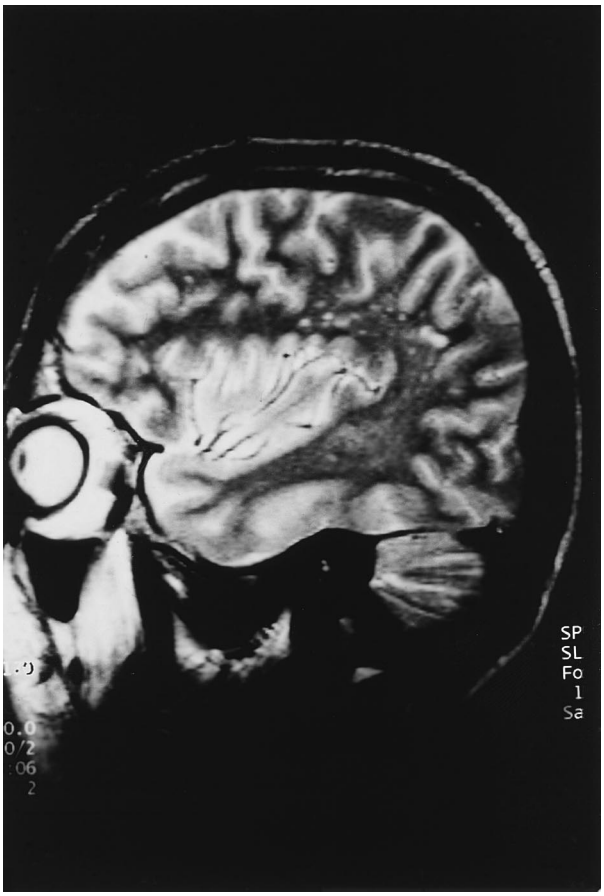

Figure. T2-weighted and proton density–weighted magnetic resonance imaging scan showing bilateral high-signal areas in parieto-occipital subcortical white matter.

We believe that our case is the first reported instance of a TTP-like syndrome secondary to ingestion of Ecstasy. Variants of TTP have been reported with infectious and autoimmune diseases, cancer, organ transplantation, and drugs. Drug abuse with stimulants such as cocaine has also been associated with TTP (4). Amphetamine-induced vasoconstriction is a mechanism (5) that may lead to secondary hemolytic microangiopathy by endothelial damage and the subsequent release of platelet procoagulant factors, even after a prolonged preclinical stage. Ecstasy may have involved the same pathophysiologic pathways in causing the TTP seen in our patient.

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Severe Hepatotoxicity Associated with Troglitazone

To the Editor: Two brief communications reported severe hepatotoxicity related to use of troglitazone (Rezulin, Parke-Davis,