time, 3 months earlier. The physician, the hotel manager and a close friend were all certain that the patient had not consumed alcohol for several years.

Comments

We have been unable to find any other recorded case of triazolam overdose. Our patient took more than 10 times the recommended hypnotic dose. The clinical picture 8 to 12 hours after ingestion of the tablets resembled that of hypnotic withdrawal delirium. Although benzodiazepine self-poisoning is frequent, confusion has rarely been described.[4] As triazolam is a short-acting benzodiazepine, overdose may be followed by a precipitous fall in the drug's serum concentration and the clinical features of hypnotic withdrawal. In fact, our patient was initially thought to be suffering from delirium tremens. Negative results of screening for benzodiazepines (as a result of the short half-life of the drug) may mean that physicians miss this diagnosis.

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Pancreatitis caused by mefenamic acid

To the editor: A 32-year-old woman was admitted to hospital because of abdominal pain for 10 hours. She described the sudden onset of severe, burning epigastric pain radiating through to her back. It was continuous and aggravated by movement and deep breathing. She had vomited bilious material several times but not blood. The woman did not have a history of hyperlipidemia, cholecystitis or peptic ulcer disease. She had no food intolerance, had never smoked and drank little alcohol (none within the past month). She had had an appendectomy and had borne four children.

The woman suffered from dysmenorrhea, for which she had undergone dilation and curettage a year earlier. She had tried a number of other analgesics but was currently taking mefenamic acid (Ponstan). The first cycle of this drug had been uneventful. The second cycle, 250 mg four times a day for 4 days, had ended 4 days before admission. The only other drugs she had taken were prednisone (orally) and beclomethasone dipropionate (as a nasal spray) for 2 weeks for rhinitis. This treatment had been stopped 6 weeks before admission.

She was in severe pain and her breathing was shallow. Her blood pressure was normal and showed no postural drop. Bowel sounds were distant. There was marked tenderness in the epigastrium and left upper quadrant but no guarding, rigidity or rebound tenderness. There was no tenderness to percussion of the flanks. Rectal examination revealed no occult blood in the stool.

The only abnormal laboratory finding was a high serum amylase level, 3315 (normally 20 to 110) IU/l, and radiologic investigation (roentgenography and ultrasonography) failed to reveal any abnormality.

She was treated with analgesics (pethidine) and intravenous fluids. Within a week she was free of pain and eating. At the time of discharge from hospital her serum amylase level was within normal limits.

The clinical and laboratory data indicate that this patient suffered an episode of acute pancreatitis. There was no evidence of a precipitating event except the recent exposure to mefenamic acid.

To implicate a drug as a cause of an illness requires demonstration that the illness occurs during exposure to that drug, that it subsides on withdrawal of the drug, and that the event is reproducible with re-exposure. Using these criteria a recent review established azathioprine, thiazides, sulfonamides, furosemide, estrogens and tetracycline as pancreatitis-inducing drugs.[1] The authors found less convincing evidence with respect to chlorthalidone, steroids and ethacrynic acid. Mefenamic acid was not mentioned. A computerized search of the medical literature yielded no references to the association of mefenamic acid and pancreatitis.

Because of evidence that primary dysmenorrhea may be due to excess uterine activity mediated by prostaglandins, drugs thought to inhibit prostaglandin synthesis and action are currently popular for treating dysmenorrhea.[2] Some studies suggest they are more effective than conventional analgesics,[3-5] although this is disputed.[2] Despite serious reservations concerning its toxicity mefenamic acid is one of those drugs currently being marketed with this indication in mind.[5] Reported adverse effects include leukopenia, eosinophilia, thrombocytopenia, agranulocytosis, pancytopenia, marrow hypoplasia and diarrhea, but pancreatitis is not mentioned.[6]

Although our case does not establish mefenamic acid as a cause of pancreatitis, physicians prescribing the drug should encourage patients to report abdominal symptoms and should investigate these reports if indicated.

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Recurrent herpes simplex

To the editor: The persistent negative reports in the lay press and by doctors who are interviewed on radio and television regarding the lack of a cure or treatment for recurrent herpes simplex has created a tremendous psychologic burden for patients with this disease. There are many diseases for which there is no cure but there is treatment.

For the past 2 years I have treated recurrent herpes simplex types 1 and 2 with cryotherapy. I see the patient within 24 hours of the first evidence of a recurrence, at which point the herpetic blisters are still intact. Theoretically most of the herpesviruses are in the epidermal cells then,[1] so they are in the best location for topical therapy. I touch the vesicles for at least 5 seconds and up to 10 seconds with a cotton-tipped applicator that has been dipped in liquid nitrogen. This causes mild discomfort. Over the next 2 days the blisters dry and scab, then they usually clear within 4 days.

It has been my observation that there are several advantages to this treatment. First, the duration of a recurrence is substantially decreased — from 10 to 14 days with no treatment to 3 to 4 days with cryotherapy. Second, the incidence of secondary bacterial infection is negligible, possibly because cryotherapy destroys bacteria in the area and dries the fluids that are a good medium for the growth of bacteria. Third, the frequency of recurrence is decreased; perhaps the cryotherapy de-