Neuroleptics

Violence as a Manifestation of Akathisia

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NEUROLEPTIC medications (eg. phenothiazines, butyrophenones) are used in medicine to control psychotic symptoms and concomitant agitated and violent behavior. They also are used to control anxiety and agitation whenever minor tranquilizers (eg. benzodiazepines) would be inappropriate. Development of akathisia as a parkinsonian side effect is confirmed in the use of these drugs. Akathisia is a condition that gives rise to the subjective desire to be in constant motion, with a feeling of inner agitation and muscle tension. The patient cannot sit still and paces constantly.1

To my knowledge, however, the literature does not contain reports that the development of akathisia can precipitate violence, resulting in the behavior the drug was meant to alleviate.

Report of a Case

A 29-year-old man had a diagnosis of sociopathic personality and transvestism and a long history of drug abuse, including amphetamines, marijuana, alcohol, LSD, mescaline, benzodiazepines, and narcotics. The patient had a prior history of violence when alcohol-intoxicated and had been in barroom brawls. He also had a lifelong history of anger and hostility toward authority figures and had a record of juvenile arrests, though none in the past nine

The patient came to therapy because of depression for one year related to internal conflicts over transvestism and joblessness. He complained of loss of appetite, difficulty sleeping at night, and social isolation. At the time of admission, he smoked one to four joints of marijuana per day because it "mellowed" him. He denied any other drug abuse.

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Treatment was started with imipramine hydrochloride, and after four weeks of a dosage of 250 mg/day, he responded with great decrease in feelings of depression. However, he became somewhat hostile and aggressive at six weeks of therapy. He complained of thought disorganization and ideas of reference, but no firm psychotic symptoms were present. The imipramine was thought to have possibly stimulated an underlying psychosis and some of the anger and hostility, although it was alleviating the depression.2

Treatment with imipramine hydrochloride was tapered to 100 mg/day. Haloperidol therapy, 2 mg at bedtime, was started for impulse control. Haloperidol was chosen for two reasons: (1) the patient had a past history of abuse of minor tranquilizers, and (2) there was the possibility of a latent schizophrenia stimulated by the imipramine. The patient responded and reported a great decrease in his level of hostility, agitation, and thought disorganization. The imipramine controlled the depression, and the haloperidol controlled the violent tendencies and thought disorganization.

One week later the patient reported that he was more agitated at night. Since it was not known at the time that akathisia was beginning, haloperidol treatment was increased to 4 mg at bedtime to decrease the agitation. Four days later, after his evening dose of 4 mg of haloperidol, he became uncontrollably agitated, could not sit still, and paced for several hours. He complained of tightness in his muscles, rigidity, a jumpy feeling inside, and violent urges to assault anyone near him. This culminated in an assault on his dog with an intent to kill. He became frightened over his loss of control and came to the emergency room. He was given 50 mg of thioridazine hydrochloride, which brought the hostility under control but did not remove it.

He subsequently discontinued the treatment with imipramine and haloperidol. The following morning he reported that the muscle tightness, jumpy feelings, and hostility were decreased but still present. Three days after drug treatment was discontinued, all of these symptoms had

ceased, and he was at his baseline of difficulty once again. The half-life of haloperidol is approximately 24 hours, and this symptom relief coincided with expected excretion of the drug.

In retrospect it was apparent that he had experienced increasing akathitic side effects from the haloperidol medication. which accounted for his increasing nighttime agitation and culminated in a stimulation of violent and aggressive activity.

The patient was subsequently treated with thioridazine hydrochloride, 50 mg at bedtime, and imipramine hydrochloride, 100 mg at bedtime, with good response, and no further episodes of akathisia and subsequent violence occurred. He also became involved in individual psychotherapy and began to deal with some of the issues causing his depression.

The description of the developing akathisia in this patient in relation to the increasing haloperidol dosage is apparent, as is its remission when therapy with the drug was discontinued.

Comment

This person had a characterological and social predisposition to violence. He received imipramine, which also increased his levels of hostility and aggressiveness. The final event precipitating a homicidal action was an akathitic reaction. Since haloperidol and other neuroleptics are used widely in patients with predispositions for violence, clinicians should be aware that it can precipitate a violent episode if there is a severe akathitic reaction.

Nonproprietary Name and Trademark of Drug

Thioridazine hydrochloride-Mellaril Hydrochloride.

References

- 1. Freedman AM, Kaplan HI, Sadock BJ: Comprehensive Textbook of Psychiatry. Baltimore, Williams & Wilkins Co, 1975, pp 817, 1794,
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